RADIOLOGY

Legend

Major Topic	Abbreviation
Digital Radiology	Dig Rad
Image Characteristics	Im Char
Miscellaneous	Misc.
Normal Anatomy	N Anat
Processing	Proc
Radiation Biology	R Bio
Radiation Characteristics	R Char
Radiation Physics	R Phys
Radiation Protection	R Prot
Techniques	Tech
X-rays	X-rays

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The area from which **x-rays emanate** is called the:

- Target
- Focal spot
- Intensifying screen
- Cone

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• **Focal spot**

***The **focal spot** is the area of tungsten on the anode that receives the impact of the speeding electrons and converts them into x-ray photons. It is one of three factors influencing image sharpness *(the others are film composition and movement)*.

The **target** *(also called the tungsten target)* is a wafer of tungsten embedded in the face of the anode at the point of electron bombardment. Tungsten is used due to its high atomic number *(x-ray production)*, high melting point, high thermal conductivity *(dissipates heat)*, and low vapor pressure *(maintains vacuum at high temperature)*. **Note:** The focal spot is actually a small area of the target.

Remember: Intensifying screens are devices used in extraoral radiography that convert x-ray energy into visible light. The light, in turn, exposes the screen film. Therefore, the radiation that a patient receives is decreased. A **cassette holder** is a light-tight device used in extraoral radiography to hold film and intensifying screens.

Important: Target film distance *(also called source-to-film distance)* is the distance from the source of x-rays *(focal spot on the tungsten target)* to the film. It is determined by the length of the position-indicating device *(also called PID)*. Two standard target-film distances are used in intraoral radiography:

1. 20 cm *(8 inches)* → is called the **short cone,** exposes more tissue by producing a more divergent beam.
2. 41 cm *(16 inches)* → is called the **long cone,** reduces the amount of exposed tissue by producing a less divergent beam and a sharper image.

Which type of structure **inhibits** the passage of x-rays?

- Radiopaque
- Radiolucent

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- **Radiopaque**

Radiopaque structures / materials:

- **Less** radiation penetrates the structure and reaches the film
- Radiopaque structures appear **white** on the processed film
- Dense materials such as **metals, enamel, dentin, and bone**

Radiolucent structures / materials:

- Allow radiation to pass through, absorbing very little
- **More** radiation penetrates the structure and reaches the film
- Radiolucent structures appear **gray to black** on processed film
- Less dense materials, including **soft tissue and air space**

Note: Radiographs show shading from **black to white** *(most radiolucent to most radiopaque).* **Example: Least to most radiopaque:** periodontal ligament space, dentin, enamel, ZOE, amalgam.

It is best to retain **dental radiographs** for how many years?

- 2 years
- 4 years
- 6 years
- Indefinitely

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- **Indefinitely**

The **dental record** must include documentation of informed consent and the exposure of radiographs *(e.g., the number and type of films, the rationale for exposure and the interpretation)*. Legally, dental radiographs are the **property of the dentist.** Patients do, however, have a right to reasonable access to the dental radiographs, which includes having a copy of the radiographs forwarded to another dentist.

Note: Patients may **refuse dental x-rays**, however, the dentist must decide whether an accurate diagnosis can be provided and whether treatment can provided.

Remember: No document can be signed by the patient that releases the dentist from liability.

Osteoradionecrosis is more common:

- In the mandible
- In the maxilla

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• **In the mandible**

Osteoradionecrosis is more common in the **mandible** than in the maxilla, probably because of the richer vascular supply to the maxilla and the fact that the mandible is more frequently irradiated.

Remember: Osteoradionecrosis is the necrosis of bone, produced by ionizing radiation.

The most common factors **precipitating** osteoradionecrosis are pre- and post-irradiation extractions and periodontal disease.

Note: Damage to the **blood vessels** *(as opposed to nerves, muscle, etc.)* predisposes a patient to the development of osteoradionecrosis.

To **prevent** osteoradionecrosis: extract all hopeless teeth three weeks prior to head / neck radiation treatment. If extracting after radiotherapy, the use of systemic antibiotics is recommended. Some studies suggest hyperbaric oxygen treatments before and after treatment to reduce the risk of osteoradionecrosis *(this is somewhat controversial)*.

Your dental hygienist has a patient that says she needs bite-wing x-rays because it has been six months since the last films were taken. Your hygienist **should respond** in which manner listed below?

- Agree with the patient
- Tell the patient that bite-wing x-rays should be taken once a year
- Tell the patient that dental x-rays are taken only when needed as judged by each patient's needs
- None of the above

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•Tell the patient that dental x-rays are taken only when needed as judged by each patient's needs

Decisions about the number, type and frequency of dental x-rays **are determined by the dentist** based on each patient's needs. Every patient has a different dental condition and thus the frequency of x-rays is different as well. There are guidelines published by the ADA that aid a dentist in prescribing the number, type and frequency of dental x-rays.

Note: Patients who have tooth decay, periodontal disease, tooth mobility, pain in one or more teeth or possible impacted teeth **need more frequent** radiographic examinations than patients without such problems. **Remember:** For a pediatric patient who is caries free *(and asymptomatic)*, the first bite-wing radiographs should not be taken until the spaces between the posterior teeth have closed.

Note: Occult diseases *(for example, small carious lesions, cysts and tumors)* are those presenting **no clinical signs or symptoms.** Because occult disease in the perioral tissues is so rare *(except for caries)*, a radiographic examination of the jaws **should not** be undertaken solely to look for it in an individual with teeth when there are no clinical signs or symptoms. However, every x-ray taken should be evaluated for these lesions.

Remember: Caries is an **exception to the above rule** because of its much higher prevalence as compared to occult cysts or tumors.

Identify the structure below that the arrows are pointing to:

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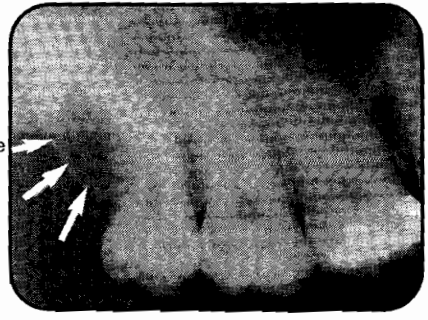
• **The hamulus**

The **hamulus** *(also known as the hamular process)* is a small hook-like projection of bone extending from the medial pterygoid plate of the sphenoid bone. The hamulus is located posterior to the maxillary tuberosity region.

On the radiograph its image is seen in proximity to the posterior surface of the tuberosity of the maxilla. It varies greatly in length, width and shape from patient to patient. It usually exhibits a bulbous point, but sometimes the point is tapered.

The maxillary tuberosity appears as a radiopaque bulge distal to the third molar region



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The image of the **coronoid process** of the mandible often appears in periapical x-rays of:

- The incisor region of the mandible
- The molar region of the mandible
- The incisor region of the maxilla
- The molar region of the maxilla

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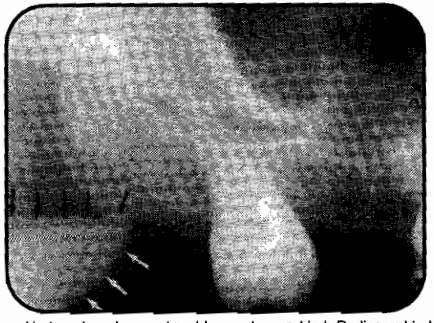
• **The molar region of the maxilla**

As the mouth is opened, the process moves forward, and therefore it comes into view **most often** when the mouth is opened to its fullest extent at the time the exposure is made. It is evidenced by a **tapered or triangular radiopacity,** which may be seen below, or in some instances, superimposed on the **molar teeth and maxilla.**

The coronoid process appears as a triangular-shaped radiopacity.



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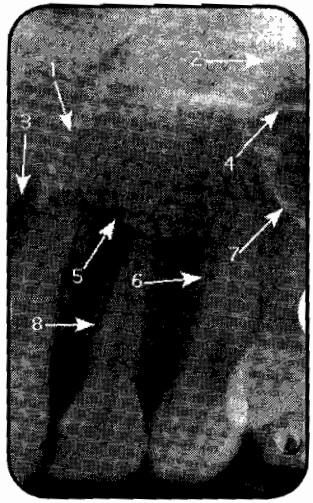
Identify each structure that the arrows 1 — 8 point to in the **anterior region** of the maxilla.



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1. **The opaque line** → Lateral wall of nasopalatine canal

2. **The opaque line** → Anterior wall of maxillary sinus

3. **The radiolucent structure** → Nasopalatine fossa

4. **The opaque line** → Floor of nasal fossa

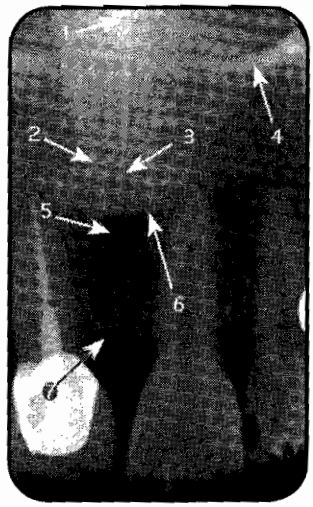
5. **The opaque structure** → Soft tissue tip of nose

6. **The opaque line** → Lamina dura

7. **The opaque line** → Border of maxillary sinus

8. **The radiolucent line** → Periodontal ligament space

Identify each structure that the arrows 1 —7 point to in the **anterior region** of the maxilla.



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1. **The opaque structure** → Anterior nasal spine

2. **The opaque line** → Lateral wall of nasopalatine canal

3. **The radiolucent line** → Intermaxillary suture

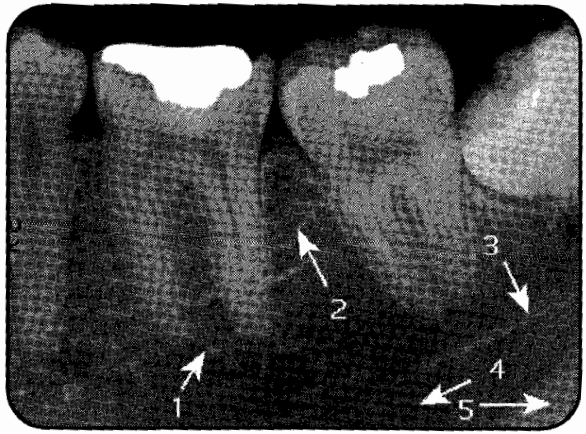
4. **The opaque line** → Floor of nasal fossa

5. **The radiolucent structure** → Nasopalatine fossa

6. **The radiopaque line** → Soft tissue tip of nose

7. **The opaque structure** → Alveolar crest

Identify each structure that the arrows 1 —5 point to in the **mandibular molar** region.



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1. **The radiopaque lines** → Nutrient canal

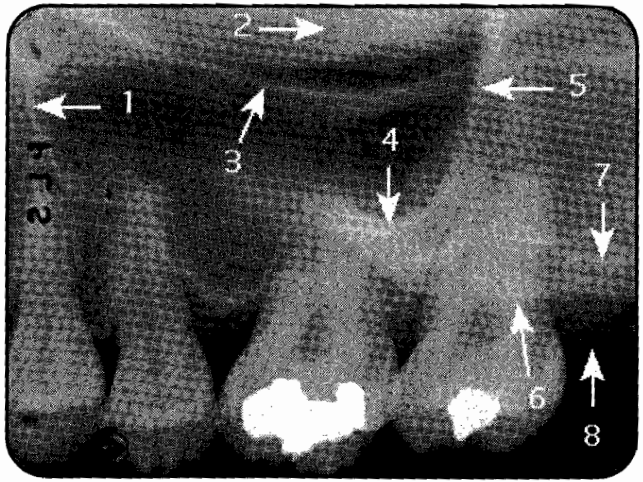
2. **The opaque line** → Bony trabecular plate

3. **The opaque line** → Inferior border of mandibular canal

4. **The radiolucent space** → Submandibular gland fossa

5. **The radiopaque structure** → Inferior border of mandible

Identify each structure that the arrows 1 — 8 point to in the **maxillary molar** region.



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1. **The opaque line** → Anterior wall of maxillary sinus

2. **The opaque mass** → Inferior concha

3. **The opaque line** → Floor of nasal fossa

4. **The opaque line** → Inferior border of zygomatic process of maxilla

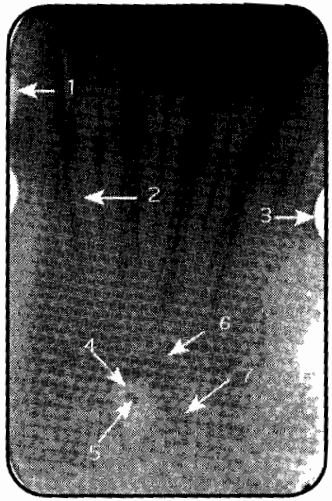
5. **The opaque line** → Posterior wall of zygomatic process of maxilla

6. **The opaque line** → Inferior border of zygoma *(zygomatic arch)*

7. **The opaque line** → Floor of maxillary sinus

8. **The opaque structure** → Mucosa over maxillary alveolar ridge

Identify each structure that the arrows 1 — 7 point to in the **mandibular incisor** region.



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1. **The opaque structure** → Lingual cusp of 1st premolar

2. **The radiolucent line** → Periodontal ligament space

3. **The opaque mass** → Film holder

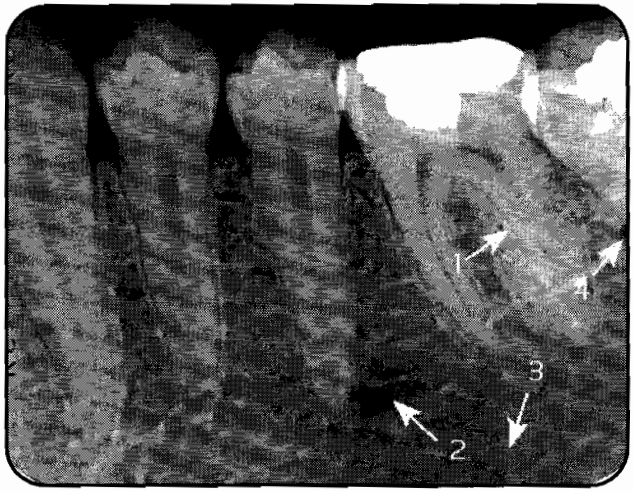
4. **The opaque mass** → Genial tubercles

5. **The radiolucent circle** → Lingual foramen

6. **The opaque line** → Bony trabecular plate

7. **The radiolucent space** → Marrow space

Identify each structure that the arrows 1 — 4 point to in the **mandibular premolar** region.



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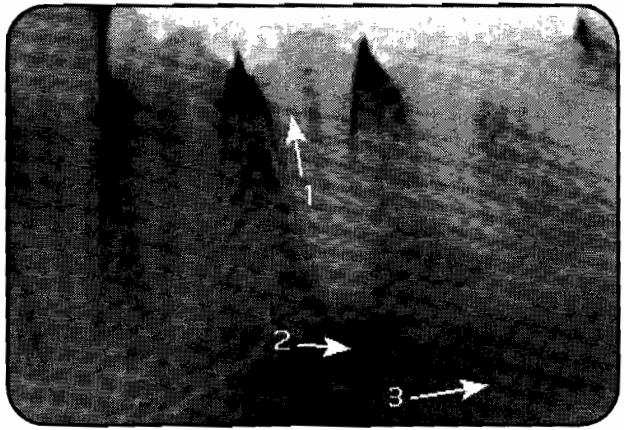
1. **The radiolucent line** → Periodontal ligament space

2. **The radiolucent space** → Mental foramen

3. **Large radiolucent space** → Submandibular gland fossa

4. **Dark dot** → Film clip mark

Identify each structure that the arrows 1 — 3 point to in the **mandibular premolar** region.



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1. **The opaque line** → Cemento-enamel junction

2. **The radiolucent space** → Mental foramen

3. **Large radiolucent space** → Submandibular gland fossa

X-ray developer contains all of the following **except:**

- A developing agent
- An antioxidant preservative
- A clearing agent
- An accelerator
- A restrainer

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- **A clearing agent**

X-ray developing solution contains the following:

- A **developing agent,** such as **hydroquinone,** which is a chemical compound that is capable of changing the exposed silver halide crystals to black metallic silver. At the same time, it produces no appreciable effect on the unexposed silver halide crystals in the emulsion. **Gives detail to the x-ray image. Note:** Also Elon which quickly generates gray tones.
- An **antioxidant preservative,** for example, **sodium sulfite,** prevents the developer solution from oxidizing in the presence of air.
- An **accelerator** – an alkali *(sodium carbonate)* → activates the developing agents and maintains the alkalinity of the developer at the correct value. It softens gelatin of emulsion.
- A **restrainer,** such as **potassium bromide,** is added to developers to control the action of the developing agent so that it does not develop the unexposed silver halide crystals to produce fog.

Remember: Developer is a chemical solution that **converts** the invisible image on a film into a visible one composed of minute masses of black metallic silver.

Important: The **function of developing** solution is to reduce silver halide crystals to black metallic silver, while the **function of fixing solution** is to stop development and remove remaining unexposed crystals.

Film processing involves the following steps: immerse film in developer → rinse film in water bath → immerse film in fixer → wash film in water bath → dry film

X-ray fixer contains all of the following **except:**

- A clearing agent
- An antioxidant preservative
- An accelerator
- An acidifier
- A hardener

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- **An accelerator**

X-ray **fixing solution** contains the following:

- A **clearing agent,** such as **sodium or ammonium thiosulfate,** commonly called hypo, dissolves and removes the underdeveloped silver halide crystals from the emulsion (**Note:** *this is one of the main* **functions** *of fixing solutions.)* The chemical "clears" the film so that the black silver image produced by the developer becomes distinctly perceptible. When the film is improperly cleared, the remaining unexposed silver halide crystals darken upon exposure to light and obscure the image.
- An **antioxidant** preservative, for example **sodium sulfite**, prevents the decomposition of the fixer chemical.
- An **acidifier** such as **acetic acid,** that is necessary for the correct action of the other chemicals and also neutralizes any alkaline developer that may be carried over by the film or hanger.
- A **hardener** such as **potassium alum** that shrinks and hardens the gelatin in the emulsion. It shortens drying time and protects the emulsion from abrasion.

Notes:

1. If a **dried radiograph** were processed a second time, there would be no change in contrast or density.
2. Fixing time is always at least **twice** as long as the developing time.

Ashley has taken three panoramic x-ray films today. During the day as she developed each film, she noticed the **films getting lighter and lighter.** What needs to be done so that this problem can be corrected?

- Decrease the temperature of the developing solution
- Increase the temperature of the developing solution
- Replenish the developing solution
- Increase the mA setting
- Increase the kVp setting

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• **Replenish the developing solution**

As the developing solution **gets weaker,** the films will get lighter. Both the developing and fixing solutions should be replenished on a daily basis. These solutions also need to be changed on a regular basis, and the tanks need to be scrubbed and cleaned as well.The following **factors** affect the life of a developing solution: the cleanliness of the tanks, the size of the films processed, the number of films processed, and the temperature of the solution.

Notes:

1. **Yellowish-brown film** will result from insufficient fixing or rinsing ***(See figure #1)***.
2. **Fogged film** may also result from improper film storage or outdated films.
3. **Low solution levels will appear as:** developer cut-off *(straight white border,* ***see figure #2)***or fixer cut-off *(straight black border,* ***see figure #3)***.
4. **Light spots** on film may result from contact with the fixer before processing ***(See figure #4)***. Developer spots appear dark or black ***(See figure #5).***

Fig. #1

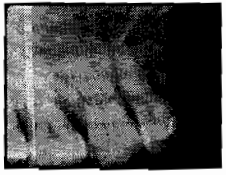

Fig. #3

Fig. #2

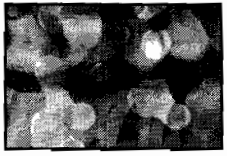

Fig. #4

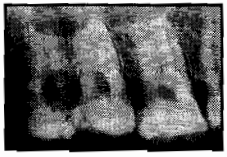

Fig. #5

After **processing** a film, you notice that it appears **brown** in color. What is the most likely cause?

- Solutions are too strong
- Solutions are too weak
- Fixing time was not long enough
- Fixing time was too long
- Film was under-developed

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- **Fixing time was not long enough**

***A film will appear brown when it is **not** completely fixed.

Some common **errors** made in the darkroom → **cause:**

- **Mounted films are improperly labeled** *(wrong patient name)* → racks not labeled properly
- **Fogged film** *(gray / lack of contrast)* → faulty safelight in darkroom; white light leaking into darkroom
- **Lost films** → films not secured properly on rack
- **Static marks** *(multiple black lines)* → friction when opening film packets causes static electricity
- **Overdeveloped film** *(dark)* → incorrect time *(too long)* and temperature *(too hot)*
- **Underdeveloped film** *(light)* → incorrect time *(too short)* and temperature *(too cold)*; weak solutions *too old or diluted)*
- **Torn emulsion** → films were allowed to touch or overlap while they were drying
- **Stained film** *(dark / white spots)* → dirty work surfaces; person developing film was sloppy
- **Scratched films** *(white lines)* → film emulsion removed by sharp object *(fingernails / rack touching)*
- **Clear films** *(emulsion washed away)* → films left in water *(wash)* for over 24 hours
- **Air bubbles** *(white spots)* → air trapped on film surface while being placed in processing

The unit for measuring the absorption of x-rays is termed:

- REM
- RAD
- Roentgen
- QF

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• **RAD** → radiation absorbed dose

The **rad** is a unit used to measure a quantity called **absorbed dose.** This relates to the amount of energy actually absorbed in some material, and is used for any type of radiation and any material. One rad is defined as the absorption of 100 ergs per gram of material. The unit rad can be used for any type of radiation, but it does not describe the biological effects of the different radiations.

The **rem** *(roentgen equivalent man)* is a unit used to derive a quantity called **equivalent dose.** This relates the absorbed dose in human tissue to the effective biological damage of the radiation. Not all radiation has the same biological effect, even for the same amount of absorbed dose. Equivalent dose is often expressed in terms of thousandths of a rem, or mrem. To determine equivalent dose *(rem)*, you multiply absorbed dose *(rad)* by a quality factor *(QF)* that is unique to the type of incident radiation. The **QF** is a factor used for radiation protection purposes that accounts for the exposure effects of different types of radiation. For x-rays QF = 1.

The **roentgen** is a unit used to measure a quantity called **exposure.** This can only be used to describe an amount of gamma and X-rays, and only in air.

Exposure is a measure of radiation quantity, the capacity of the radiation to ionize air.

Equivalent dose is used to compare the biologic effects of different types of radiation to a tissue or organ.

Effective dose is used to estimate the risk in humans.

Which of the following is considered **radioresistant?**

- Immature reproductive cells
- Young bone cells
- Mature bone cells
- Epithelial cells

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• **Mature bone cells**

Cells in the body have **different sensitivities** to radiation than others.

Radiosensitive Cells	**Radioresistant Cells**
Small lymphocytes *(immature blood cells)*	Mature bone
Bone Marrow	Muscle
Reproductive cells *(sperm and ova)*	Nerve
Immature bone cells	

In general, the **greater the rate or potential for mitosis** and the **more immature the cells and tissues** are, the **greater the sensitivity** or susceptibility to radiation.

It is recommended that the operator stand **at least how many feet away** from the patient when taking radiographs?

- Two feet
- Four feet
- Six feet
- Eight feet

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- **Six feet**

Radiation exposure to the operator can be **reduced** by standing at least six feet away, behind a lead shield, or both when exposing radiographs. The operator should **never** remain in the room holding the x-ray packet in place for the patient. If a film **must** be held in place by someone else *(for a child),* drape the parent and have him or her hold the film. All dental personnel should wear film badges that monitor exposure dosages.

The operator **must avoid** the primary beam by positioning themselves at a 90 to 135 degree angle to the beam.

Note: Regarding the taking and processing of dental radiographs, always remember to maintain proper infection control at all times!!!

Kilovoltage controls the speed of:

- Photons
- Electrons
- Anodes
- Cathodes

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• **Electrons**

The speed with which **electrons** travel from the filament of the cathode to the target of the anode depends upon the potential difference between the two electrodes *(kilovoltage).* This, in turn, has a **very important effect** on the x-rays produced at the focal spot.

The kilovoltage has nothing to do with the number of electrons that compose the stream flowing from cathode to anode. The **number of electrons** *(which determines the quantity of x-rays produced)* is controlled by the **temperature of the tungsten filament** *(milliamperage setting).* The hotter the filament, the more electrodes are emitted and available to form the electron stream *(the x-ray tube current).* In the x-ray tube the number of electrons flowing per second is measured in **milliamperes**. The intensity of x-rays produced at a particular kilovoltage depends on that number. **Note:** Setting the x-ray machine for a specific milliamperage actually means adjusting the filament temperature to yield the current flow indicated. The milliamperage range for dental radiography is 7-15 mA.

Notes:

1. In dental radiography, the **quality** of the x-ray beam is controlled by kVp.
2. The kilovoltage range for most dental x-ray machines is **65-100 kV.**
3. A **higher kilovoltage** produces x-rays with greater energy levels, shorter wavelengths and more penetrating ability.
4. **To increase film density,** you should **increase** mA, kVp and time. Also, you should **decrease** the source-object distance.

The **inverse square law** formula is:

- $\frac{\text{New intensity}}{\text{Original intensity}} = \frac{\text{Original distance}^2}{\text{New distance}^2}$
- $\frac{\text{New intensity}}{\text{Original intensity}} = \frac{\text{New distance}^2}{\text{Original distance}^2}$
- $\frac{\text{Original intensity}}{\text{New intensity}} = \frac{\text{New distance}^2}{\text{Original distance}^2}$
- None of the above

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- $\frac{\text{Original intensity}}{\text{New intensity}} = \frac{\text{New distance}^2}{\text{Original distance}^2}$

Important point: For a given beam of radiation the intensity is inversely proportional to the square of the distance from the source of radiation.

The **intensity** of an x-ray beam is the total energy of the x-ray beam; it is the product of the quantity *(number of x-ray photons)* and quality *(energy of each photon)* per unit of area per time of exposure.

The intensity of an x-ray beam at a given point is **dependent** on the distance of the measuring device from the focal spot. The reason for this decrease in intensity *(why it is* **inversely** *proportional)* is that the x-ray beam spreads out as it moves from the source. The "spread out" beam is less intense.

For example, when the PID length is changed from 8 to 16 inches, the source-to-film distance is doubled. According to the Inverse Square Law, the resultant beam is one-fourth as intense. When the PID length is changed from 16 to 8 inches, the source-to-film distance is reduced by one-half. According to the Inverse Square Law, the resultant beam is four times as intense.

Remember: The intensity of the radiation is inversely proportional to the square of the distance.

Increasing the kilovoltage *(kVp)* causes the resultant x-ray to have:

- Decreased density
- More latitude
- A shorter scale of contrast
- A longer scale of contrast

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• **A longer scale of contrast**

Remember: Kilovoltage controls the **speed of electrons.**

One effect of a **change in kilovoltage** is a change in the penetrating power of the x-rays. Increasing kilovoltage reduces subject contrast *(and the longer the scale of contrast);* **decreasing** kilovoltage increases subject contrast *(and the shorter the scale of contrast).* A second effect of an increase in kilovoltage is that not only are new, more penetrating x-rays produced, but more of the less penetrating rays which were also produced at the lower kilovoltage are omitted.

Conclusion → kilovoltage **influences the x-ray beam** and radiograph by:
• Altering contrast quality *(for patients with* **thick jaws,** *increase kilovoltage)*
• Determining the quality of the x-rays produced
• Determining the velocity of the electrons to the anode

The quantity of radiation produced is controlled by:

- Kilovoltage *(kVp)*
- Milliamperage *(mA)*
- Exposure time
- Milliamperage *(mA)* and exposure time

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- **Milliamperage *(mA)* and exposure time**

The **operator** of an x-ray unit is in control of three factors:

1. **Kilovoltage** → the **quality** or **penetrating power** of the x-ray beam
2. **Milliamperage** → the **quantity** or **number** of x-rays produced
3. **Exposure time** → the length of time x-rays are produced and patient is exposed to them

Suitable ranges of dental x-rays are 65-100 kVp and 7-15 mA.

Some x-ray machines are calibrated in **"impulses"** *(there are 60 impulses in 1 second).*

Density refers to the overall darkness *(blackness)* of a radiograph

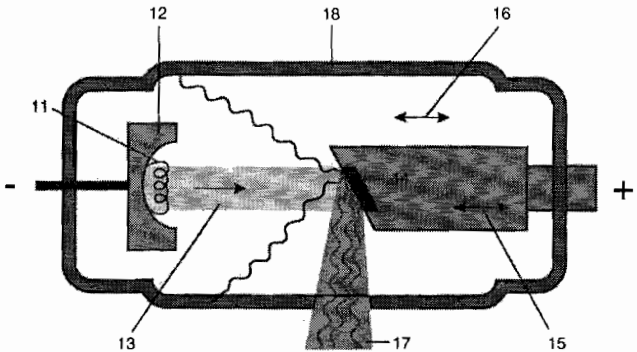
- Density will **increase** as mA, kVp, or exposure time is **increased**
- Density will **decrease** as mA, kVp, or exposure time is **decreased**

Contrast refers to the difference in degrees of blackness between adjacent areas on a radiograph

- **High** → very dark and very light areas
- **Low** → many shades of gray → **preferred in dentistry**
- **Only one exposure factor affects contrast → kilovoltage** *(kVp)* **Note:** Filtration plays a role here also. Higher kVp settings produce more shades of gray or low contrast.

Remember: Increasing milliamperage results in an **increase** in the number of x-rays produced and an **increase** in the temperature of the filament.

Identify the following parts the **dental x-ray tube.**



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11. Filament
12. Molybdenum cup
13. Electron stream
14. Tungsten target
15. Copper sleeve / Anode
16. Vacuum
17. X-ray beam
18. Leaded glass housing

X-rays are **generated** when a stream of electrons *(which are produced by the filament)* travels from the cathode to the anode and is suddenly stopped by its impact on the tungsten target. The filament located in the cathode is made of tungsten wire. The small area on the target that the electrons strike is called the **focal spot** → it is the source of x-rays.

Notes:

1. The size of the focal spot **directly influences** the x-ray definition → the larger the focal spot, the greater the loss of definition and sharpness of the image.
2. **Copper** is used to house the anode because it is a good thermal conductor, dissipating heat from the tungsten target and reducing the risk of melting the target.
3. The **Molybdenum cup,** also known as the electron focusing cup, electrostatically directs the electrons from the filament in the cathode to the focal spot on the anode.

The amount of material that is required to reduce the intensity of an x-ray beam to half is called the:

- Value layer
- Intensity value layer
- Half-value layer
- Full-value layer

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• **Half-value layer**

For x-ray beams, this is normally expressed in aluminum or copper thickness, but can also be expressed in other materials or media, such as water. Strictly, the half value layer is defined for different quantities: photon fluence *(number of photons/cm2)*, energy fluence *(number of photons x photon energy/cm2)* or absorbed dose. The term intensity is commonly used but is too vague and should therefore be avoided.

Due to the spectral nature of x-rays, the half-value layer *(HVL)* is not constant. When measuring multiple half value layers, the second HVL is greater than the first. This is due to the fact that the mean energy of the x-ray spectrum is increased following passage of the first HVL, which results in the x-rays becoming more penetrating.

In oral diagnostic radiography, the half-value layer of the beam of radiation is approximately **2 mm of aluminum.** This means that half of the x-rays exiting the vacuum tube are absorbed by 2 mm of aluminum. It should be noted that doubling the thickness of aluminum **will not absorb** all of the x-rays, but only half of the remaining x-rays.

Important point to remember: The half-value layer is an indicator of the penetrating **quality** of an x-ray beam. The higher the half-value layer, the more penetrating the beam. **Note:** X-rays and gamma rays are examples of **non-particulate** radiation energy.

The **radiation generated** at the anode of the x-ray tube is called:

- Primary radiation
- Scattered radiation
- Potential radiation
- Reverse radiation

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• **Primary radiation**

It is attenuated by the filter and the object. The amount of primary radiation follows the **inverse square law** measured from the focal spot. The attenuation of primary radiation should be measured with a narrow beam geometry to exclude all secondary *(i.e., scattered radiation)*.

Secondary radiation *(scattered radiation)* which arises from interactions of the primary radiation beam with the atoms in the object being imaged. Because the scattered radiation deviates from the straight line path between the x-ray focus and the image receptor, scattered radiation is a major source of image degradation in both x-ray and nuclear medicine imaging techniques. When x-ray radiation passes through a patient, three types of interactions can occur, including coherent scattering *(coherent scatter)*, photo electric absorption and Compton scattering. Of these three events, the great majority of scattered x-rays in diagnostic x-ray imaging arise from Compton scattering.

Notes:

1. **Lead is most effective** in stopping x-rays *(patients should* **always** *wear a lead apron and thyroid collar).*
2. X-rays have **more energy** than light.
3. The following belong to a group of radiations known as **electromagnetic radiations:** microwaves, x-radiation, visible light, and gamma radiation.
4. Approximately 1% of the energy released in the x-ray tube is released as x-rays.
5. **MPD** stands for maximum permissible dose of radiation exposure.
6. The yearly MPD for a **non-occupationally exposed** person is **0.5 REM** compared with **5 REM** for someone who works near radiation.
7. The type of cone *(PID)* that **best** reduces the amount of scatter radiation that the patient receives is a **leaded, rectangular** one since this greatly reduces the size of the beam.

The removal of parts of the x-ray spectrum using absorbing materials in the x-ray beam is called:

- Elimination
- Filtration
- Collimation
- Reduction

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• **Filtration**

The x-ray spectrum reaching the patient is filtered by attenuating material in its path. Filtering of the beam is used in order to modify the spectral or spatial distribution of x-rays, or both. Filtration is in principal divided in two parts:

1. **Inherent filtration** → the filtration of an x-ray beam by any parts of the x-ray tube or tube shield through which the beam must pass. The parts include the glass envelope of the x-ray tube, the oil cooling the tube and the exit window in the tube housing. The inherent filtration corresponds to approximately 0.51 mm of aluminum.
2. **Added filtration** → is obtained by placing thin sheets of aluminum in the cone to filter the useful beam further.

The **total filtration** of the x-ray beam before it reaches the patient consists of the inherent filtration plus the added filtration. **Recommended total:** equivalent of 0.5 mm *(below 50 kVp)* and 2.5 mm *(over 70 kVp)* of aluminum.

Notes:

1. **Longer wavelength x-rays** *(those produced at lower kilovoltages)* are **easily absorbed.**
2. **Shorter wavelength x-rays** *(those produced at higher kilovoltages)* **penetrate objects** more readily *(they form the image on the film).*

Remember: The x-ray beam is composed of rays of different wavelengths and penetrating power *(the term used for this is* **polychromatic***)* because the potential across the tube changes constantly as the voltage varies.

The **most effective means** in reducing the time of exposure, the amount of radiation reaching the patient and the amount of radiation scattered to the dentist is:

- A lead apron
- Ekta-speed film
- Lead diaphragms
- Increasing target-film distance

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- **Ekta-speed film**

All of the following **reduce** the amount of radiation to the **patient:**
- A lead apron
- Increased filtration using an aluminum disk
- Ekta-speed film
- Lead diaphragms placed within the cone of an x-ray tubehead
- Collimating an x-ray beam
- Increasing source-film distance
- Intensifying screens *(**used for all extraoral radiography** → panoramic, cephs.)*

Note: The Committee on Radiation Protection of the National Bureau of Standards recommends that a person who works near radiation be exposed in one year to a maximum dose of **5 REM** *(0.1 REM per week).* **Secondary** *(scatter radiation)* pose the greatest hazard to the dental team.

Important: Carcinogenesis and genetic mutation are important and serious effects of repeated exposure to low doses of x-radiation. The mechanisms involved may be frameshift mutations, synergism with chemical carcinogens and altered DNA repair enzyme functions.

The use of metal plates, slots, bars, etc., to confine and direct radiation *(e.g., x-rays or gamma-rays)* to a specific region and / or to discriminate against radiation from unwanted directions *(e.g., scattered radiation)* is called:

- Discrimination
- Collimation
- Filtration
- Coning

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• **Collimation**

Collimation refers to the control of the size and shape of x-ray beam.

In x-ray imaging systems, a collimator mounted to the x-ray tube is used to define the dimensions of the beam which is to be incident on the subject and the detector . To minimize radiation dose and to comply with government regulations, a certain level of precision must be maintained.

It is a basic rule of radiation hygiene that the radiation beam be **as small as practical**. For intraoral radiography, by state law, the diameter of a circular beam of radiation at the patient's skin can be **no greater than 2.75 inches.** One can use a diaphragm or metal cylinders, cones or tubes to collimate the beam.

These devices **do not reduce** the amount of radiation received by the exposed tissues, **but reduce** the radiation to surrounding tissues due to x-ray beam divergence.

Remember → The x-ray beam consists of many **different wavelengths.** The **short wavelength** *(high energy)* rays have great penetrating power; **long wavelength** *(low energy)* rays have low penetrating power and do not reach the film in reasonable quantities since they are attenuated by the soft tissues. Low energy rays add only to the total amount of radiation the patient receives. **Aluminum discs** are used to filter out the useless long wave rays, **increasing the overall quality of the beam.**

Important: Filtration **reduces** patient dose, **decreases** contrast and **decreases** the density of the film.

A herringbone or diamond effect will appear on the processed film when:

- The film is bent
- The film is placed backwards in the mouth
- An improper vertical angulation is used
- An improper horizontal angulation is used

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• **The film is placed backwards in the mouth** *(See figure #1)*

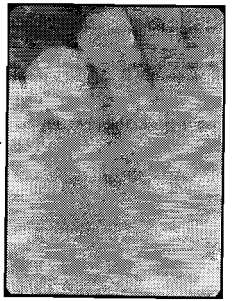

Figure #1. A reversed film appears light with a herring-bone effect.

Figure #2. The film demonstrates a double exposure

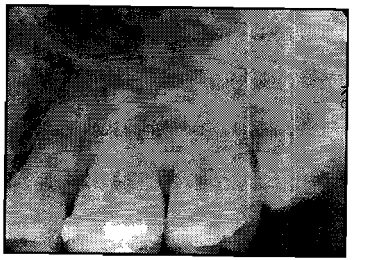

Figure #3. The bent film appears distorted.

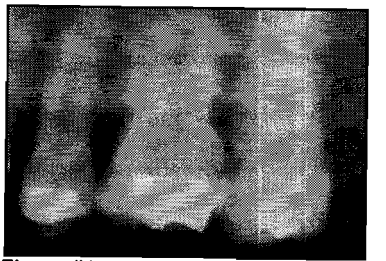

Figure #4. Movement results in a blurred image.

Image magnification may be minimized by:

- Using a short cone
- Placing the film as far from the tooth as possible
- Using a long cone
- Shortening the exposure time

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- **Using a long cone**

Five rules for accurate image formation when taking x-rays:

1. Use the **smallest focal spot** that is practical.
   Note: The size of the focal spot influences radiographic definition or sharpness. They are inversely proportional. The operator **cannot** control the size of the focal spot.

2. Use the **longest source-film distance** that is practical in the particular situation.

3. Place the film **as close as possible** to the structure being radiographed.

4. Direct the central ray at **as close to a right angle** to the film as anatomical structures will allow.

5. As far as is practical, **keep the film parallel** to the structure being radiographed.

Foreshortening and elongation are produced by:

- Incorrect horizontal angulation
- Incorrect vertical angulation
- Either of the above

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• **Incorrect vertical angulation**

Vertical angulation is directing x-rays so that they pass vertically through the part being examined. This is accomplished by positioning the tubehead and direction of the central ray in an **up-and-down** *(vertical)* plane. **Important: Foreshortening** (See *figure #1)* refers to a shortened image and **elongation** *(See figure #2)* refers to an elongated image. Both are produced by an **incorrect vertical angulation.** Excessive vertical angulation causes foreshortened images, while insufficient vertical angulation causes elongated images.

Figure #1. If the vertical angulation is too steep, the images are foreshortened.

Figure #2. If the vertical angulation is too flat, the images are elongated.

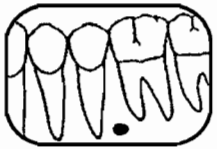
Horizontal angulation is maintaining the central ray at 0 degrees as the tube is moved around the head. This is accomplished by positioning the tubehead and direction of the central ray in a **side-to-side** *(horizontal)* plane. **Note:** The general rule for horizontal angulation is that the central ray should be **perpendicular** to the mean anteroposterior plane of the teeth being x-rayed. **Important:** Incorrect horizontal tube angulation causes **overlapping** *(teeth images are superimposed on each other).*

The **central ray** is said to be at **0 degrees** when the x-ray tube is adjusted so that the central ray is parallel to the floor. If the tubehead is directed at the floor, it is called **positive angulation;** if it is directed toward the ceiling, it is called **negative angulation**.

The two radiographs below were taken with the **buccal object rule** in mind. In film #2, the x-ray tube was directed from a mesial angulation. What is the **spacial position** of the circular object in these radiographs?

- The object lies **lingual** to the first molar
- The object lies **buccal** to the first molar
- The object lies **between** the second premolar and the first molar
- The object lies **directly apical** to the first molar

Film #1



Film #2

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• **The object lies lingual to the first molar**

The **buccal object rule** *(also called the tube shift technique)* is used to determine an object's spacial position within the jaws. This technique utilizes two radiographs of an object exposed with slightly different tube angulations. It then compares the object's position on the radiograph with respect to a **reference point** *(e.g., the* **root of a tooth***)*.

If the tube is shifted and directed from a **more mesial** direction, and the object in question appears to have moved **mesially** with respect to the reference point, then the object lies **lingual** to that reference point. Conversely, if the tube is shifted mesially and the object in question moves **distally**, it lies on the **buccal** aspect of the reference object.

Remember the acronym **SLOB** → **S**ame-**L**ingual, **O**pposite-**B**uccal.

***If the object in question appears to move in the **same direction** as the x-ray tube, it is on the **lingual** aspect. If it appears to move in the **opposite direction** as the x-ray tube, it is on the **buccal** aspect.

Which of the following errors in radiographic technique is the **most likely** reason that an image on a radiograph would appear **elongated?**

- Too much vertical angulation
- Too little vertical angulation
- Incorrect horizontal angulation
- Beam not aimed at center of film

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- **Too little vertical angulation**

Some errors often made when taking dental radiographs:

- **Elongation** *(most common error)* → **teeth appear too long** → may be caused by **too little** vertical angulation, the film not parallel to the long axis of the teeth or the occlusal plane not being parallel to the floor.
- **Foreshortening → teeth appear too short** → may be caused by **too much** vertical angulation or poor chair position.
- **Cone cutting → portion of film will appear clear with a curved line** → the beam was not aimed at the center of the film. **See figure #1.**
- **Herringbone effect → zigzagged pattern appears on the film** → the film was placed backwards in the mouth.
- **Poor film placement →** the film was not placed far enough back or not forward enough in the mouth. **See figure #2.**
- **Overlapping → interproximal areas are overlapped, reduces diagnostic quality of film** → due to incorrect horizontal angulation *(the central x-ray was not directed perpendicular to the curvature of the arch and through the contacts).* **See figure #3.**

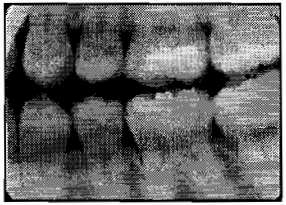


Figure #1. A cone-cut appears as a curved unexposed *(clear)* area on a radiograph.

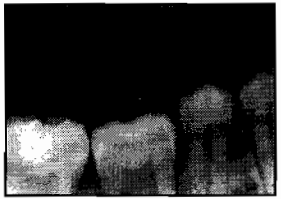


Figure #2. Improper film placement: no apices appear on this film.

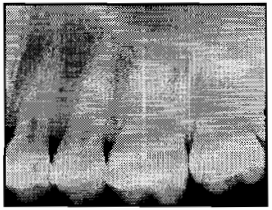
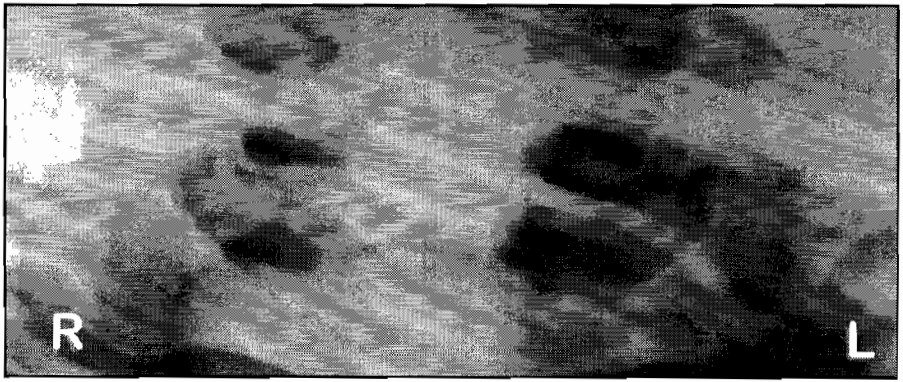


Figure #3. Incorrect horizontal angulation results in overlapped contact areas.

Which of the following positioning errors is the most likely cause of the **reverse occlusal plane** curve on the panorex below?

- Chin tilted too far upward
- Chin tilted too far downward
- Head turned slightly



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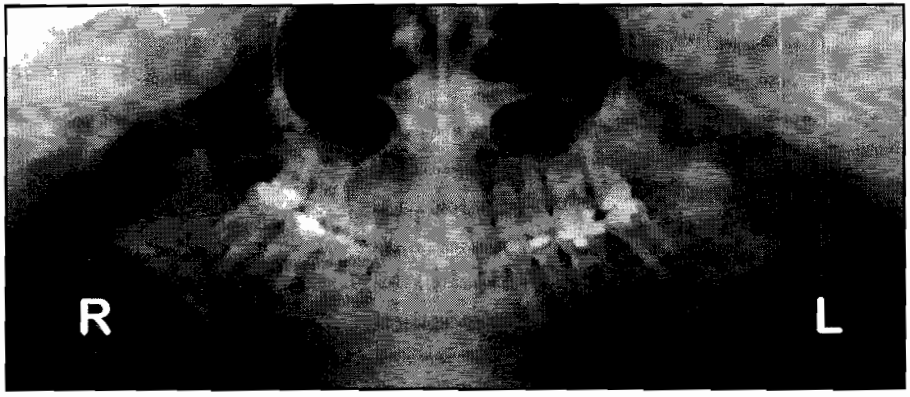
• **Chin tilted too far <u>upward</u>**

***Mandibular structures look narrower and maxillary structures look wider *(looks like a "frown")*.

Chin tilted too far <u>downward</u>:

1. Occlusal plane shows an excessive upward curve *(looks like a "big smile")*. **See figure below.**
2. Severe interproximal overlapping, anterior teeth appear very distorted



The periapical x-ray below appears distorted. What is the **most likely** cause of this?

- Overbent film
- Patient had glasses on
- Exposure to secondary radiation
- Cone cutting
- X-ray arm drifted

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- **Overbent film** → cracked emulsion

Some other common **errors** made when taking dental radiographs → **cause:**

- **Light films** *(underexposed / image NOT dense enough)* → incorrect milliamperage *(too low)* or exposure *(too short)*; incorrect focal film distance; cone too far from patient's face, film placed backwards. **See figure # 1.**
- **Dark films** *(overexposed / image too dense)* → incorrect milliamperage *(too high)*, exposure *(too long)*, incorrect kVp *(too high).* **See figure # 2.**
- **Double exposure** → film was used twice.
- **Fogged films** → exposed to radiation other than primary beam. **See figure # 3.**
- **Artifacts** → patient didn't remove eyeglasses, earrings, or removable prosthetic appliances.
- **Poor contrast** → incorrect kVp *(too high).*
- **Blurred image** → patient movement or drifting of x-ray arm.
- **Clear films** → were not exposed to radiation.

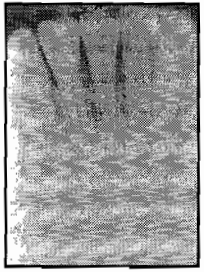

Figure #1. An underexposed film appears light.

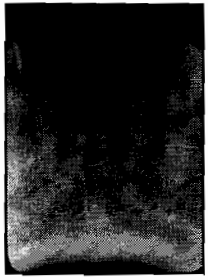

Figure #2. An overexposed film appears dark.

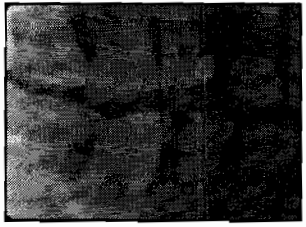

Figure #3. A fogged film appears gray and lacks detail and contrast.

Which of the following is a **major disadvantage** of the **paralleling technique?**

- The image formed on the film will **not** have dimensional accuracy
- Due to the amount of distortion, periodontal bone height **cannot** be accurately diagnosed
- An **increase** in exposure time is necessary due to the use of a **long** cone
- An **increase** in exposure time is necessary due to the use of a **short** cone

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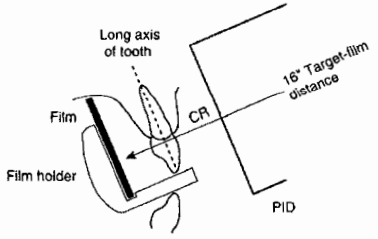
- **An increase in exposure time is necessary due to the use of a long cone**

The **paralleling technique** is based on the concept of parallelism. Other names for this technique include XCP *(extension cone paralleling technique)*, right-angle technique, and long-cone technique.

Basic Principles:

- Film is placed parallel to the long axis of the tooth being x-rayed.
- Central x-ray is directed perpendicular to both the film and the long axis of the tooth.
- A film holder *(XCP)* **must** be used to keep the film parallel to the long axis of the tooth.
- The **object-film distance** must be **increased** to keep the film parallel. This results in image magnification and loss of definition.
- The **source-film distance** must also be **increased** to compensate for the image magnification and to make sure that only the **most parallel rays** will be aimed at the tooth and the film. Using a **long** cone *(16 inch target-film distance)* results in greater definition and less image magnification.



Positions of the film, teeth, and central ray of the x-ray beam in the paralleling technique. The film and long axis of the tooth are parallel. The central ray is perpendicular to the tooth and film. An increased target-film distance *(16 inches)* is required.

Which of the following is **not a disadvantage** of the **bisecting technique?**

- Image on x-ray film may be dimensionally distorted *(amount may vary)*
- Increased exposure time
- Due to the use of a short cone *(which results in divergent rays)*, the image is not a true reproduction of the object
- May not be able to judge the correct alveolar bone height

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- **Increased exposure time**

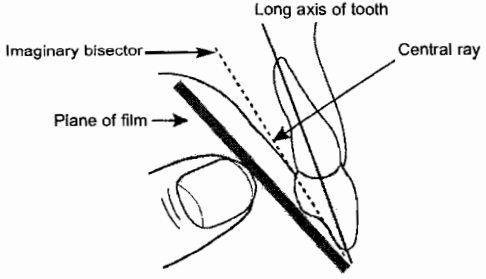
***The exposure time is **actually decreased.**

The **bisecting angle technique** is based on the geometric principal known as the rule of isometry. The rule states that two triangles are equal if they have two equal angles and share a common side.

The following best describes the **bisecting technique:**

- The dental x-ray film is placed along the **lingual surface** of the tooth.
- At the point where the film contacts the tooth, an **angle** is formed by the plane of the film and the long axis of the tooth.
- The person taking the x-ray needs to visualize a **plane** that **bisects** this **angle.** This plane is called the imaginary bisector → this creates two equal angles and provides a common side for the two imaginary equal triangles.
- The central ray is positioned **perpendicular** to the imaginary bisector.

Important: When this technique is **followed strictly,** the image of the tooth produced is accurate.



Name the two x-ray projection techniques used in dental radiography.

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1. **Bisecting technique (See Figure #1)**
2. **Paralleling technique (See Figure #2)**

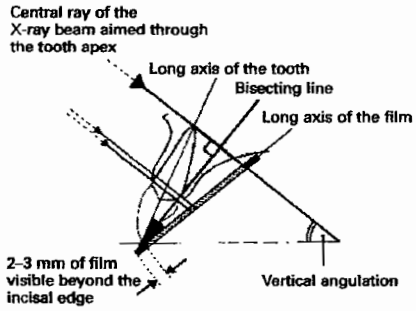

Figure #1. Theoretical basis of the bisected angle technique. The angle between the long axes of the tooth and film is bisected and x-ray beam aimed at right angles to this line, through the apex of the tooth. With this geometrical arrangement, the length of the tooth in the mouth is equal to the length of the image of the tooth on the film, but as shown, the periodontal bone levels will not be represented accurately.

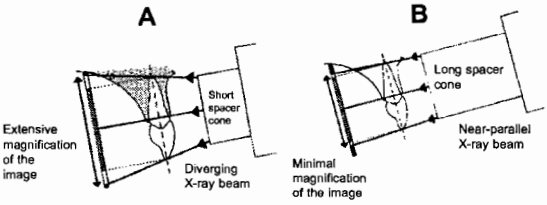

Figure #2. Diagrams showing the magnification of the image that results from using **(A)** a short cone and a diverging x-ray beam and **(B)** a long cone and a near-parallel x-ray beam.

Which of the following projections is best for examination of fractures of the zygomatic arch?

- Waters projection
- Submentovertex projection
- Reverse Towne projection
- Lateral cephalometric projection

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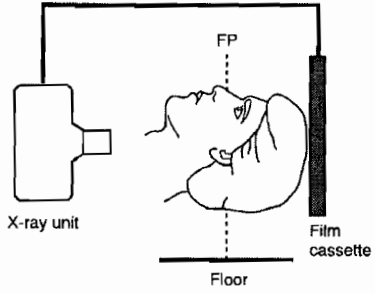
• **Submentovertex projection**

For this projection the neck is maximally extended and the film cassette touches the top of the head. The x-ray beam enters the head under the chin *(near the mental tubercle of the mandible)* and exits at the vertex. This view is used in conjunction with other projections, and allows direct visualization of the base of the skull. The **zygomatic arches** stand out like the handles of a jug on this view.



Which of the following projections is best for examination of the maxillary sinus?

- Lateral jaw projection
- Reverse Towne projection
- Waters projection
- Submentovertex projection

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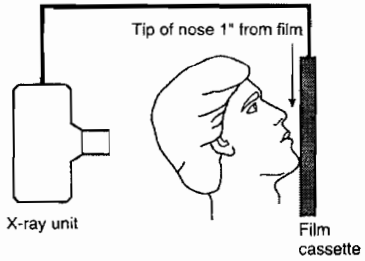
• **Waters projection**

This is a posterior-anterior projection with the patient's face lying against the film and the x-ray source behind the patent's head. Waters' projection is the most useful conventional radiographic technique to image the **maxillary sinuses.** In this projection, the radiographic densities of normal maxillary sinuses are the same on both sides and equal to those of the orbits. If one of the sinuses is diseased, Waters projection will exhibit either a radiopaque *(fluid)* level, a sinus opacification, mucosal hyperplasia, a radiopaque growth or a loss of cortical borders of sinus. Other useful projections include periapical, panoramic, occlusal, lateral head, and Caldwell. It is also one of the best films for radiographic diagnosis of **mid-facial fractures.**



Which of the following projections is best for examination of the **condyles** and neck of the mandible *(from an anterior-posterior projection)*?

- Waters projection
- Transcranial projection
- Townes projection
- Submentovertex projection

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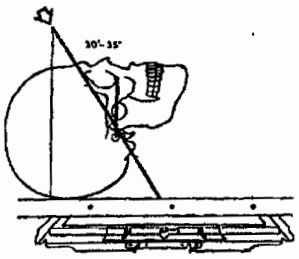
• **Townes projection**

The patient **lies on his back** with the film under his head. The x-ray source is from the front, but rotated 30 degrees from the Frankfort plane and is directed right at the condyles.

The **Townes projection** is often of value in assessing the status of the condyles, condylar neck and rami because superimposition of the mastoid and zygoma over the condylar neck region in the straight postero-anterior projection often makes interpretation difficult. The Townes projection eliminates this superimposition, thus giving **good visualization of the condylar area and rami.**

Note: The **"reverse Towne projection"** is used to identify **fractures of the condylar neck and ramus area.**



The following can be demonstrated on conventional **TMJ radiographs:**

- Position of the condyles in the glenoid fossa
- The range of antero-posterior movement of the condyles
- Areas of bone destruction on condylar heads

All of the following are **advantages** of a panoramic radiograph **except:**

- It shows areas that may not be visible on a full mouth series
- It shows both arches on the same film
- It gives better detail and definition than periapical radiographs
- It is more comfortable for the patient *(eliminates gagging)*
- It requires less time than a full mouth series

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- **It gives better detail and definition than periapical radiographs**

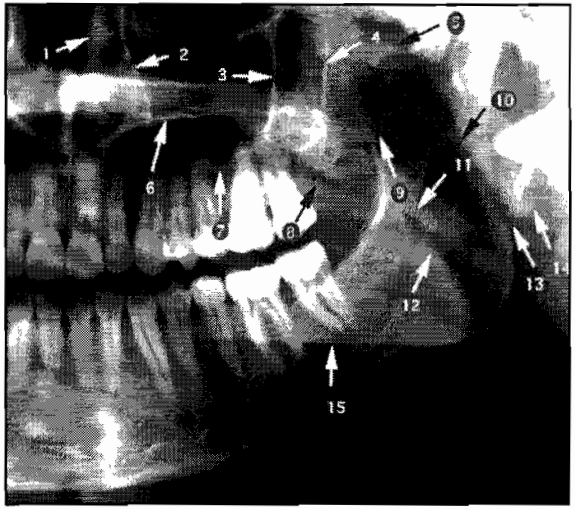
***This is **false;** a panoramic radiograph gives **less detail and definition** than periapical radiographs due to intensifying screens, movement of the x-ray tube and film and increased object-film distance.

Indications for a panoramic radiograph:
- Diagnosis of oral pathology that may not be visible on periapical radiographs
- Treatment planning *(especially orthodontic cases)*
- Evaluation of anomalies
- As one part of the follow-up evaluation in surgical and trauma cases
- Edentulous patients *(prior to constructing full dentures)*
- Patients that are unable to tolerate intra-oral x-rays

***The drawback of a panoramic radiograph is that there is a loss of image detail *(it is hard to diagnose early carious lesions).* Bite-wing x-rays are required for the diagnosis of carious lesions.

Other **disadvantages** of a panoramic radiograph:
- Distortion of image due to increased object-film distance
- Inadequate for interproximal caries detection or for detecting periodontal breakdown *(bone loss)*
- Proximal overlapping *(especially in premolar and molar areas)*
- Added exposure to a large area of body tissue, in addition to the oral tissues

Identify each structure numbered in the partial panoramic radiograph below?



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1. **The opaque mass** → Inferior concha
2. **The opaque line** → Medial wall of maxillary sinus
3. **The opaque line** → Posterior wall of zygomatic process of maxilla
4. **The opaque line** → Posterior wall of maxillary sinus
5. **The opaque mass** → Zygomatic arch
6. **The opaque line** → Hard palate / floor of nasal fossa
7. **The opaque line** → Floor of maxillary sinus
8. **The line of contrast** → Dorsum of tongue
9. **The opaque line of contrast** → Inferior border of pterygoid plates
10. **The vertical line of contrast** → Posterior wall of nasopharynx
11. **The opaque mass** → Soft palate
12. **The line of contrast** → Dorsum of tongue
13. **The opaque mass** → Calcified stylo-hyoid ligament
14. **The opaque mass** → Ear lobe
15. **The line of contrast** → Inferior border of opposite mandible

The panoramic radiograph is excellent for **third molar pathology** as well as to observe the TMJ, the sinuses, and sialography *(which is a technique used in radiology in which a salivary gland is filmed after an opaque substance is injected into its duct).* A sialolith which is located in Wharton's duct, however, can best be viewed by using a cross-sectional occlusal x-ray.

A phenomenon caused by a relatively lower x-ray absorption on the mesial or distal aspect of teeth, between the edge of the enamel and the adjacent crest of the alveolar ridge is called:

- Apical burnout
- Cervical burnout
- Coronal burnout
- Root burnout

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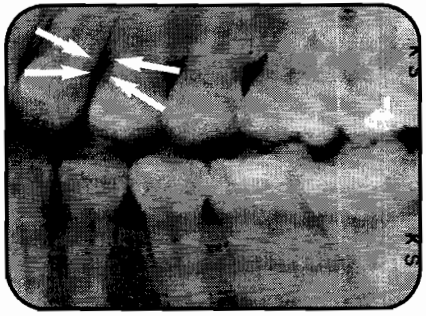
• **Cervical burnout**

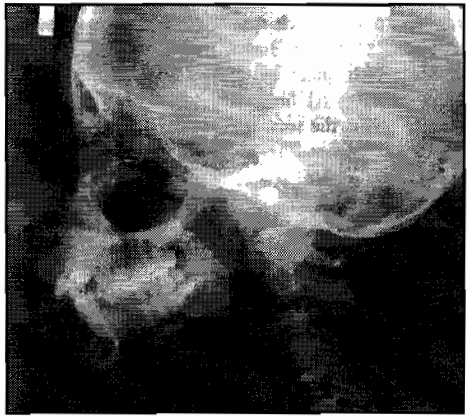
Because of the relative diminished x-ray absorption, these areas appear relatively radiolucent with ill-defined margins.

It is caused by the normal configuration of the affected teeth (*the cementoenamel junction),* which results in decreased x-ray absorption in those areas.

Important: These radiolucencies should be anticipated when viewing x-rays of almost any tooth and **should not be mistaken** for a carious lesion.



Identify this view? What are its **indications?** How is the patient **positioned?**



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- **Lateral cephalometric**
- **Evaluation of growth and development of face**
- **Left side near film, 60 inches between anode and sagittal plane, soft tissue filter near source**

The **lateral cephalometric** x-ray must be compared with "normal" lateral radiographs from an accepted norm. **Linear and angular measurements** are obtained utilizing known anatomical landmarks in the lateral head radiography of the patient. These measurements are then compared with those considered within normal limits and in that way enable the orthodontist to assess aberrations in the dentition and jaw structures which result in mal-occlusion.

Analysis of cephalometric radiographs is **not limited** to the hard structures such as bone and teeth, but also includes measurements of soft tissue structures such as the nose, lips, and soft tissue chin.

Superimposition in longitudinal cephalometric studies is generally on a **reference plane and a registration point.** This will best demonstrate the growth of structures farthest from the plane and the point. The most stable area from which to evaluate craniofacial growth is the **anterior cranial base** because of its early cessation of growth.

Cephalometrics are useful in **assessing** tooth-to-tooth, bone-to-bone, and tooth-to-bone relationships. Serial cephalometric films can show the amount and direction of growth.

Which of the following types of intra-oral radiographs are **most** useful in detecting **interproximal caries?**

- Periapical radiographs
- Bitewing radiographs
- Occlusal radiographs

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• **Bitewing radiographs**

***These x-rays show the crowns of both Max. and Mand. teeth; **not** root apices.
The **primary reason** for taking bitewing radiographs is to detect **interproximal caries.** They are also useful in monitoring the progression of periodontal disease. These films show crestal bone levels as well as interproximal areas of both arches. In order for the film to be of diagnostic use, the quality of the following must be excellent: dimensional accuracy, open contacts, and optimum contrast and clarity of the image.

When taking bitewing radiographs, the film must be placed in either **a horizontal or vertical position.** Vertical bitewings provide more periodontal information, such as bony defects and furcation involvement. A fuzzy or indistinct image of crestal bone is often associated with early periodontitis. Two bitewings are usually taken on a child, one on each side. If the child has primary dentition only, number "0" film is used. If the child has mixed dentition, number "1" film is utilized. Once the individual has second molars, two to four number "2" films are conventionally utilized. If using four films, one film images the premolar area, while the other images the molar area. Sometimes two, long, number "3" films are utilized *(one for each side)* instead of two number "2" films on each side. This practice is not recommended due to the curvature of the arch making it difficult to open all contacts on one film.

Notes:

1. **The vertical angulation** for bitewing radiographs should be between +8 and +10 degrees.
2. **Adjust horizontal angulation** to direct the central ray toward the center of the film.
3. **Alveolar bone resorption** is **best** demonstrated on bitewing x-rays.
4. **Vertical bitewing x-rays** will show **more alveolar bone** than traditional horizontal bitewings.
5. The **largest** intraoral film size is # "4".
6. The **standard** film size is # "2".
7. The ideal **temperature and humidity** levels for film storage is 50 to 70° F and 30 to 50%.

The period between radiation exposure and the onset of symptoms is called the:

- Latent period
- Period of cell injury
- Recovery period

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• Latent period

The **latent period** is the period of time between radiation exposure and the onset of symptoms. It may be short or long, depending on the total dose of radiation received and the amount of time it took to receive the dose.

The **period of cell injury** follows the latent period. Cellular injury may result in cell death, changes in cell function or abnormal mitosis of cells.

The **recovery period** is the last event in the sequence of radiation injury. Some cells recover from the radiation injury, especially if the radiation is **"low level".**

Note: The effects of radiation exposure are **additive** and the damage that remains unrepaired accumulates in the tissues. The cumulative effects of repeated radiation exposure can lead to various serious health problems (*e.g.*, ***carcinogenesis****, which leads to various carcinomas, genetic mutations which cause birth defects, different kinds of leukemia and cataracts).*

Which type of **digital image receptor** is most common at this time?

- CID *(Charge Injection Device)*
- CMOS / APS *(Complementary Metal Oxide Semiconductor / Active Pixel Sensor)*
- CCD *(Charge-Coupled Device)*

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• **CCD** ***(Charge-Coupled Device)***

To produce a direct digital x-ray image, three components are necessary: an x-radiation source, a sensor, and a computer. The images are captured using a solid-state detector or sensor such as a charge-coupled device *(CCD)*, a complementary metal oxide semiconductor / active pixel sensor *(CMOS / APS)*, or a charge injection device *(CID)*. Most direct digital systems use a CCD device. CCD, CMOS, and CID sensors are referred to as "wired" because they are linked by a fiberoptic cable to the computer. The sensor itself is basically a silicon chip with an electronic circuit on it.

The **CCD** is the most common device used today. Its sensor is about the same size as a #2 intra-oral film and is connected to the computer by an optic fiber wire in most cases. Microwave technology has been used with CCDs, eliminating the need for the wire tether, but requires additional electronic components. The CCD consists of a silicon chip with an active array of rows and columns called pixels *(picture elements)*. These pixels are analogous to silver crystals in conventional films but are 80% more sensitive to radiation, thus the reduction in radiation dose to patients. Smaller pixel sizes mean more pixels fit onto the sensor, which decreases the size of the receptor but increases the cost. CCDs are available in large enough sizes to accommodate panoramic films, and manufacturers have made CCDs that can be retrofitted to existing units to ease the cost of equipment conversion. With cephalometric imaging, a CCD large enough to capture an entire skull is too expensive to manufacture and sell, so a different technology has been employed that involves a CCD array and a scan lasting several seconds.

The main advantage of digital radiography over traditional film is that **digital images are available immediately.** This saves time in treatment requiring progress films or if a shot needs to be retaken. Patient education is improved by an image displayed immediately on a chairside monitor, rather than making the patient leave the chair to squint into a viewbox at a miniscule shadow. The ability to enhance an image, particularly by increasing brightness or contrast, is a tremendous benefit to the practitioner, since often a film is not viewed until after the patient has left the office.

All of the following are advantages of digital radiography **except:**

- Digital subtraction
- The ability to enhance the image
- Size of the intraoral sensor
- Patient education

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• **Size of the intraoral sensor**

Many advantages have been ascribed to digital radiography. First, they allow a **reduction in the amount of radiation reaching the patient,** which is always a concern. With direct systems, images are **displayed immediately** on the computer monitor so less chair time is required during diagnosis and treatment appointments. This is a particularly attractive feature for clinicians performing endodontic treatment where several images are usually made during an appointment. Clinicians also find it useful to be able to manipulate the image because it **enhances diagnosis.** Image storage and electronic transmission are possible, and patients can be **educated** about their diagnosis and treatment using the images. Many users appreciate the fact that there is **a lot less mess** associated with producing digital images than conventional ones, because there is no need for film, film processors, processing chemicals, darkrooms, or film mounts.

Of course, no technology is without some **disadvantages.** Commonly mentioned ones for digital radiography include the high initial set-up cost, the need for staff training, and the bulkiness *(i.e., thickness)* of the sensors. **Note:** The sensor itself is basically a silicon chip with an electronic circuit on it. Sensors range in thickness from 3.2 mm to 8.8 mm.

The method of obtaining a digital image similar to scanning a photograph to a computer screen is termed:

- Indirect digital imaging
- Direct digital imaging
- Storage phosphor imaging
- CMOS / APS

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• **Indirect digital imaging**

There are three options available for capturing a digital x-ray image: indirect, direct, and storage phosphor imaging.

Indirect digital x-ray images are produced by placing a conventional x-ray film on a desktop scanner and allowing a transparency adapter to shine light through the image as it is scanned into the computer. This converts the original analog image *(i.e., dental radiograph)* into a digital image by scanning. Once digitized, the image can be processed like any other digital image.

To produce a **direct** digital x-ray image, three components are necessary: an x-radiation source, a sensor, and a computer. The images are captured using a solid-state detector or sensor such as a charge-coupled device *(CCD)*, a complementary metal oxide semiconductor / active pixel sensor *(CMOS / APS)*, or a charge injection device *(CID)*.

A third method of obtaining a digital image is **storage phosphor imaging,** a wireless digital radiography system. In this system, a reusable imaging plate coated with phosphors is used instead of a sensor with a fiber optic cable. The plates are described as "wireless" because they are not connected via cable or wire to the computer. The plates are similar in every way to conventional intraoral film, including size, thickness, rigidity and placement. These plates store the energy from incoming x-rays, and are then placed in a scanning device. The scanner stimulates the stored x-ray information by subjecting the plate to a laser light. When the light strikes the phosphor material, energy is released as a light signal in an electronic waveform and is converted to a digital image by the computer. The image can not instantaneously be viewed on the monitor, but takes from 30 seconds to 5.5 minutes depending upon the system and certain variables.

Digital radiography requires **less radiation** than conventional radiography because:

- The sensor is larger
- The exposure time is increased
- The sensor is more sensitive to x-rays
- The pixels sense transmitted light quickly

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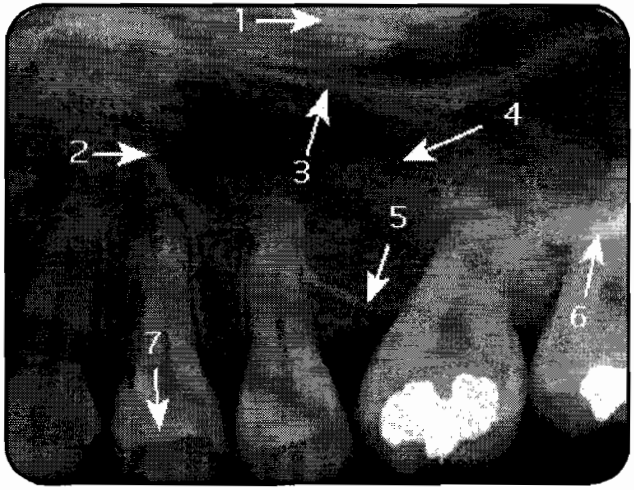
• **The sensor is more sensitive to x-rays**

One of the positive features of digital radiography is that it requires less radiation than conventional radiography, because the sensor is more sensitive to x-rays than dental film. Exposure times for digital radiography are from **50% to 80% shorter** than those for E-speed film. This translates into less radiation exposure for the patient.

Notes:

1. All direct and PSP digital radiography systems use a conventional dental x-ray unit. The literature emphasizes that the x-ray unit must have the ability to reduce exposure times to 0.01 seconds to reduce the likelihood of oversaturating the sensor.
2. In digital radiography, a sensor, or small detector is placed inside the mouth of the patient to capture the radiographic image. The sensor is used instead of intraoral film. As in conventional radiography the x-ray beam is aimed to strike the sensor. An electronic charge is produced on the surface of the sensor; this electronic signal is digitized, or converted into "digital" form.
3. Digital radiography systems are not limited to intraoral images; panoramic and cephalometric images may also be obtained.

Identify each structure that the corresponding arrows point to in the **maxillary premolar** region.



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1. **The opaque mass** → Inferior concha

2. **The opaque line** → Anterior wall of maxillary sinus

3. **The opaque line** → Floor of nasal fossa

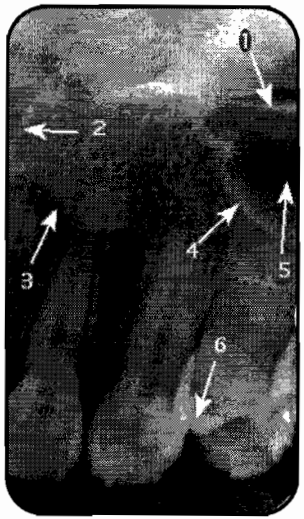
4. **The radiolucent space** → Maxillary sinus

5. **The opaque line** → Floor of maxillary sinus

6. **The opaque structure** → Inferior border of zygomatic process of the maxilla

7. **The opaque line** → Lingual cusp of first premolar

Identify each structure that the corresponding arrows point to in the **maxillary canine** region.



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1. **The opaque line** → Floor of nasal fossa
2. **The opaque line** → Lateral wall in nasopalatine canal
3. **The opaque line** → Ala of nose
4. **The opaque line** → Anterior wall of maxillary sinus
5. **The radiolucent space** → Maxillary sinus
6. **The opaque line** → Lingual cusp of 1st premolar

Identify each structure that the corresponding arrows point to in the **maxillary molar** region.

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1. **The opaque line** → DEJ

2. **The lucent line** → Periodontal ligament space

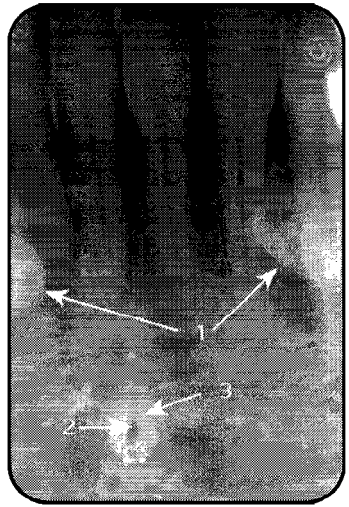
3. **The opaque line** → Lamina dura

4. **The lucent line** → Periodontal ligament space of palatal root

5. **The opaque spot** → Film holder

6. **The opaque region** → Mucosa over maxillary ridge

Identify each structure that the corresponding arrows point to in the **mandibular incisor** region.



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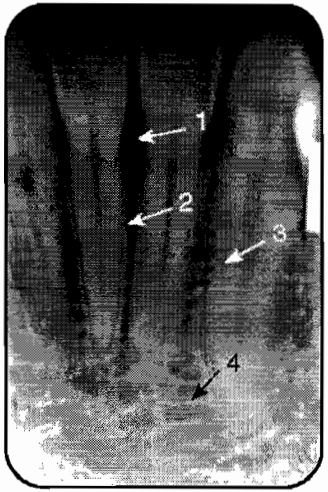
1. **The radiopaque masses** → Mandibular tori

2. **The radiolucent circle** → Lingual foramen

3. **The radiopaque mass** → Genial tubercles

Identify each structure that the corresponding arrows point to in the **mandibular incisor / canine** region.



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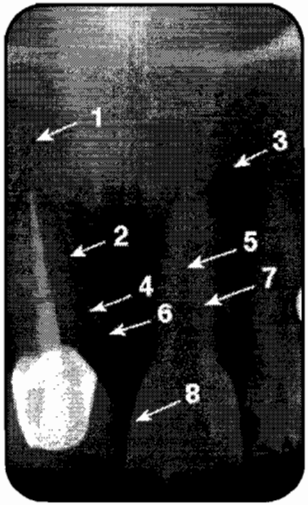
1. **The radiopaque structure** → Alveolar crest

2. **The radiopaque line** → Lamina dura

3. **The radiolucent line** → Periodontal ligament space

4. **The radiopaque line** → Bony trabecular plate

Identify each structure that the corresponding arrows point to in the **maxillary incisor** region.



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1. **The radiolucent space** → Marrow space

2. **The radiolucent line** → Periodontal ligament space

3. **The radiopaque line** → Bony trabecular plate

4. **The radiopaque line** → Lamina dura

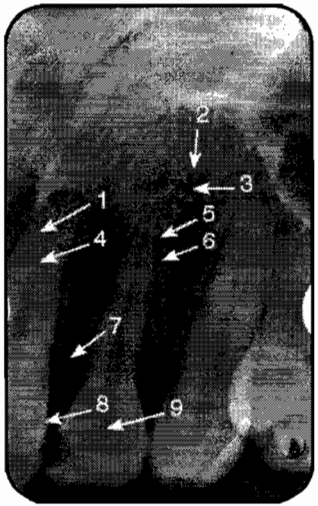
5. **The lucent line** → Pulp canal

6. **The opaque structure** → Alveolar crest

7. **The opaque structure** → Dentin *(root)*

8. **The opaque structure** → Enamel of crown

Identify each structure that the corresponding arrows point to in the **maxillary incisor** region.



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1. **The opaque material** → Dentin

2. **The radiolucent line** → Bony trabecular plate

3. **The radiolucent space** → Bony marrow space

4. **The lucent structure** → Pulp canal

5. **The lucent line** → Periodontal ligament space

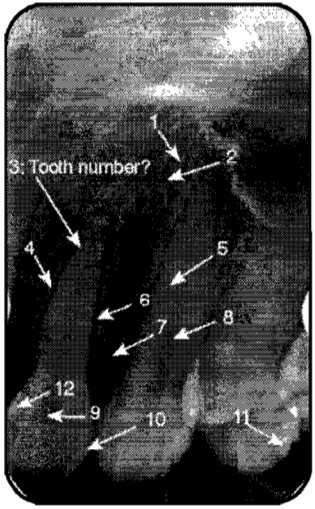
6. **The opaque line** → Lamina dura

7. **The opaque structure** → Alveolar crest

8. **The opaque structure** → Enamel

9. **The lucent structure** → Pulp chamber

Identify each structure that the corresponding arrows point to in the **maxillary canine** region.



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1. **The opaque line** → Trabecular plate

2. **The lucent space** → Marrow space

3. **Tooth number?** → 10

4. **The opaque line** → Lamina dura

5. **The opaque material** → Dentin

6. **The radiolucent line** → Periodontal ligament space

7. **The opaque structure** → Alveolar crest

8. **The radiolucent structure** → Pulp canal

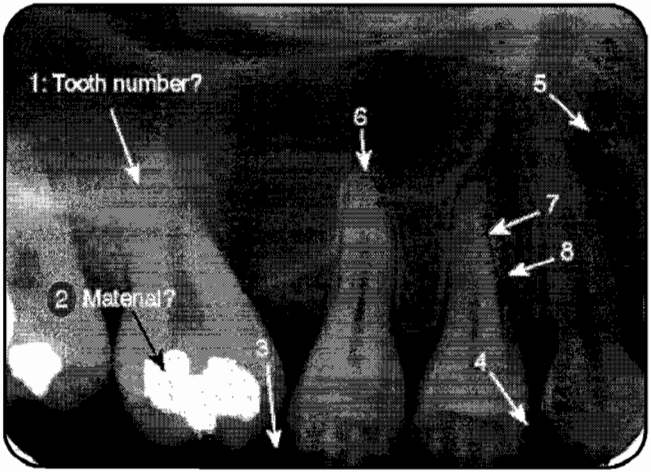
9. **The radiolucent structure** → Pulp chamber

10. **The opaque material** → Enamel

11. **The opaque circle** → Premolar buccal cusp over raised film dot

12. **The opaque line** → DEJ

Identify each structure that the corresponding arrows point to in the **maxillary premolar** region.



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1. **Tooth number?** → 3

2. **What material is this?** → Silver amalgam

3. **What is this opacity?** → Plastic bite block

4. **The black dot** → Film dot

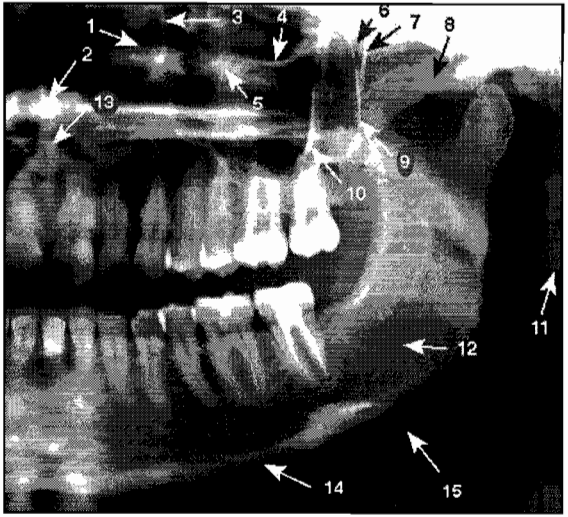
5. **The black marks** → PLS for Kodak Ektaspeed plus film

6. **The opaque line** → Lamina dura

7. **The lucent line** → Periodontal ligament space

8. **The opaque line** → Lamina dura

Identify each structure that the corresponding arrows point to in the **partial panorex.**



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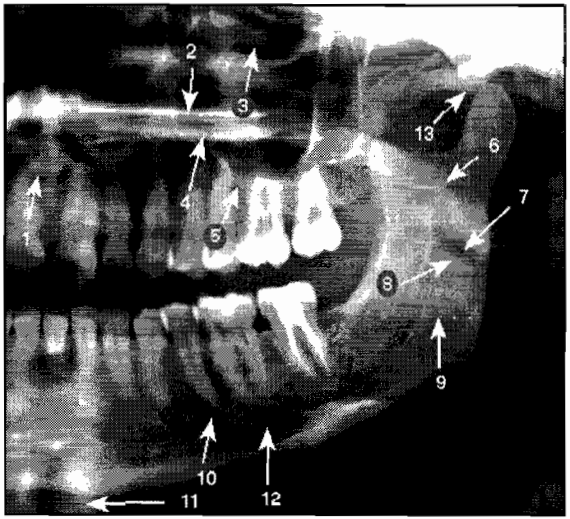
1. **The lucent space** → Air in nasal fossa
2. **The opaque line** → Nasal septum
3. **The opaque line** → Lateral wall of nasal fossa, medial wall of maxillary sinus
4. **The opaque line** → Infraorbital rim
5. **The opaque line** → Border of infraorbital canal
6. **The radiolucent space** → Pterygomaxillary fissure
7. **The opaque line** → Pterygoid spine of sphenoid bone
8. **The opaque mass** → Zygomatic arch
9. **The opaque line** → Posterior wall of maxilla *(maxillary sinus)*
10. **The opaque line** → Posterior wall of zygomatic process of maxilla
11. **The opaque mass** → Ear lobe
12. **The opaque line** → Inferior border of mandibular canal
13. **The opaque mass** → Anterior nasal spine
14. **The opaque line** → Inferior border of mandible
15. **The opaque mass** → Hyoid bone

Identify each structure that the corresponding arrows point to in the **partial panorex.**



"Courtesy Dr. Stuart C. White, UCLA School of Dentistry."

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1. **The opacity** → Tip of nose

2. **The opaque line** → Hard palate / floor of nasal fossa

3. **The lucent area** → Orbit

4. **The opaque line** → Hard palate / floor of nasal fossa

5. **The opaque line** → Floor of maxillary sinus

6. **The opaque structure** → Soft palate

7. **The radiolucent space** → Air between the soft palate and the dorsum of tongue

8. **The opaque line** → Dorsum of tongue

9. **The opaque line (dots)** → Shadow of opposite mandible

10. **The lucent oval** → Mental foramen

11. **The diffuse opacity** → Shadow of cervical spine

12. **The broad lucency** → Submandibular gland fossa

13. **The opacity** → Articular tubercle

ENDODONTICS

Legend

Major Topic	Abbreviation
Diagnostic Methods / Tests	Diag Meth
Individual Teeth	Ind Tth
Instruments / Materials / Techniques	Inst / Mat / Tech
Miscellaneous	Misc.
Pulp	Pulp
Replantation *Intentional & *Avulsed Teeth	Replant
Resorption	Resorp
Terms / Conditions	Terms / Cond

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Which of the following are **useful diagnostic aids** that can be used to determine if a tooth has a **vertical crown-root fracture?**

- **Fiberoptic light** for transillumination
- **Wedging** the tooth in question and then taking an x-ray
- **Persistent periodontal defects** in an otherwise healthy tooth
- Having a patient **bite forcefully on a bite stick**
- All of the above

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• **All of the above**

Important: Radiographs *(without first wedging the tooth)* **rarely** will show vertical fractures.

Vertical fractures will often be recognized radiographically by their effect on the bony attachment apparatus that is seen as a diffuse radiolucency or "halo" surrounding the root. This can be differentiated from other periapical radiolucencies because it surrounds the tooth uniformly rather than being located at the portal of exit of the apical foramen or lateral canal.

Notes

1. A tooth with a **vertical fracture** through root structure has a **poor prognosis.**
2. Studies have indicated that **most** vertical root fractures are caused by **too much condensation** force during obturation with gutta-percha.

Therapy for **horizontal fractures** of the root always involves considerable difficulty. Root canal treatment is **not indicated** if the fracture sites remain in close proximity and if the pulp retains its vitality. **However**, if clinical symptoms develop or the segments appear to be separating according to the x-ray, some treatment is necessary.

Remember: Root fracture can only be visualized on a radiograph if the x-ray beam passes through the fracture line. As the fracture line could extend diagonally, an additional radiograph is taken with a 45° *(steep)* vertical angulation in addition to the conventional 90°.

An **electric pulp tester** *(EPT or Vitalometer)* usually elicits a response at a higher current than normal if a tooth, being tested has:

- Acute pulpitis
- Chronic pulpitis
- Hyperemia
- Open apex
- A pus-filled canal
- Suffered a trauma

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• **Chronic pulpitis**

EPT checks the sensibility of a tooth by stimulating nerve endings with a **low current and high potential difference** in voltage. Although manufacturers of this device give normal reference values of current, the best way to check "normal / baseline" values is to use it on adjacent *(non-pathological)* teeth. This is then compared with the values obtained on the tooth being questioned. The EPT uses electrical excitation to stimulate the **A-delta sensory fibers** in the pulp. A positive response does not provide any information about the health or integrity of the pulp; it simply indicates that there are **vital sensory fibers** present. The EPT fails to provide any information about the **vascular supply** to the pulp, which is the true determinant of pulp vitality.

Note: EPT is not considered reliable in the following conditions.

1. A pus-filled canal → false positive
2. A nervous patient → false positive
3. Recent dental trauma → false negative
4. Insulating restoration → false negative
5. Secondary dentin deposits
6. Moisture contamination
7. Immature tooth *(open apex)*
8. Patient who has taken analgesics
9. Improper application / weak batteries in EPT

Important: Never wear gloves while using the EPT as this impedes completion and results in a false-negative response.

Response to EPT:
Acute pulpitis → lower than normal current, as acute inflammation mediators lower the pain threshold.
Hyperemia → lower than normal, but higher than that seen in acute pulpitis.
Pulp necrosis / Abscess → no response at any current-level.

According to the **buccal object rule,** when the x-ray tube is repositioned either at a more mesial or at a more distal angulation and a film is exposed, the root or canal farther from the film *(the buccal)* will:

- Move in the opposite direction that the cone is directed
- Move in the same direction that the cone is directed
- Not move at all

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• **Move in the same direction that the cone is directed**

Therefore, when the cone is **aimed to the distal** *(angled from the mesial direction)* the **buccal root or canal moves to the distal** and appears distal to the lingual or palatal root *(or canal)*.

Note: In order to apply this rule, you must have a **reference object.**

When treating **multicanaled bicuspids and molars**, it is often difficult to ascertain on the radiograph which canal is more toward the buccal. When a straight-on exposure is taken of a bicanaled tooth, the canals become superimposed on the film, and visualization of each canal is impossible. If the x-ray cone is moved to give an angled exposure, the roots will be separate on the film.

By applying the buccal object rule you will be able to determine which canal is the buccal and which is the lingual.

Another way to explain this shift-cone technique is a **SLOB (S**ame **L**ingual, **O**pposite **B**uccal) rule; the object toward the lingual side *(closer to the film)* will appear to shift on the film to the same direction as the repositioned X-ray cone. → if the X-ray cone is mesially angulated, the lingual / palatal object *(root)* will shift toward the same *(mesial)* side in the resultant radiograph film, and thus easily visualized.

Note: Using this technique you can determine:
1. Working length of superimposed canals.
2. Curvatures of root / canals.
3. Facial-Lingual orientation of instruments, or other anatomical objects.

Explain the diagnostic tests that are **indicated** for teeth that have recently **been traumatized.**

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• **The dental examination should include:**

Soft tissue exam → observe the lips, face, tongue, etc.

Hard tissue exam → visually look and then **palpate** the injured tooth and alveolus to reveal the extent of tooth mobility as well as alveolar fractures and area of inflammation. Check for occlusal disharmonies to help detect tooth displacements and jaw fractures.

Radiographic examination → x-rays reveal tooth displacement and root fractures as well as other important facts *(previous root canal, periapical radiolucencies, etc.).*

Other diagnostic tests → pulp vitality testing is **contraindicated** because the traumatized pulp undergoes a temporary paresthesia and would give a false reading. The percussion test **is not** usually performed, since it is painful.

Observe the adjacent and opposing teeth for injury.

Teeth that have been **traumatized** may be fine for a long time, however, many will develop radiolucencies. Do not indiscriminately do root canals **without first** checking pulp vitality, and perform root canal therapy only in those teeth that do not respond to pulp testing. **Example:** Trauma to maxillary anterior teeth. A few years later x-rays reveal radiolucencies around the region of the apices of the incisors. Check the pulp vitality of all anterior teeth before performing root canals.

Note: Trauma *(causing deep intrusion)* to a permanent tooth will most likely result in **necrosis of the pulp** and conventional root canal therapy will be necessary.

A **trapezoidal outline** of the pulp chamber is characteristic of:

- Maxillary molars
- Maxillary premolars
- Mandibular molars
- Mandibular premolars
- Mandibular canines

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• **Mandibular molars**

Usually the trapezoidal outline is formed by two canals in the mesial root and one oval canal in the distal root. In approximately **40%** of the cases the distal root may have a second canal *(fourth canal overall)*. The pulp chamber is located in the mesial two-thirds of the crown. You must look for the **fourth canal** if the first-found canal in the distal root lies more toward the buccal, instead of being located in the center.

Note: The **lingual** wall of mandibular teeth is **most easily perforated** when preparing an access opening due to the lingual inclination of these teeth.

Remember: The mandibular first molar requires endodontic treatment more frequently than any other tooth in the oral cavity.

Maxillary molars have a **triangle** outline of the chamber:
– The base of it is formed by the buccal canals, the apex by the palatal canal
– The line connecting the mesial with the palatal canal is the longest
– If a fourth canal is present, it is usually located lingual to the orifice of the mesiobuccal canal, and in the mesiobuccal root. It is much more common than previously thought.

Note: The mesiobuccal of the maxillary molars is the the most complex root in the entire dentition because 90% have either second canals or major fins leading off of the mesiobuccal canal.

The maxillary **first** molar is the posterior tooth with the **highest endodontic failure rate.** The lingual or palatal root is the longest, has the largest diameter, and offers the easiest access. The clinician should always assume there are two canals in the mesiobuccal root until it is proven there is only one.

***Maxillary anterior teeth** have a single root and canal.

Which of the following teeth **most often** refer pain to the temporal region?

- Mandibular molars
- Maxillary incisors
- Maxillary second premolars
- Maxillary molars

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• **Maxillary second premolars**

If careful diagnosis **does not reveal** the affected tooth, other teeth and related anatomic structures become suspect. Pulpitis in one tooth may cause pain in other areas → **the pain is referred.**

Site of Pain Referral	Tooth Pulp Causing Pain
Forehead region	Maxillary incisors
Nasolabial area	Maxillary canines, premolars
Temporal region	Maxillary second premolars
Ear	Mandibular molars
Mental region of mandible	Mandible incisors, canines, and premolars

Important: The nerve endings of cranial nerves VII, IX, and X are widely distributed within the subnucleus caudalis of the trigeminal *(V)* nerve. A profuse intermingling of these nerve fibers creates the **potential for the referral of dental pain to many sites.**

Which tooth will **almost always** have two canals?

- Maxillary **first** premolar
- Maxillary **second** premolar
- Mandibular **first** premolar
- Mandibular **second** premolar

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• **Maxillary first premolar**

Approximately 60% have two roots, one buccal and the other palatal, each with a single canal. The two roots may be completely separate or merely twin projections rising from the middle third of the root to the apex *(this is more common)*. The two roots are usually equal in length from apex to cusp. However, the lingual root and canal may be wider.

In approximately 40% of **maxillary first premolars, only one root is present, usually with two separate canals**. A cross section at the cervical line shows a canal shaped like a figure eight *(ellipse)*. The access opening is a thin oval. **Be careful not to perforate on the mesial** *(the concavity on the mesial makes perforation very common)*.

The apical foramen of the **maxillary first premolar** is usually close to the anatomic apex, and the apical portion of the roots often taper rapidly, ending in extremely narrow and curved root tips. The buccal root can fenestrate through the bone, leading to problems such as inaccurate apex location, chronic post-operative sensitivity to palpation over the apex, and increased risk of an irrigation accident. This tooth is also prone to mesiodistal root fractures and fractures at the base of the cusps, especially the buccal cusp.

Maxillary second premolars: The most common configuration in this **tooth is a single root,** occurring approximately 85% of the time. Approximately 15% of the time, two separate roots are present, each with a single canal. The access opening is **exactly the same** as that for maxillary first premolars *(thin oval)*.

Notes:

1. **When only one canal is present** *(first or second premolar)*, it is usually found in the center of the access preparation. If only one canal is found, **but** it is not in the center of the tooth, **it is probable that another canal is present.**
2. **Overfilling either tooth** may force materials directly into the maxillary sinus.

Which of the following canals in a **maxillary first molar** is usually the most difficult to locate?

- Palatal
- Distobuccal
- Mesiobuccal
- All of the canals are relatively easy to find

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• **Mesiobuccal**

Canal orifices of a **maxillary first molar** are arranged in the shape of a triangle. The orifice to the **mesiobuccal canal is usually the most difficult to locate, since it is under the mesiobuccal cusp and must be entered from a distolingual position.** This canal is the small canal and often splits into two canals. It may be calcified and difficult to instrument. The palatal canal is the straightest, widest, and most tapering canal. The most common curvature of the palatal root is to the facial. The **distobuccal** canal is also small and tapering. The orifice to this canal has no direct relation to its cusp. The distobuccal orifice is usually located by means of its relation to the mesiobuccal orifice, with the distobuccal found approximately 2 to 3 mm to the distal and slightly to the palatal aspect of the mesiobuccal orifice.

Note: In approximately 59% of maxillary first molar teeth, **a fourth canal is present with its orifice being just lingual to the orifice of the mesiobuccal canal.** The canal is located in the mesiobuccal root and may join the mesiobuccal canal or exit through a separate foramen. If a lesion is present on the mesiobuccal root prior to root canal therapy and doesn't heal in the usual amount of time *(6-12 months)* following treatment, it is most likely due to a missed canal *(mesio-lingual).*

Fracture of the maxillary first molar is usually through the central groove or at the base of the buccal cusp. These fractures can extend into the furcation, creating an untreatable periodontal defect.

Remember: The **U-shaped radiopacity** commonly seen overlying the apex of the palatal root of the maxillary first molar is most likely the **zygomatic process** of the maxilla.

Which tooth may have a pulp chamber that is **somewhat triangular** as opposed to oval?

- Maxillary central incisor
- Mandibular central incisor
- Maxillary lateral incisor
- Mandibular lateral incisor

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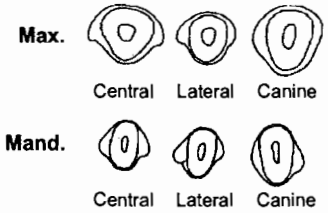
• **Maxillary central incisor**

The **base of the triangle** will be the facial. The **apex** will be the lingual. If it is **not triangular,** then it will be oval.

Over 60% of maxillary central incisors show accessory canals, and the apical foramen is found apart from the apex in 45% of these teeth.

Ideal access preparation of maxillary central incisors is a rounded-triangle shape on the lingual surface of the tooth with a slight curve lingually to avoid reducing the incisal edge.

The cervical cross sections below of the anterior teeth show the relationship of the crown outline to the pulp chamber and the root canal.



Approximately what percentage of mandibular first premolars may have **two canals** with **two apical foramina?**

- 5%
- 25%
- 45%
- 65%

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• **25%**

***One fourth of all mandibular first premolars may have two canals with two foramina.

The treatment of mandibular first premolars can really be tricky! At least 23% may have two or three canals starting anywhere down the root. This is quite different from the mandibular second premolar – 97% are found to have one canal at the apex.

The **second premolar** has few variations than the first premolar, usually having one root and one well-centered canal. The access opening is oval. Consideration must be given to the **mental foramen** which lies in close proximity to the apex. Avoid over-instrumentation and overfill. When viewing an x-ray of this area, the mental foramen is sometimes misdiagnosed as a premolar abscess. Therefore, before performing root canal therapy, **make sure all diagnostic tests confirm your finding.**

Note: If a straight-on preoperative radiograph of a mandibular first premolar shows the pulp canal disappearing in midroot, **this is an important indication that two canals are present.**

Remember: Maxillary second premolars have a higher incidence of accessory canals *(60%)*, than do maxillary first premolars.

The root canal for a **mandibular canine** is:

- Wide mesiodistally but thin labiolingually
- Thin mesiodistally but wide labiolingually
- The same width mesiodistally and labiolingually

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• **Thin mesiodistally but wide labiolingually**

Mandibular canines usually have only **one root** but in rare cases may have two separate roots. The access opening is a **large oval** with the greatest width placed incisogingivally.

This tooth usually has **a slightly labial axial inclination of the crown,** therefore the **access opening** needs to be **directed towards the lingual surface.**

The canal of the mandibular canine is somewhat ovoid at the cervical area but it becomes rounder at the apex.

Which of the following teeth is **most likely** to have a curved root?

- Maxillary central incisor
- Maxillary lateral incisor
- Maxillary canine
- Mandibular central incisor

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• **Maxillary lateral incisor**

The maxillary lateral incisor **always has one root with one canal.** The root is more slender than in the maxillary central incisor and frequently *(55%)* has a distal and / or lingual curvature or dilaceration. The access opening is oval.

Maxillary central incisor: The maxillary central incisor **always has one root and one canal.** The root is bulky, with a slight distal axial inclination but rarely has a dilaceration. The access opening is **oval-triangular.**

Maxillary canine: The maxillary canine always has one root and one canal. This tooth is the longest in the arch. The access opening is **oval.**

Note: The maxillary central, lateral, and canine roots and hence, canals **all have a distal axial inclination**. This means in penetrating along the long axis of the tooth, the bur must be slightly angled toward the distal surface. **Failure to do this may lead to perforation of the mesial portion of the root.**

Mandibular central incisor: The mandibular incisors *(laterals and centrals)* have **only one root which is narrow mesiodistally but relatively wide labiolingually** and may have a distal and / or lingual curvature. **Two canals may be present.** When there are two canals, the **labial canal is the straighter one**. The access opening for a mandibular central or lateral is a **long oval,** with the greatest width placed incisogingivally and the incisal extent very close to the incisal edge.

Which of the following are related to **vital teeth** and usually do not warrant endodontic therapy?

- Apical scar
- Cementoma
- Traumatic bone cyst
- Globulomaxillary cyst
- Radicular cyst
- Chronic dental abscess
- Chronic periapical granuloma

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- **Cementoma**
- **Traumatic bone cyst**
- **Globulomaxillary cyst**

An **apical scar** is represented by a periapical granuloma, cyst, or abscess that heals with scar tissue. Well-circumscribed radiolucency resembling a granuloma. Tooth is **non-vital**.

A **radicular cyst** usually occurs in a pre-existing granuloma. Seldom is painful. Radiolucency at apex of **non-vital tooth.**

A **chronic dental abscess** is often a result of a periapical granuloma. Radiolucent area at apex of **non-vital tooth.** Fistula is often found leading from an abscess cavity. Once drainage is established, the tooth stops being painful. **A chronic periapical abscess is often the cause of a sinus tract in the gingival tissues of children.**

A **chronic periapical granuloma** is the **most common sequelae of pulpitis.** It is asymptomatic and associated with a **non-vital tooth.**

A **cementoma** occurs most frequently in the **anterior region of the mandible**. It starts as a radiolucent lesion and then calcifies. The cementoma **does not affect pulp vitality.** Also called periapical cemental dysplasia.

A **traumatic bone cyst** is not a true cyst since there is no epithelial lining. Found mostly in young people, asymptomatic. Radiolucency which appears to scallop around the roots of teeth. Teeth are **usually vital**.

A **globulomaxillary cyst** is found at the junction of the globular and maxillary processes of the maxilla, between the lateral incisor and the canine roots. **Teeth are vital.**

The most superior of all other retro-fillings material → **mineral trioxide aggregate** *(MTA)* has all of the following advantages, **except** two. Which two **are not** properties of MTA?

- Radiopaque
- Easy to manipulate
- Hydrophilic
- Biocompatible
- Not toxic
- Short setting time
- Induction of hard tissue formation

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- **Easy to manipulate**
- **Short setting time**

The main ions found in MTA are calcium and phosphorous. MTA has a **high pH** so it induces hard tissue formation. MTA has superior sealing ability and is **not adversely** affected by blood contaminants. It also causes only **low levels** of inflammation because it forms fibrous connective tissue and cementum when in contact with the periodontium. **Note:** MTA is **difficult** to manipulate and has a **long** setting time. Despite these disadvantages, it's the material of choice today.

A **retrofilling** *(also called a reverse filling or retrograde amalgam filling)* is placed to seal the apical portion of the root canal. This procedure is used **when an apicoectomy alone will not yield a good result.** Whenever there is any chance whatsoever that an apical seal may be faulty, **a reverse filling material must be placed.** For example, if the root canal appears calcified, it would be impossible to obturate most of the canal and get a seal. If just the root apex were cut off *(apicoectomy),* the incompletely filled canal might act as a source of reinfection. To prevent this after the root tip is resected, the foramen is found, enlarged, and filled with a zinc-free amalgam to create a seal.

An **apicoectomy** *(root resection, root amputation)* is a procedure where the buccal tissue is flapped back, the buccal bone about the apex is removed, **the root apex is removed**, and the area is curetted out. **Indications for apicoectomy**: 1. A **reverse filling** needs to be placed. 2. It is **necessary** to gain access to an area of pathosis. 3. The **poorly filled apical portion** of the root is to be removed to the level of canal obliteration. **Note:** A retrograde amalgam filling **should always** be done after an apicoectomy. Teeth that have **posts** in them and need to be retreated are the **most common reason** for an apicoectomy and a retrograde filling.

Remember: Periapical curettage is the same procedure as an apicoectomy *(as far as flap and removal of buccal bone)* but without removing the root apex. Removal and examination of the diseased tissue and determination of the extent of the lesion are the objectives of apical curettage.

The earliest and **most common** symptom associated with an inflamed pulp is:

- A dull throbbing pain on mastication
- Sensitivity to hot, and or, cold stimuli
- A persistent feeling of discomfort
- Mild bleeding
- Pain on percussion

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• **Sensitivity to hot, and or, cold stimuli**

***Thermal sensitivity** is the earliest and most common symptom of an inflamed pulp.

As caries enters the dentin it begins with a lateral spread at the DEJ. This is due to the increased organic content and the involvement of many dentinal tubules. The Tomes fibers react, causing fatty degeneration, then later decalcification *(sclerosis)*. As caries progresses, destruction of dentin is followed by the bacterial invasion of the tubules and complete destruction of dentin. Once odontoblasts are **involved, pulpal changes occur.** Initially there is vascular dilation and local edema. The **earliest common symptom** of this edema *(acute pulpitis)* is **thermal sensitivity** *(usually increased and persistent pain on application of cold)*.

Remember: The only reliable clinical evidence that secondary dentin has formed is **decreased tooth sensitivity** *(usually seen a few weeks after placement of a filling)*. When dentinal tubules become completely calcified, the dentin is insensitive.

Notes:

1. The best method to elicit a most accurate thermal response is to individually isolate the suspected teeth with a rubber dam and then bathe each tooth in hot or cold water. This is done because all other methods may stimulate the tooth at only one section of one surface.
2. Thermal tests may be false-negative in immature, recently traumatized teeth or because of premedication with an analgesic.

Which condition is an apical lesion that develops as an acute exacerbation of a **chronic apical abscess** *(also called a suppurative apical periodontitis)*?

- Cyst
- Phoenix abscess
- Granuloma
- None of the above

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• **Phoenix abscess**

It is also known as a **recrudescent abscess.** It develops as the granulomatous zone becomes contaminated or infected by elements from the root canal. Diagnosis is based on the acute symptoms *(pain to percussion)* plus radiographic examination, which **reveals a large periapical radiolucency. Note:** A phoenix abscess is **always preceded** by chronic apical periodontitis. Signs and symptoms are identical to those of an acute periradicular abscess, but a radiograph will show a periapical radiolucency that indicates the presence of a chronic disease.

A **granuloma** is defined as a growth of granulomatous tissue continuous with the periodontal ligament resulting from pulpal death with diffusion of toxic products into the periapical area. In most cases a **granuloma is symptomless.** Radiographically, one sees a well-defined area of rarefaction with some irregularities, while clinically the tooth is not sensitive. A massive invasion of pulpal contaminants **will result** in the formation of an acute abscess *(Phoenix abscess).*

A **cyst** is an inflammatory response of the periapex, which **develops from preexisting granulomatous tissue** *(granuloma).* It is characterized by a **central, fluid-filled, epithelium-lined cavity,** surrounded by granulomatous tissue and peripheral fibrous encapsulation. It is often associated with a chronically infected tooth. The tooth may be mobile. On radiographs, one will see a **well-defined area of rarefaction** *(radiolucency)* which is limited by a continuous radiopaque, sclerotic border of bone. It is usually **asymptomatic.**

Remember: A granuloma or a cyst can only be **differentially diagnosed** by histological examination.

The **chronic apical abscess** *(CAA)* is generally:

- Very painful
- Asymptomatic
- Mildly painful

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• **Asymptomatic**

The **chronic apical abscess** *(also called suppurative apical periodontitis)* is sometimes so painless that it may go undetected for years until revealed by an x-ray. It is a long-standing, low-grade infection of the periapical bone with the root canal being the source of the infection. This condition may follow an acute alveolar abscess or unsatisfactory root canal therapy. Radiographs will reveal a diffuse radiolucency and PDL thickening. The tooth may be slightly loose or tender to percussion. The chronic abscess may be differentiated from cysts and granulomas by the fact that both cysts and granulomas have well-defined radiolucencies associated with them. **The treatment is conventional root canal treatment.**

Remember: 30% to 50% of bone calcium must be altered before radiographic evidence of periapical breakdown occurs *(this alteration takes place at the junction between the cortical and cancellous bone).*

The **acute apical abscess** *(AAA)* is a localized collection of pus in the alveolar bone at the root apex following death of the pulp with extension of the infection into the periapical tissue. The first symptom may be a slight tenderness of the tooth. This later develops into a **severe throbbing pain to percussion with swelling** of the overlying mucosa. The tooth becomes more painful, elongated and loose. At times the pain may decrease or disappear completely. The patient may appear weakened, irritable and present with a fever. The diagnosis is based on the history, exam, and radiographs. The tooth will **not respond** to the EPT or cold test but **may respond** to heat. Treatment of an acute alveolar abscess includes establishing drainage and debriding the canal system of necrotic tissue which will relieve the acute symptoms. This is followed at a later date by conventional root canal therapy.

Note: If the abscess ruptures through the periosteum into the soft tissue, the patient's symptoms will subside.

The most common cause of **acute osteomyelitis** of the jaws is:

- Unknown
- Iatrogenic
- Dental infection
- Radiation

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• **Dental infection**

It is **not a particularly** common disease. It is a serious sequela of periapical infection that often results in a diffuse spread of infection throughout the medullary spaces, with subsequent necrosis of a variable amount of bone.

Acute or **subacute** osteomyelitis may involve either the maxilla or the mandible. In the **maxilla**, the disease usually remains fairly well-localized to the area of initial infection. In the **mandible**, bone involvement tends to be **more diffuse** and **widespread.**

Clinically, the person afflicted with **acute** osteomyelitis is usually in rather **severe pain** and manifests an elevation of temperature with regional lymphadenopathy. The teeth in the area of involvement are loose and sore so that eating is difficult, if not impossible. **Note:** Another clinical symptom of acute osteomyelitis is **leukocytosis,** an elevated number of white cells in the blood.

Radiographically, acute osteomyelitis progresses **rapidly** and demonstrates little radiographic evidence of its presence until the disease has developed for at least one to two weeks. At that time, diffuse lytic changes in the bone begin to appear. A "moth-eaten" radiolucent appearance is evident.

The general principles of treatment demand that **drainage** be established and maintained and that the infection be treated with antibiotics to prevent further spread and complications.

Which condition is the **result** of a pulpal infection that extends through the apical foramen to the periapical tissues?

- Periodontal abscess
- Gingival abscess
- Periapical abscess

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• **Periapical abscess**

Of all the dental abscesses, the periapical is the **most common** type. It is a localized collection of pus in the alveolar bone at the root apex following death of the pulp with extension of the infection into the periapical tissue. The first symptom may be a slight tenderness of the tooth. This later develops into a severe throbbing pain *(acute abscess)* with swelling of the overlying mucosa. The tooth **will not respond** to the EPT or cold tests but may respond to heat. Emergency treatment includes establishing **drainage** *(ideally through the canal)* and prescribing antibiotics and analgesics. This will relieve the acute symptoms followed by conventional endodontic therapy at a later date. For endodontic infections that do not respond to penicillin, clindamycin is often recommended. It produces high bone levels and is effective against anaerobic bacteria but must be used with caution because of the potential for pseudomembranous colitis.

The **periodontal abscess** is an acute abscess that develops through the periodontal pocket. Alveolar bone loss, pocket formation and periodontal pathologic conditions are suggestive of the periodontal abscess. The tooth will usually be palpation and percussion positive. It **will respond** to the electric pulp tester *(unlike the periapical abscess)*. Bacteria associated with this abscess include gram-negative rods such as Capnocytophaga species, Vibrio-corroding organisms and Fusobacterium species.

The **gingival abscess** is a relative rarity that occurs when the bacteria invade through some break in the gingival surface. Such abrasions may be the result of mastication, oral hygiene procedures, or dental treatment.

Which condition is characterized by pain that is **spontaneous** and has periods of cessation *(intermittent in nature)*?

- Reversible pulpitis
- Irreversible pulpitis

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• **Irreversible pulpitis**

The severity of the clinical symptoms will vary as the inflammatory response increases. Pain will vary from a mild and readily tolerated discomfort to a severe, throbbing and excruciating pain. The pain is **spontaneous** and is **intermittent** in nature. The pain **lingers after the removal** of the irritant. The pain is usually not readily localized by the patient but is diffuse in character. Lying down or bending over intensifies the pain of irreversible pulpitis because the overall increase in cephalic blood pressure is relayed to the confined pulp tissue. The tooth may be tender to percussion, heat may intensify the pain response while cold may relieve it *(in advanced stages).* Usually they both will cause severe and lasting pain. The radiographs will **usually** disclose **no periapical pathology.** Treatment is root canal therapy. **Note:** In cases of **irreversible** pulpitis, an acutely inflamed pulp is symptomatic whereas a chronically inflamed pulp is asymptomatic in most cases.

Reversible pulpitis *(hyperemia)* → the pain associated with hyperemia **does not occur** spontaneously. It requires an external irritant to evoke a painful response *(i.e., cold, sweets).* The pains are **sharp** and of **brief duration,** ceasing when the irritant is removed. Radiographs appear normal *(may show deep caries or cavity preparation).* The tooth is usually percussion negative. In thermal tests, the pulp responds more readily to cold stimuli than to hot *(the response leaves shortly after removal of the stimulus).* **Treatment usually is a sedative filling or new restoration with a base.**

Causes of **reversible** pulpitis include early caries, periodontal scaling, root planning, microleakage, and restorations placed without a base. Reversible pulpitis is not a disease, rather it is a symptom.

Note: Pulpal inflammation *(hyperemia)* is most commonly caused by **bacteria.**

A seven year old boy arrives at the office with a complaint of sinus discharge from an earlier traumatized permanent maxillary central incisor. Vitality tests are negative. The required treatment would be:

- Extraction
- Apexogenesis / pulpotomy
- Root canal treatment
- Periodontal surgery to remove sinus
- It is only necessary to give the child analgesics and antibiotics for pain and infection
- None of the above

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• **None of the above**

Apexification is a technique whose goal is to **induce further root development** in a pulpless tooth by stimulating the formation of a hard substance at the apex, so as to allow obturation of the root canal space. Apexification may be required after pulpectomy as at seven years of age the apex of this tooth must be open. **Remember:** Apex closes 2-3 years after eruption.

The technique consists of isolation of the field with a rubber dam, making an access cavity and removing all pulpal tissue by the use of reamers and files. A premixed syringe of a **calcium hydroxide-methylcellulose paste** *(for example, a Pulpdent syringe)* is injected into the canal until it is filled to the cervical level. The paste must reach the apical portion of the canal to stimulate the tissues to form a calcific barrier. A double seal of cement is made to close off the access cavity. The patient is recalled after three months to see if apexification has taken place. If not, a fresh supply of paste is placed. If apexification has occurred, conventional root canal therapy is instituted.

The action of calcium hydroxide in **promoting formation** of a hard substance at the apex is **best explained** by the fact that calcium hydroxide creates an alkaline environment that promotes hard tissue deposition.

Note: If a permanent tooth fractures and has a fully formed root and the pulp is exposed *(large exposure)*, the treatment of choice is **complete root canal therapy.** Apexification is **not needed** because the root is fully formed. If the exposure is small and the length of time is short *(1/2 hour to 1 hour)*, then a direct pulp cap with CaOH followed by a restoration is the treatment of choice.

While doing **vital pulpotomy** on a young, immature permanent tooth, the hemorrhage after pulp amputation could not be controlled with cotton pellets even after several minutes. What is the next step in completing this treatment?

- Control the hemorrhage with hemostatic agents
- Apply formocresol with cotton pellets at the amputation site
- Irrigate the canal with sodium hypochlorite then apply calcium hydroxide
- Perform the amputation at a more apical level
- Stop the procedure and close the tooth with an interim restoration
- All of the above

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• **Perform the amputation at a more apical level**

***Uncontrolled bleeding is a sign of inflamed pulp tissue. The radicular pulp must be uninflamed for the success of this procedure. It is not uncommon to find uninflamed pulp at a more apical level, especially in cariously exposed teeth. If bleeding does not stop even after more apical amputation, hemostatic agents are used as a compromise treatment. These are closely monitored and if vitality is lost, apexification *(pulpectomy)* procedures should be instituted.

Pulpotomy is removal of a portion of the pulp. The **common indications** include:
• Cariously exposed deciduous teeth → with healthy radicular pulps.
• Traumatic or carious exposure of permanent teeth with undeveloped roots.
• An alternative to extraction when endodontic treatment is not available.
• Emergency treatment in permanent teeth with acute pulpitis.

Unfortunately, pulpotomy procedures performed in fully developed permanent teeth are **not found** to be successful. For this reason it is regarded as a temporary procedure in these teeth.

Which **two** of the following situations **offer better success** for pulp capping?

- Accidental exposure of the pulp
- Pulp of a middle-aged person
- Carious exposure of the pulp
- Pulp of a young child

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- **Accidental exposure of the pulp**
- **Pulp of a young child**

Pulp capping is the placing of a **sedative** and antiseptic dressing on an **exposed healthy pulp** in order to allow it to recover and maintain normal function and vitality. The dressing **most commonly used** is $CaOH_2$ *(Dycal)*. Pulp capping is overused in dentistry today. In reality it has only very few indications for its use. **Young** pulps are more vascularized and, therefore, more amenable to repair. Pulp cappings **are more successful** if the exposure was accidental *(trauma or with a dental bur)* as opposed to carious. In addition, the exposure should only be pinpoint to expect success. Repair is accomplished by the formation of a **dentin bridge** at the site of exposure. Even a small carious exposure should have root canal therapy for the **best long-term prognosis.**

Direct pulp capping is indicated if there is a small mechanical exposure *(or small traumatic exposure)*, an asymptomatic vital pulp, and no coronal or periapical pathology.

A tooth may stay asymptomatic for several weeks after pulp capping has been performed. However, this may be only temporary. Unfortunately, if pulp capping fails and the tooth becomes symptomatic, it may be difficult, **if not impossible,** to treat with routine endodontics because of the severe calcifications in the root canal. Perforations may occur during attempts to follow the obliterated canal to gain patency to the apex. **Note:** Perforations into furcations of multi-rooted teeth have the poorest prognosis.

Indirect pulp capping involves removing infected dentin almost up to the point of pulpal exposure. Calcium hydroxide is placed and then a resin modified glass ionomer cement is placed over that. Formation of secondary dentin should occur and then a final restoration is placed after removal of the intermediate restoration and residual caries. The goal of indirect pulp capping is to have the tooth participate in its own recovery. **Indications for indirect pulp capping** include deep carious lesions that encroach but are not actually in the pulp, no history of chronic pain, no radiographic pathology, vital pulp, and normal tooth mobility and color.

Which of the following is **not** an indication to use solvent-softened custom cones?

- "Tugback" within 1 mm of working length
- Lack of an apical stop
- An abnormally large apical portion of the canal
- An irregular apical portion of the canal
- After an apexification procedure

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• **"Tugback" within 1 mm of working length**

***Since studies show that solvent softening **does not** ultimately result in a better apical seal, this time consuming procedure can be reserved for the other indications listed on the front of the card. This **slight resistance to dislodgement** is referred to as **"tugback."** The cone should also have a definite apical seat → it should not be able to be pushed further apically.

If the preparation is **properly flared,** fitting the master cone is not a time-consuming procedure. A gutta-percha cone the same size as the file used last during preparation *(MAF)* is selected and placed as far as possible into the canal, **but not** beyond the working length. Once satisfactory tug-back and apical positioning appear to be obtained, a radiograph is taken to verify cone positioning. If an accurate determination and careful enlargement have been performed, the x-ray will show that the master cone **reaches the most apical position of the preparation or extends to a point just short of that** *(1 mm)*. When the cone is slightly short, the pressure of condensation **plus** the lubricating action of the sealer will be sufficient to produce complete seating of the cone.

If the cone is **more than 1 mm** from the radiographic apex, discard the cone and fit a smaller one **or** instrument more in the apical third.

Remember: The main reason for **recapitulation** *(using your MAF after each increase in file size)* during instrumentation of the canal is to clean the apical segment of the canal of any dentin filings that **were not removed** by irrigation.

Note: Common solvents used to soften gutta percha are chloroform, methylchloroformate, halothane, rectified white turpentine, and eucalyptol.

All of the following criteria **must be met** before a canal is considered ready to fill with the gutta-percha **except:**

- The canal must be prepared in a manner that ensures optimum debridement and access to the apical area so that the filling material can be condensed to obturate the entire preparation
- The tooth must be **asymptomatic**
- At the time of fill, the **canal must be dry**
- If a bacteriologic culture test is being used, a **negative culture** must be obtained
- The tooth responds to thermal tests

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• **The tooth responds to thermal tests**

***This indicates inadequate debridement, as a pulpless tooth **should not** respond to any stimuli.

The **most important consideration** before filling a root canal is **proper cleaning** (*debridement)* and **shaping** *(instrumenting)* of the canal. Once the canal is obturated, any organisms that have entered the periapical tissues from the canal are eliminated by the natural defenses of the body.

Objectives of root canal obturation:
• To develop a fluid-tight seal at the apical foramen
• Complete filling of the root canal space
• To create a favorable biologic environment for the process of tissue healing

In endodontic treatment the importance of canal obturation *(filling)* is **second only** to canal debridement. Approximately 40% of failures are believed to be caused by incomplete obturation of the root canal. If the canal is not filled, tissue fluid and microorganisms from the periapical tissues are able to enter the voids, with failure as the ultimate result. However, if an accessory canal is **not totally filled during obturation,** the appropriate treatment is to observe the tooth and evaluate every three months.

Note: After endodontic therapy is completed on a tooth with a periapical radiolucency, it usually takes **6-12 months** before marked reduction in the size of the radiolucency is evident on an x-ray. **Desired periapical tissue** changes include regeneration of alveolar bone, deposition of apical cementum, and re-establishment of the PDL.

The most commonly used **irrigant** in endodontics, Sodium Hypochlorite *(NaOCl)* is used in the concentration of:

- 1%
- 2.6%
- 5.25%
- All of the above

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• **All of the above**

***To date there is no agreement on any single concentration-value of **Sodium Hypochlorite** *(NaOCl)* as being the most effective while being the safest. All listed are considered acceptable.

It is the **most widely used irrigant** and has effectively aided canal preparation for many years. A 5.25% solution provides excellent **germicidal solvent action,** but is dilute enough to cause only mild irritation when contacting periapical tissue. NaOCl is a good tissue solvent as well as having some antimicrobial effect. It also acts as a lubricant for root canal instrumentation. **Note: It is toxic to vital tissue; always use rubber dam.**

Hydrogen peroxide *(3% solution)* is also widely used in endodontics with two modes of action. The bubbling of the solution when in contact with tissue and certain chemicals physically foams debris from the canal *(effervescent effect).* In addition, the liberation of oxygen will destroy strictly anaerobic microorganisms. The solvent action of hydrogen peroxide is much less than that of NaOCl. **However,** many clinicians use the solutions alternately during treatment.

Urea peroxide is available in an anhydrous glycerol base, as **Gly-Oxide,** to prevent decomposition and is a useful irrigant. It is better tolerated by periapical tissue than NaOCl, yet has greater solvent action and is more germicidal than hydrogen peroxide. Therefore, it is an excellent irrigant for treating canals with normal periapical tissue and wide apices. **The best use for Gly-Oxide is in narrow and / or curved canals, utilizing the slippery effect of the glycerol.**

Note: Irrigants perform the important biologic function of destroying bacteria during endodontic therapy. **Their action is unquestionably more significant than that supplied by the use of intracanal medicaments.** Irrigants should be **used copiously** throughout the instrumentation phase of root canal procedures.

While regaining canal patency during endodontic retreatment, all of the following statements are true **except:**

- Eucalyptol is the reagent of choice to dissolve gutta-percha
- A "crown down" sequence *(larger to smaller)* of instruments is used from coronal to apical
- Rotary instruments work faster and improve the access early in the treatment as compared to heated instruments
- A very light apical pressure must be applied while using Nickel Titanium rotary files
- Over-extended gutta-percha cones had to be removed by extending file periapically

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• **Eucalyptol is the reagent of choice to dissolve gutta-percha**

***This is **false;** chloroform is the reagent of choice → gutta-percha is slightly soluble in Eucalyptol.

Highly concentrated chloroform is very effective but should be used with caution. Its vapor is potentially hazardous so it is dripped directly in the canal **avoiding excessive flooding.**

Other chemicals which can dissolve gutta-percha to a varying degree include: **xylol,** halothane, benzene, carbon disulfide, essential oils, methyl chloroform and white rectified turpentine.

If a gutta-percha cone has passed beyond the apex then a file must be used beyond the apex in order to avoid breakage of the cone. A broken cone in the periapical area may result in an orthograde re-treatment failure.

Techniques to remove gutta-percha include:
- Rotary removal
- Ultrasonic removal
- Heat removal
- Heat and instrument removal
- File and chemical removal

Notes:

1. Gutta-percha points may be **disinfected** by placing them in a 5.25% NaOCl solution for one minute.
2. A **glass bead sterilizer** can sterilize endodontic files in **15 seconds** at 220° C *(428° F).*

All of the following statements regarding **ethylene diamine tetra-acetic acid** *(EDTA)* are true **except:**

- It is a chelating agent with the capability to remove the mineralized portion of the smear layer
- It can decalcify up to a 50 μm thin layer of the root canal wall
- Normally it is used in a concentration of 17%
- RC-Prep and EDTAC are other preparations of EDTA
- The decalcifying process induced by EDTA is self-limiting
- It is also an excellent irrigation solution

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• **It is also an excellent irrigation solution**

***This is **false;** it has a limited value as irrigation solution. The decalcifying process induced by EDTA is self-limiting and stops as soon as the chelator is used up.

Chelating agents are used to aid and simplify preparation for very sclerotic canals after the apex has already been reached with a fine instrument. These agents act on **calcified tissues only** and have little effect on periapical tissue. Their action is to substitute sodium ions, which combine with the dentin to give soluble salts for the calcium ions that are bound in less soluble combination. The edges of the canal are thus softer, and canal enlargement is facilitated.

EDTA will remain active in the canal for 5 days **if not** inactivated. For this reason, at the completion of the appointment the canal **must be irrigated** with a sodium hypochlorite *(NaOCl)* containing solution. **Note:** Rinsing for 1 minute with EDTA eliminates the smear layer, opens dentinal tubules, and **provides a cleaner surface** for gutta-percha and sealer to adapt.

EDTAC is EDTA with the **addition of Cetavlon,** a quaternary ammonium compound. It has greater antimicrobial action than EDTA. However, it has greater inflammatory potential to tissue as well. **The inactivator for EDTAC is NaOCl.**

RC-PREP combines the functions of **EDTA plus urea peroxide** to provide both chelation and irrigation. The foamy solution has a natural effervescence that is increased by irrigation with NaOCl to aid in the removal of debris.

The most acceptable method to achieve adequate root canal debridement is:

- To obtain clean shavings of the canal
- To attain a clean irrigating solution
- To achieve glassy smooth walls of the canal
- All of the above criteria are reliable
- None of the above criteria is acceptable

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- **To achieve glassy smooth walls of the canal**

***Clean shavings are difficult to see on a file. The attainment of a clean irrigating solution is considered an inaccurate way to determine the end point of debridement.

Debridement is defined as the removal of foreign material and contaminated or devitalized tissue from or adjacent to a traumatic infected lesion until surrounded healthy tissue is exposed. Chemomechanical debridement of the root canal system is the **most crucial** aspect of root canal treatment.

Complete debridement of the canal is the **most effective means** to reduce root canal microorganisms. It can be carried out in various ways as the case demands, and may include instrumentation of the canal, placement of medicaments and irrigants and / or surgery.

Remember:

- The **most common cause** of root canal failure is incompletely and inadequately disinfected root canal systems.
- The **second most common cause** of failures of root canals is leakage from a poorly filled canal.This is common even after apical curettage. **Example:** Root canal treatment performed on a tooth with apical curettage of a lesion that was found to be a cyst. Three years later the lesion is even bigger than it was before. The most likely cause of this failure is **leakage** from a poorly filled canal.

While cleaning and shaping the canal, an instrument separates in the canal. Your first attempt to retrieve it results in a broken instrument passing through the apex. How should you manage this case now?

- Use a smaller H file to bypass it and try retrieving it
- Use Gates Glidden drills to widen the canal and then try retrieving it
- Raise a flap and remove the instrument surgically followed by gutta-percha filling the canal
- Extract the tooth as irreparable damage has occurred to the apex
- Just inform the patient, fill the canal with gutta-percha and monitor

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- **Raise a flap and remove the instrument surgically followed by gutta-percha filling of the canal**

Generally, when a broken instrument protrudes past the apex, surgery should be performed. This constant irritant must be removed.

Note: It is relatively easier to retrieve an instrument if it is wedged coronal to the curvature or at the curvature of the canal but very difficult if it has passed the curvature.

When an instrument **breaks off anywhere** in the canal and a periapical radiolucency is present and minimal canal enlargement has been performed before the accident, surgery is indicated **since the periapical tissues have had little opportunity for healing to be stimulated.** You would prepare and obturate to the point of blockage and then perform an apicoectomy and retrofilling.

However, when an instrument is broken off in the **apical third** and is lodged tightly with no periapical radiolucency evident, the remaining root canal space can be filled. The patient should be **informed** of this and placed on a 3-6 month recall.

Prognosis of a tooth with a broken instrument is **best** if the tooth had a vital pulp and no periapical lesion.

Which of the following are acceptable methods to clean and shape a canal using Nickel titanium instruments?

- Push and pull stroke
- Reaming motion
- Engine-driven rotary motion
- All of the above

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• **All of the above**

***The engine driven instruments, however, use only the reaming motion. **Nickel titanium instruments** can be both hand operated and engine-driven.

Generally, **hand instrumentation** is done by either filing *(push and pull)* or reaming *(repeated rotations)*.

Filing is a push-pull action with emphasis on the withdrawal stroke. Its efficiency is **greatest with files** than with reamers for removing dentin because of the greater number of flutes in contact with the canal walls during the rasping motion of removing the instrument. Filing action produces an irregularly shaped canal and therefore must be filled with gutta-percha in a condensation procedure.

Reaming is defined as the repeated clockwise rotation of the instrument, particularly during insertion. Reaming produces a canal that is round. Reaming is recommended if using a silver cone to fill canals.

Circumferential filing is a **push-pull action** with emphasis on scraping the canal walls to create a **smooth, tapered preparation.** It is a method of filing whereby the instrument is moved first towards the buccal side of the canal, then reinserted, and removed slightly mesially. This is done all the way around the tooth until all the dentin walls have been planed. **This technique enhances preparation when a flaring method is used.**

The major advantage of **zinc oxide-eugenol** based sealer types is:

- Non-staining property
- Fast setting time
- Adhesion
- Insolubility
- Long history of successful usage

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• **Long history of successful usage**

Remember: The primary function of a root canal sealer is to **fill in the discrepancies between the core-filling material and the dentin wall.** In fact it is said that it is more important than the core filling material.

Other **purposes** or **functions** of a root canal sealer include:
• **To act as a lubricant**, facilitating placement of the gutta-percha
• **To form a bond** between the filling material and the dentin walls
• **To exert antibacterial activity** *(some exert more than others).* This activity is the **highest in the period of time immediately after its placement**

Most root canal sealers are some type of **zinc oxide-eugenol cement** and are capable of producing a seal while being well-tolerated by periapical tissues.

All sealers display some degree of **radiopacity** *(caused by metallic salts in the sealer)*; thus are visible on a radiograph. This helps disclose the presence of accessory canals, resorptive areas, root fractures, and the shape of the apical foramen and other structures of interest.

Note: After filling a tooth with **gutta-percha,** if you see a horizontal line of material *(gutta-percha or sealer)* extending **both mesially** and **distally** from the canal to the periodontal ligament space, this is **indicative of a root fracture.**

ZOE disadvantages: staining, slow setting time, non-adhesion, solubility.

During access preparation on **mandibular molars**, two regions tend to be "overcut" which results in the undesirable over preparation of the tooth. Which are those two mostly abused areas?

- The mesial aspect under the marginal ridge
- The distal aspect under the marginal ridge
- The lingual surface under the lingual cusps
- The buccal surface under the buccal cusps

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- **The mesial aspect under the marginal ridge**
- **The lingual surface under the lingual cusps**

***Mandibular molars** tip mesially and lingually. If a bur is directed straight inferior it may cause unnecessary loss of tooth structure from the above mentioned areas.

Major objectives of the access preparation:

1. Straight-line access
2. Conservation of tooth structure
3. Unroofing of the chamber and to remove pulp horns

Access to the root canal is the **initial step** in canal preparation. It is necessary to establish **straight-line access** to the apical foramen to ensure free movement of the instrument during debridement and preparation of the canal. All the treatment that follows **hinges on the correctness** of the access preparation. All access cavities are made through the **lingual on anterior teeth** and **through the occlusal on posterior teeth.**

Note: A **facial** approach is recommended for an access opening on **maxillary primary incisors.**

Remember: Mandibular incisors and maxillary first premolars require the most care to avoid perforation during preparation of the access opening. This is due to the narrow mesio-distal dimension of the mandibular incisors and the mesial concavity of the maxillary first pre-molars.

A **reaming action** produces a canal that is relatively:

- Square in shape
- Irregular in shape
- Round in shape
- Triangular in shape

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• **Round in shape**

Studies have shown that the **action** of using the instrument, rather than the instrument used, determines the general shape of the canal preparation. Therefore, **a reaming action** produces a canal that is relatively **round in shape** while a **filing action** produces a canal that is **irregular in shape.**

Important: A canal should be instrumented and shaped so that it has a continuously tapering funnel shape. The **widest diameter** would be at the canal opening and the **narrowest** at the **dentinocemental junction** *(.5 to 1.0 mm from the radiographic apex)*. This is where all teeth should be filed to and filled to *(ideally)*.

Which of the following intracanal instruments is **designed for** the removal of pulp tissue, cotton pellet absorbent points, and other soft materials, **but not** for canal enlargement?

- Files
- Reamers
- Broaches
- None of the above

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- **Broaches**

The barbs are notched out of the instrument shaft and represent a **weakened point.** If the broach is not used with the **utmost of care** or if it is **forced apically,** the barbs will be bent and will engage the walls, making removal difficult. It is not used for canal enlargement.

K-type instruments:

- **Files** are the most useful instruments in endodontics for the removal of hard tissue in canal enlargements. They are manufactured by twisting a blank, which is a square rod, producing a series of cutting flutes. The action used for placing this type of file into a canal should resemble a clockwise-counterclockwise motion with pressure directed apically *(can be a filing or reaming action).* **Note:** These files are the **strongest** of all files and cut the **least aggressively.** A modification to this type of file is the **K-flex file.**
- **Reamers** are manufactured in a manner similar to files, only they have fewer flutes. They are used in canal preparation to shave dentin and enlarge canals with a **reaming action only.** They remove intracanal debris with clockwise reaming action. They are also used to place materials into the apical portion of the canal by using a counterclockwise rotation.

H-type instruments:

- **Hedstrom files** are manufactured by using a sharp, rotating cutter to gauge triangular segments out of a round blank shaft. This produces a very sharp edge and therefore an effective cutting instrument. If used carefully, **with filing action only,** it will successfully plane the dentin walls much faster than K-type files or reamers. A modification of this file is the **S-file.**

Note: All of the above are made of **stainless steel.**

Which cells **do not** characterize the cellular response at the onset of pulpal inflammation?

- Plasma cells
- Macrophages
- Lymphocytes
- Polymorphonuclear *(PMN)* Leukocytes

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• **Polymorphonuclear *(PMN)* Leukocytes**

The **onset** of pulpal inflammation is an insidious process and is characterized by a **chronic cellular response** *(plasma cells, macrophages and lymphocytes).* There is no direct exposure of the pulp to dental caries and the response, therefore, is not acute. After **pulp exposure,** the acute inflammatory cells *(mainly PMN cells)* are chemotactically attracted to the area. **Histologically,** the tissue is likely to show signs of acute inflammation near the site of the exposure and a band of chronic inflammatory cells between the acute inflammation and the underlying normal pulp.

The response of **vital pulp to microbial invasion** is very resistant. Based on the observation that even after two weeks of traumatic pulp exposure, only 2 mm of coronal pulp may "give in" to microorganisms. **Non-vital pulp,** in contrast, is a "fertile ground" for the growth of microorganisms.

Remember: Carious exposures in permanent teeth generally require root canal treatment. **Immature** *(open apex)* permanent teeth with carious exposures can be treated by pulp capping or pulpotomy procedures.

Pulp capping is **not recommended** in primary teeth with carious exposures due to its high failure rate and because pulpotomy, having similar time requirements, has shown to be very successful. Pulp capping can be done, however, in mechanical exposures.

When symptoms and clinical tests show the presence of pulpal pathosis in a **posterior tooth** and the radiograph shows no decay or restoration in any proximity to the pulp, this is virtually **pathognomonic** of:

- Condensing osteitis
- A vertical fracture of the tooth
- Periodontal abscess
- Secondary occlusal trauma

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• **A vertical fracture of the tooth**

Radiographic examination **seldom reveals the fracture** because the crack is usually parallel to the x-ray film. One of the most puzzling and frustrating dental conditions involving the possible need for endodontic treatment is the **cracked tooth syndrome.** Symptoms from this condition usually are characterized by a **sharp but brief pain** occurring unexpectedly only when the patient is chewing. Having a patient bite forcefully on a bite stick and noticing the cusps that occlude when the pain occurs will aid in the location of the offending tooth.

Vertical fractures through root structure, however, have an **almost hopeless** prognosis. If the fractured segment can be removed and gingivoplasty and alveoloplasty performed, treatment can be successful. However, unrealistic or overambitious case selection leads to a high degree of failure.

When an anterior tooth fractures, it generally occurs in a more horizontal plane and may show up on the x-ray. The cause is usually accidental trauma such as a blow to the jaw or teeth. If the fracture line is not too far down the root of the tooth, it may be able to be saved with a root canal and a crown.

Inlays have been shown to be a **cause** of fractures. If a patient complains of pain on mastication since the placement of an inlay, suspect a fractured cusp *(using a bite stick will help determine which cusp may be fractured)*.

Which of the following is the **most commonly** used bleaching agent for endodontically treated teeth?

- Ether
- Superoxol
- Chloroform
- Sodium hypochlorite

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• **Superoxol**

Superoxol is a **30% aqueous solution** by weight of hydrogen peroxide in distilled water. It is potent oxidizing agent whose bleaching effect results from direct oxidation of stain-producing substances.

Chairside technique: Application of heat to Superoxol-saturated cotton pellets in the tooth chamber. Repeat until tooth is lighter. **Note:** The heat liberates the **oxygen** in the bleaching agent.

Important:

- Cervical root resorption relating to bleaching is a potential side effect; usually it does not manifest for at least 6 months. This is a reason why recall appointments are important.
- The most probable **postoperative complication** of bleaching a tooth that has not been adequately obturated is an **acute apical periodontitis.**
- Tooth bleaching **causes** a color change in both **enamel** and **dentin.**

Walking bleach technique: Place a thick paste consisting of **sodium perborate** and **2-3 drops of Superoxol** in the tooth chamber with a temporary restoration for four to seven days. Several repetitions of this procedure can work quite well.

Hydrogen peroxide is the most effective bleaching agent; used in concentrations of 30-50%. It is best delivered in an alkaline medium.

Tooth # 9 requires root-end surgery. Which flap design is generally **not** indicated?

- A submarginal curved flap
- A submarginal triangular and rectangular flap
- A full mucoperiosteal flap
- None of the above

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• **A submarginal curved flap** → also called **semilunar** flap

This half-moon shaped flap is raised with a curved horizontal incision in the mucosa or attached gingival with the concavity towards the apex. Although it's simple and does not impinge on the surrounding tissue, the **disadvantages outweigh** its advantages. These include: **1)** Limited access and visibility, **2)** Tearing of corners of the incisions when an attempt is made to improve accessibility by stretching the flap, **3)** If somehow a lesion is found to be bigger than anticipated, the incisions come to lie over the bony defect. Healing occurs by scarring, **4)** Its extent is also limited by attachments *(e.g., frenum, muscles etc.)*. Therefore, this technique is not used for anterior root end surgery.

Submarginal triangular and **rectangular flap** *(Ochsenbein-Leubke)* requires at least 4 mm of attached gingiva and a healthy periodontium. It is raised by a scalloped incision in the attached gingiva with one or two vertical incisions. Less risk of incising over bony defects and no post-surgical recession of gingiva. Its disadvantages include hemorrhage from the cut margins and scarring. Access and visibility is better *(and acceptable)* than semilunar flap but not as good as full mucoperiosteal flap.

Full mucoperiosteal flap allows maximal access and visibility. It is raised from the gingival sulcus *(elevating gingival crest and interdental gingiva)*. This wide outline of the flap precludes any incisions over bony defects and allows various periodontal procedures including curettage, root planing and bone re-shaping. A large flap may be difficult to reposition, suture and make alterations. Post surgical gingival recession is also a possibility.

Which of the following are **contraindications** to endodontic therapy?

- A **non-restorable** tooth
- A tooth with **insufficient periodontal support**
- A tooth with a **vertical root fracture**
- All of the above

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- **All of the above**

Other **contraindications** include:

- A **non-strategic tooth** → a tooth not in occlusion
- A tooth with massive **internal** or **external resorption**
- A tooth that has a canal **unsuitable** for instrumentation or for surgery *(i.e., broken instruments, dentinal sclerosis, sharp dilacerations, etc.)*

A medical condition such as hemophilia is **not** a contraindication to conventional endodontic therapy. However, it is strongly recommended that a dentist obtain clearance from the patient's physician prior to treatment.

Note: Example of a **special case:** A previously traumatized tooth may show complete obliteration of the pulp chamber and canal. The periodontal ligament may appear normal. The patient will be asymptomatic and the tooth will **not respond** to pulp vitality testing. The treatment of choice is to observe as long as the tooth **remains asymptomatic** and no periapical changes are evident.

A periodontal probing defect which may **not** be managed by endodontic treatment alone is:

- A conical shaped probing
- A narrow sinus tract type probing
- A blow-out type probing
- None of the above

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• **A conical shaped probing** → this is typical of primarily, a periodontal problem.

***In "blow-out type" and "sinus tract type" probings, another clue for diagnosis is a non-vital *(necrosed)* pulp → these two lesions can completely heal after root canal treatment.

Acute or blow-out lesions → a tooth with this type of lesion will show normal sulcus depth all the way around the tooth until the area of the swelling is probed. At this point, the probe drops suddenly, to a level near the apex. The probing depths in all other areas are within normal limits.

Periodontal lesions characteristically show bone loss which begins at the crestal bone level and progresses apically. Hence probing defect would be **conical** in shape. This type of lesion may not be amenable to root canal treatment alone even if it is associated with a pulpless tooth. However, endodontic treatment must be completed prior to tackling the periodontal problem.

A narrow sinus tract type lesion → the probing reveals normal depths all around the tooth except at one very narrow area. Here, the probe can pass down the root surface to some distance and sometimes even to the apex. The tooth is pulpless *(non-vital)*. Once the root canal treatment is completed, the lesion heals within one week.

Remember: A perio-endo abscess is a combined lesion. The lesion usually demonstrates radiographic involvement of the periodontium and the apex of the involved tooth.

Note: A common clinical finding of a periodontal problem is **pain to lateral percussion** on a tooth with a wide sulcular pocket.

Regarding the restoration of endodontically treated teeth, all of the following are generally believed to be true **except:**

- A major disadvantage of posts / dowels is that it does not reinforce the tooth structure, in fact, it weakens it
- All post designs are predisposed to leakage
- At least 4 mm of gutta-percha must remain to preserve the apical seal
- Threaded screw posts are preferred over parallel sided and tapered posts
- Pins add to stresses and microfractures in dentin and should not be used
- Cusps adjacent to lost marginal ridges should be restored with an onlay

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• **Threaded screw posts are preferred over parallel sided and tapered posts**

***These may actually increase the chance of fracture. The **parallel-sided** posts are preferred.

Options available when **restoring** endodontically treated posterior teeth:
Restoration of occlusal opening only → in **rare instances** the access opening and caries destruction **do not** encroach on the cusps and marginal ridges. These teeth may be restored with an occlusal amalgam; however, **a cuspal coverage restoration would provide protection from fracture.**

Onlay restoration → in most cases it is imperative that root canal treated teeth be protected from fracture by a cusp-coverage type of restoration. The minimum (*most conservative)* preparation should be for an **onlay** covering the cusps and marginal ridges.

Crown → a full-coverage crown is preferred when the remaining coronal tooth structure does not afford sufficient tooth structure for an onlay.

Crown with post and core → to reinforce the treated tooth and provide suitable coronal tooth structure for an optimum crown preparation, the use of a post and core is often indicated. Be very careful when placing posts. **Perforations and vertical root fractures can occur.**

Notes:

1. If you are performing a pulp chamber-retained amalgam, **you need to place amalgam 3 mm into each canal for retention.**
2. **Endodontically** treated posterior teeth are more prone to fracture than untreated posterior teeth **due mainly** to the destruction of the coronal tooth structure → they have reduced structural integrity.

An **apicoectomy** is a resection:

- Of the most coronal portion of the root
- Of the coronal portion of the pulp horn
- Of the most apical portion of the root
- Of the entire pulp horn

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• **Of the most apical portion of the root**

***An **apicoectomy** is best accomplished by **obliquely resecting the most apical portion** of the involved root.

If a tooth has had previous endodontic therapy and becomes **reinfected,** it is usually best to try and retreat it conventionally → remove filling material, debride the canals, and refill. **However,** if the tooth has been restored with a post, core, and crown then apical curettage, apicoectomy, and a retrofill should be performed. Retreating a tooth with a post is the **most common reason** for an apicoectomy and retrograde filling.

Indications for apicoectomy:
- A reverse filling needs to be placed.
- It is necessary to gain access to an area of pathosis.
- The poorly filled apical portion of the root is to be removed to the level of canal obliteration.

Indications of periradicular surgery:
- Non-negotiable canal, blockage or severe root curvature in which non-surgical treatment is impossible.
- Complications arising from procedural accidents *(e.g., separation of instruments, ledging and / or perforations)* which cannot be handled without surgical exposure of the site.
- Failed treatment due to irretrievable posts or root fillings.
- Horizontal apical fractures in which apical end of the pulp becomes necrotic.
- Biopsy → to diagnose non-odontogenic causes of symptoms *(e.g., patient with a history of previous malignancy, lip paresthesia or anesthesia).*

Which **two** of the following **decrease with age** in the dental pulp?

- Number of collagen fibers
- Number of reticulin fibers
- The size of the pulp
- Calcifications within the pulp

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- **The number of reticulin fibers**
- **The size of the pulp**

As the pulp ages there is a **decrease in reticulin fibers** *(the pulp becomes less cellular and more fibrous)*. The size of the pulp **also decreases** because of the continued deposition of dentin.

As the pulp ages there is an **increase in the number of collagen fibers** and **calcifications** within the pulp *(called denticles or pulp stones)*. **Note: Pulp stones** are associated with chronic pulpal disease → from advanced carious lesions or large restorations.

The pulp contains both **myelinated** and **unmyelinated** nerve fibers. They are afferent and sympathetic. The myelinated fibers are **sensory** and the **unmyelinated** fibers are **motor** → they play a role in the regulation of the lumen size of the blood vessels.
Note: Proprioceptors *(which respond to stimuli regarding movement)* are **not found** in the pulp.

The **only type** of nerve ending found in the pulp is the **free nerve ending,** which is a specific receptor for pain. Regardless of the source of stimulation *(heat, cold, pressure)*, the **only response** will be pain.

The **absence** of which layer of dentin predisposes it to internal resorption by cells present in the pulp?

- Mantle dentin
- Circumpulpal dentin
- Predentin
- Secondary dentin
- Tertiary dentin

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• **Predentin**

Immediately adjacent to the odontoblast layer in the pulp, 10-47 μm of the dentin matrix remain unmineralized. If this unmineralized layer of dentin is lost *(e.g., due to trauma or infectious process)* it predisposes the dentin to internal resorption by odontoclasts.

Mantle dentin → is first-formed dentin which is laid before odontoblast layer gets organized. Hence the pattern of deposition and size of collagen fibers is different from circumpulpal dentin.

Circumpulpal dentin → represents most of the dentin which is formed.

Secondary dentin → forms after eruption of a tooth and throughout life resulting in a gradual but asymmetric reduction in pulp size.

Tertiary dentin or reparative dentin → is an irregular and disorganized layer of dentin laid down in response to any injurious / irritant stimuli.

Dentin formation is the primary function of pulp.

Other functions include:

- **Induction** → forms dentin which in turn induces enamel formation
- **Nutrition** → dentinal tubules are linked to the pulp which maintains its hydration and formation of peritubular dentin.

Normally, which portion of the pulp contains **more** collagen?

- Coronal portion
- Apical portion

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• **Apical portion**

***This facilitates a pulpectomy with the help of barbed broaches or endodontic files.

Mainly, **Type I** and **Type III** collagen is present in the pulp in a ratio of 55%:45%. **Type V** is found in small amounts. **In dentin,** Type I collagen predominates. Odontoblasts synthesize **Type I** while fibroblasts in the pulp synthesize both Type I and II.

The **central zone** or **pulp proper** contains large nerves and blood vessels. This area is lined peripherally by a specialized odontogenic area which has three layers *(from innermost to outermost):*

1. **Cell rich zone; innermost** pulp layer which contain fibroblasts.
2. **Cell-free** zone or **zone of Weil** which is rich in both capillaries and nerve networks. The **nerve plexus of Rashkow** is located here.
3. **Odontoblastic layer; outermost** pulp layer which contains odontoblasts and lies next to the predentin and mature dentin.

Cells found in the dental pulp include fibroblasts *(the principal cell),* odontoblasts, histiocytes *(macrophages),* and lymphocytes.

Note: In a **diseased pulp,** the following cells are present: **PMN's,** plasma cells, basophils, eosinophils, lymphocytes, and mast cells *(contain histamine and heparin).*

Which of the following statements regarding the management of an avulsed tooth are **true?**

- After 60 minutes of dry storage of an avulsed tooth, few periodontal ligament cells survive
- The storage of an avulsed tooth in tap water is as bad as dry storage
- Saliva is hypotonic and can allow storage of the tooth up to 2 hours
- Milk has a maximum storage time of up to 6 hours
- Teeth with complete root development should be treated endodontically as soon as possible even if replanted within 30 minutes
- Teeth with incomplete root development and replanted within 30 minutes may not require endodontic treatment
- All of the statements are true

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• **All of the statements are true**

Five factors that are critical to the management of **traumatic avulsion injuries** to teeth:

1. **Time** → the time interval from injury to replacement of the tooth is a **major factor** in the maintenance of ligament viability and subsequent root resorption. Teeth replanted within 30 minutes have been reported to exhibit very little resorption, whereas most of the teeth replanted after 2 hours show a lot of external root resorption *(which is the main cause of failure of replanted teeth).*
2. **Storage media** → if the tooth cannot be immediately replanted, the proper storage of the tooth can **favorably influence** the viability of PDL cells. Milk is considered best for this purpose because of its near neutral pH *(6.5-6.8)* and osmolality, conducive for the survival of cells. Other storage media are physiologic saline and saliva.
3. **Tooth socket** → should not be damaged by curettage or forceful replantation.
4. **Splint stabilization** → a splint that allows the physiologic movement is placed for a maximum of 2 weeks. This time period allows for the initial reattachment of the periodontal ligament fibers.
5. **Root surface** → should **not be** scraped, dried, or manipulated with caustic chemicals.

Note: The above information changes when a tooth has been out of the mouth **for more than 2 hours** → mainly the treatment of the tooth socket and root surfaces as well as the time for splint stabilization.

Which procedure is probably **not considered** essential for the success of intentional replantation?

- A skillful extraction
- Minimum out-of-socket time
- Minimum damage to the root while tooth is held during apical root end preparation
- Curettage of the socket to remove periapical pathosis
- Repair of any perforation or resorptive defect before replantation

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- **Curettage of the socket to remove periapical pathosis**

***This is **probably unnecessary.** In fact, socket wall should be minimally manipulated.

Intentional replantation implies that a tooth requiring endodontic therapy is purposely removed from its socket, some type of canal or apical preparation and / or filling is performed, and the tooth is **returned** to its original socket.

Indications for intentional replantation *(also called replant surgery):*

- When routine endodontic therapy of a tooth is **impractical** or **impossible**
- When an **obstruction of a canal** is present, such as a broken instrument or a calcification, and periapical surgery is impractical *(a lower molar with the mandibular canal in close proximity).*
- When perforating **internal** or **external resorption** is present, yet surgery is impractical
- When a previous treatment **has failed** but nonsurgical treatment or surgery is impractical.

Note: Intentional replantation should be considered only when there's **no other alternative treatment** to maintain a "strategic" tooth. Long term follow up is required to monitor for complications including periodontal defects and ankylosis with replacement resorption.

The **main benefit** of primary incisor replantation is:

- Maintenance of a normal anterior dentition
- To relieve parental guilt
- To maintain child's self-esteem
- To maintain child's social acceptance

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• **Maintenance of a normal anterior dentition**

***The question of whether to replant primary teeth has been a focus of debate and controversy in the dental literature. **However,** most dental textbooks uniformly recommend that **primary teeth** not be replanted. Replantation of a primary tooth is **not** recommended because of the potential danger to the permanent successor from sequels of trauma *(e.g., infection, ankylosis, or damage due to manipulation during procedure itself)*.

Proper management of an **avulsed permanent tooth** that has been **replanted within two hours** of the accident:

- Ten days to two weeks after replantation, the root canal is prepared *(cleaned and shaped)* and a **calcium hydroxide paste** is placed into the canals.
- This paste is **replaced every three months** for one year.
- **If after one year,** it appears that resorption has reversed or stopped, a permanent gutta-percha filling can be placed.

Important: If a tooth is out of the mouth for **more than two hours:**

- **Ankylosis and external root resorption** will probably result within two years. **Ankylosis** resulting from replacement would give a better prognosis than **external resorption,** which lead to failure.
- Root canal therapy is performed in its entirety **prior to replantation.**
- **The tooth is soaked in a 2.4% fluoride solution** acidulated at pH 5.5 for 20 minutes or more. The fluoride will slow the resorptive process.
- **Gently curette blood clot** out of the alveolar socket and **irrigate with saline.**
- **Rinse tooth with saline,** replant into socket, and splint for 4-6 weeks.

Note: Resorption is the most frequent sequela to replantation. Three different types of resorption have been identified: surface, inflammatory and **replacement** *(ankylotic resorption)*. **Replacement resorption** refers to resorption of the root surface and its substitution by bone, **resulting in ankylosis.**

Which of the following is generally believed to be the **cause of** internal resorption of a tooth?

- Active orthodontic treatment
- Periodontitis with deep infra-bony pockets
- Inflammation due to an infected coronal pulp
- Dento-alveolar abscess

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• **Inflammation due to an infected coronal pulp**

***This condition is frequently precipitated by traumatic injury to the tooth. Undifferentiated reserve connective tissue cells of the pulp are activated to form **dentinoclasts,** which resorb the tooth structure in contact with the pulp.

Internal *(inflammatory)* **resorption** is usually asymptomatic and is discovered on routine radiographic evaluation. The anatomic configuration of the root canal is altered and increases in size with internal resorption. It will appear as an irregular radiolucency anywhere along the canal space. The tooth involved **may respond** to pulp vitality tests. When internal resorption is detected, a **pulpectomy** should be performed. Once the pulp tissue responsible is removed, all resorption ceases. To "wait and see" may result in sufficient destruction of the tooth to create a perforation of the root.

Typical depiction of radiographic appearance for internal resorption

Note: Although, internal resorption can occur only when some of the pulp tissue is still vital, a negative sensitivity test does not rule out this etiology. Also remember that sometimes on a radiograph, an external resorptive lesion can superimpose the canal space to mimic internal resorption. In such cases, another radiograph should be exposed at an angle to the tooth. The radiolucent lesion inside the canal space will not shift.

The **external resorption** in which an infected pulp may further complicate the resorptive process, is termed as:

- Surface resorption
- Inflammatory resorption
- Replacement resorption

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• **Inflammatory resorption**

Bowl-shaped areas of resorption involving cementum and dentin characterize external inflammatory root resorption. This type of resorption is rapidly progressive and will continue if treatment is not instituted. Since both a necrotic pulp and the presence of bacteria are necessary components of inflammatory resorption, the process **can be arrested** by immediate root canal treatment. The tooth is opened and the canal is cleaned and shaped. A **calcium hydroxide paste** is placed in the canal. This is replaced every three months for one year. If after one year, it appears that the resorption has stopped, a permanent root canal filling *(gutta-percha)* can be placed. A calcium hydroxide-based **root canal sealer** is strongly recommended.

Surface resorption is caused by acute injury to the periodontal ligament and root surface. If injury is not repeated, healing takes place with new cementum and PDL. Root surface resorption is limited to cementum, may heal itself, and is not radiographically visible.

Replacement resorption refers to resorption of the root surface and its substitution by bone, resulting in ankylosis. Replacement absorption accompanies dentoavleolar ankylosis due to extensive trauma to the tooths attachment apparatus. **Remember:** This is often seen in **unsuccessful** replant cases.

Remember the etiology of external and internal resorption:
- **External resorption** → periradicular inflammation, dental trauma *(resulting in damage to attachment apparatus)*, excessive orthodontic forces, impacted teeth, bleaching of non-vital teeth.
- **Internal resorption** → dental trauma *(resulting in loss of vitality and subsequent infection)*, dental caries, pulp capping with calcium hydroxide, cracked tooth.

Endodontic procedures involve taking multiple radiographs. How should you protect yourself or your staff while taking radiographs **if there is no barrier available** to stand behind?

- Stand at least 4 feet away anywhere around the patient
- Stand at least 5 feet away exactly opposite the x-ray beam source
- Stand at least 6 feet away and in the area that lies between 90 to 135 degrees to x-ray beam
- Stand at least 7 feet away and in the area that lies between 60 to 90 degrees to x-ray beam
- Never take an x-ray without a barrier

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- **Stand at least 6 feet away and in the area that lies between 90 to 135 degrees to x-ray beam**

Notes related to radiation safety:

1. A fast *(sensitive)* film, for example E-speed film should be preferred over slower films as faster films require less radiation exposure while providing quality image.
2. Dental units should operate at 70kVp or higher. The higher the kVp, the lower the patient's skin doses.
3. Collimation *(i.e., restriction of the x-ray beam size so that it does not exceed 2.5 inches at the patient's skin, reduces exposure).*
4. Patient should be protected with a lead apron and a thyroid collar for **each** exposure.
5. If there is no barrier for the clinician to stand behind while exposing films, he / she should stand in an **area of minimal scatter radiation** *(i.e., 6 feet away and in the area that lies between 90° to 135° to x-ray beam).*
6. Dental personnel who may get exposed to occupational x-radiation must wear film badges to record exposure and must never exceed the maximum permissible dose *(MPD)* of 50 mSv per year / whole body.
7. An operator should never remain in the room holding an X-ray packet in place for a patient. If film must be held in place by someone else *(i.e. for a child)*, drape the patient and have him / her hold the film.

All of the following statements regarding adjuncts to endodontic treatment are true, ***except***:

- Transplanted teeth with partial root development have a better prognosis than those with developed roots
- Orthodontic extrusion is a common indication prior to implant placement
- Intentional replantation is a viable alternative to endodontic surgery
- To stabilize an intentionally replanted tooth, a very effective method is to ask the patient to close in centric occlusion for the remainder of the day
- A major disadvantage of endodontic implants is the lack of an apical seal

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• **Intentional replantation is a viable alternative to endodontic surgery**

***Intentional replantation** is not a substitute for endodontic surgery if it can be undertaken.

Transplantation is the transfer of a tooth from one alveolar socket to another either in the same person or in another person.

Orthodontic extrusion is defined as force-controlled vertical tooth movement occlusally in the socket. Indications include untreatable subgingival pathoses e.g., cervical caries, cervical fracture, periodontal defects, **resorptive lesions** and **perforations** in the cervical area.

Crown lengthening is a procedure used to apically position the gingival margin and / or to reduce the cervical bone. It is employed during the treatment of subgingival caries perforations and resorption.

Root submersion involves resection of tooth roots 3 mm below the alveolar crest and then cover with a mucoperiosteal flap. Indications include rampant caries, adverse periodontal conditions and in cases that have had repeated prosthetic failures. The submerged roots will prevent alveolar resorption and maintain better proprioception. This is especially useful in medically compromised or handicapped patients requiring better denture control. Sometimes, this is also done to avoid formation of an esthetic defect that may result after extraction.

Several of the significant bacteroides species involved in pulpal-periradicular infection have undergone taxonomic revision. They are now reclassified as:

- Porphyromonas and Prevotella
- Eubacterium and Fusobacterium
- Actinomycetes and Spirochetes
- Wolinella and Veillonella species

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• **Porphyromonas and Prevotella**

***These species, which were previously classified under bacteroid species merited a separate genus due to their distinct characteristics.

Predominant bacterial species isolated from **infected root canals** include:
• Eubacterium species
• Peptostreptococcus species
• Fusobacterium species
• Porphyromonas species
• Prevotella species

Virulence factors which play a role in **periradicular pathosis** include:
• **Lipopolysaccharide** *(LPS)* → found on the surface of gram negative bacteria
• **Enzymes** → neutralize antibodies and complement components
• **Extracellular vesicles** → involved in bacterial adhesion, proteolytic activities, hemagglutination and hemolysis
• **Fatty acids** → affect chemotaxis and phagocytosis

A **vital pulp** resists bacterial invasion. Even if the pulp is exposed to microorganisms for 2 weeks, the penetration of bacteria may extend no more than 2 mm into the pulp. In contrast, **non-vital pulp** is a fertile ground for the growth of microorganisms and leads to necrosis.

Remember: Streptococcus spp. may be more important in the initiation of rather than the progress of a carious lesion leading to a pulp exposure. Strict anaerobes are found to play a significant role in periapical pathoses.

All of the following statements regarding inflammatory resorption of teeth are true **except:**

- External resorption is always accompanied by resorption of the bone
- The teeth with internal resorption have a history of trauma, crown preparation or pulpotomy
- Cervical root resorption does not occur exclusively at the cervical area of the root
- The usual radiographic presentation of internal resorption is a fairly uniform enlargement of the root canal space
- "Pink" tooth is considered to be pathognomonic of replacement resorption
- The pulp plays no role in cervical root resorption

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• **"Pink" tooth is considered to be pathognomonic of replacement resorption**

Traditionally pink tooth has been considered pathognomonic of **internal resorption** and is sometimes a feature of cervical root resorption. It is characterized by a pinkish appearance of the tooth due to growth of granulation undermining the coronal dentin.

Replacement resorption, which accompanies dento-alveolar ankylosis resulting from extensive trauma to the attachment apparatus of the tooth is characterized by progressive replacement of the root by the bone. Histologically, it shows direct contact between dentin and bone with no intervening PDL or cemental layer.

Remember: This condition's **pathognomonic** signs are:
1. Lack of mobility
2. Metallic sound to percussion
3. Infra-occlusion of the involved tooth in the developing dentition

During a routine radiographic evaluation, you notice bone loss extending from the cemento-enamel junction to the apex of tooth #21. Further evaluation reveals that probing depths are above normal limits all around the tooth, however, at one point the probe drops precipitously to an even greater depth. Vitality test is negative. This patient may require:

- Extensive periodontal treatment followed by vitality re-assessment
- Endodontic treatment only
- Endodontic treatment followed by periodontic treatment
- Root end surgery
- Periodontic treatment followed by endodontic treatment

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• **Endodontic treatment followed by periodontic treatment**

***In a combined **perio-endo lesion,** endodontic treatment generally takes precedence over periodontal management.

Combined endodontic-periodontal therapy is widely used because the anatomic and clinical connections between the pulp and periodontal structures are close and numerous. In most cases of this nature, endodontic procedures are preformed first and, when necessary, are followed by periodontal measures.

In these cases, the value of precise pocket probing and correct appraisal of the vitality of the pulp is crucial. In some doubtful cases, the **better part of wisdom** is to wait until after the completion of the root canal therapy to see whether spontaneous resolution *(pocket closure and osseous fill-in)* will occur before surgical periodontal procedures are begun.

Periodontal therapy should be **initiated first only** in the case of a primary periodontal lesion with subsequent secondary endodontic involvement.

Note: A common clinical finding of a periodontal problem is pain to lateral percussion on a tooth with a wide sulcular pocket.

ORTHODONTICS

Legend

Major Topic	Abbreviation
Appliances	Appl
Cephalometrics	Ceph
Crossbites & Openbites	Cb & Ob
General Information	Gen Info
Malocclusion	Mal Occl
Miscellaneous	Misc.
Space Maintainers	Space Mnt

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"Primate spaces" in the primary dentition are found in which two locations?

- In the maxillary arch, the primate space is located between the central incisors and lateral incisors
- In the maxillary arch, the primate space is located between the lateral incisors and canines
- In the mandibular arch, the primate space is located between the canines and first molars
- In the mandibular arch, the primate space is located between the lateral incisors and canines
- There are no primate spaces in the primary dentition

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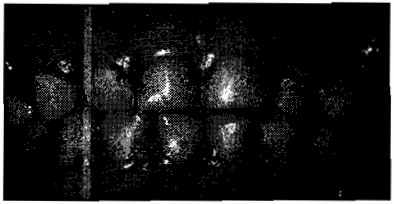
- **In the maxillary arch, the primate space is located between the lateral incisors and canines**
- **In the mandibular arch, the primate space is located between the canines and first molars**

***Spacing is normal throughout the anterior part of the primary dentition, **but is most noticeable** in these two locations.

These primate spaces are normally present from the time the teeth erupt. Developmental spaces between the incisors are often present from the beginning, but become somewhat larger as the child grows and the alveolar processes expand. Generalized spacing of the primary teeth is a requirement for proper alignment of the permanent incisors. This spacing is **most frequently caused** by the growth of the dental arches.



A preschool child is shown with normal dentition. Note the spaces between the maxillary lateral incisor and cuspid and the mandibular cuspid and first primary molar. These spaces are termed primate spaces and their presence allows for the space to be filled by permanent teeth as they erupt.

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If spacing is present, there is a possibility that **drifting** of the adjacent teeth will occur if there is a loss of a primary incisor. However, if there is **no spacing present** and the primary anterior teeth were in contact before the loss, a collapse in the arch after the loss of one of the primary incisors is almost certain.

This is not true in the case of a lost permanent incisor. Space closure occurs rapidly whether spacing is present or not prior to the loss. Space maintenance would be indicated.

Remember: One of the **most common causes of malocclusion** is inadequate space management following early loss of primary teeth.

Which of the following is the **normal relationship** of the primary molars in the deciduous dentition?

- Distal step
- Flush-terminal-plane
- Mesial step
- None of the above

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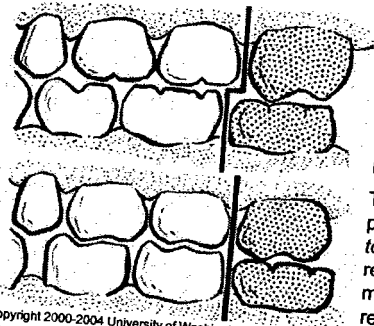
• **Flush-terminal-plane**



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Primary molar relationships are known as **step** relationships. The primary molar relationship shown in this figure is a **mesial-step** relationship, as the distal surface of the lower second primary molar is mesial to the distal surface of the upper second primary molar. The mesial-step molar relationship allows for the first permanent molars to erupt into a normal occlusion immediately on eruption. Note that the permanent molars are in a normal Class I occlusion.

This figure demonstrates the **flush-terminal-plane** relationship for primary molars *(also known as the flat-plane relationship and the end-to-end relationship)*. As shown in the figure, the flush-terminal-plane relationship describes a situation where the distal surfaces of the maxillary and mandibular second primary molars are in an end-to-end relationship.

In these cases *(the flush-terminal-plane)*, the first permanent molars do not erupt immediately into a normal relationship. As you can see, the first permanent molars are in a Class II relationship. The Class II relationship usually is temporary until the second primary molars are lost and the permanent molars move into a Class I relationship. This occurs at approximately age ten or eleven and is called the late mesial shift. Both the mesial-step and flush-terminal-plane relationships usually result in the development of a Class I permanent molar occlusion, although the flush-terminal-plane relationship can result in a Class II relationship if the late mesial shift does not occur. Another step relationship involves a situation where the distal surface of the mandibular primary second molar is located to the distal of the distal surface of the maxillary primary second molar. This is termed a **distal-step** relationship. In these cases, the permanent molars erupt into a Class II relationship. **Important:** The **terminal plane** relationship of **primary second molars** determines the future anteroposterior positions of permanent first molars.

The **permanent** mandibular canines erupt in what direction relative to the primary mandibular canines?

- Lingually
- Facially
- Distally
- Mesially

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• **Facially** *(labially)*

However, often they are **right in line** with the primary canines. If there are problems in eruption, these teeth can be displaced either lingually or labially, but usually they are displaced labially if there is not enough room to accommodate them within the arch.

Note: The mesial inclined plane of the primary maxillary canine articulates with the distal inclined plane of the primary mandibular canine. **This is the normal relationship.**

In both the **maxillary** and **mandibular** arches, the permanent incisor tooth buds lie lingual as well as apical *(inferior)* to the primary incisors. The result is a tendency for the mandibular permanent incisors to erupt somewhat lingually and in a slightly irregular position. This occurs even in children who have normal dental arches and normal spacing within the arches.

***Permanent teeth normally move occlusally and buccally while erupting.

Remember: The maxillary arch is slightly longer *(approximately 128 mm)* than the mandibular arch *(approximately 126 mm)*.

Which of the following is defined by the **difference** in the sum of the **mesiodistal widths** of the **primary** canine, first molar and second molar, and the **permanent canine,** first premolar and second premolar?

- Primate space
- Leeway space
- Moyer's space
- Anatomic space

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• **Leeway space**

Note: The **permanent** successors are **most often smaller** than their **primary** predecessors.

The **mandibular** leeway space averages **3-4 mm** while the **maxillary** leeway space averages **2-2.5 mm.** The important factor is that **some space will be available** in the posterior part of the mouth. This leeway space serves to at least accommodate the permanent canines, which are generally larger than the primary canines.

During the **canine-premolar transition period,** the **permanent first molars** generally move mesially into the leeway space after the primary second molars are shed, this causing a **loss in arch length.** (This is referred to as **"the late mesial shift** of a permanent first molar").

Overbite is:

- The horizontal projection of the maxillary anterior teeth beyond the mandibular anterior teeth
- The vertical overlapping of the maxillary anterior teeth over the mandibular anterior teeth
- A malocclusion in which the anterior teeth do not close or come together
- A malocclusion where some of the maxillary teeth are inside of the mandibular teeth when they occlude

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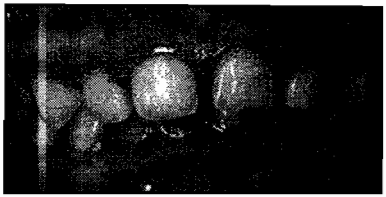
• **The vertical overlapping of the maxillary anterior teeth over the mandibular anterior teeth**



A case of overbite **(deep bite)** is demonstrated with this patient.

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Overjet is the horizontal projection of the maxillary anterior teeth beyond the mandibular anterior teeth *(labial axial inclination of the maxillary incisors).*

This is an example of excessive protrusion of maxillary incisors **(overjet).**

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Other terms to know include:

- **Physiologic** occlusion → although not necessarily an ideal or Class I occlusion, it is an occlusion that adapts to the stress of function and can be maintained indefinitely.
- **Pathological** occlusion → cannot function without contributing to it's own destruction. It may manifest itself by any combination of: excessive wear of the teeth without sufficient compensatory mechanisms,TMJ problems, pulpal changes ranging from pulpitis to necrosis and periodontal changes.

Note: Tooth movement caused by pathologic conditions is termed pathologic tooth movement.

A **phase of dentition** during which some of the teeth present in the oral cavity are **permanent** and some are **primary** is referred to as:

- Intermediate dentition
- Succedaneous dentition
- Mixed dentition
- Non-succedaneous dentition

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• **Mixed dentition**

The **earliest** indication of a mixed dentition consists of the **primary** dentition and the **permanent mandibular first molars.**

A **mixed dentition analysis** *(transitional dentition analysis)* determines **space available versus space required.** The analysis is based on a correlation of tooth size; one may measure a tooth or a group of teeth and predict accurately the size of the other teeth in the same mouth.

In the **Moyers' mixed dentition analysis,** the size of the unerupted canines and premolars is predicted from **knowledge of the size** *(mesiodistal width)* **of the mandibular incisors** that have already erupted into the mouth early in the mixed dentition. The maxillary incisors are not used in any of the predictive procedures, since they show too much variability in size.

Note: The **mandibular incisors** are measured to predict the size of maxillary as well as mandibular posterior teeth.

If mandibular anterior crowding is noted during the mixed dentition phase, the most appropriate approach to management is to take study models and perform an arch length analysis. This mandibular incisor crowding usually results from a tooth size-arch length discrepancy.

Remember: Supervision of a child's development of occlusion is most critical at ages 7-10 years *(mixed dentition).*

Which of the following is true concerning a **mixed dentition analysis?**

- It is used to predict the amount of crowding after the permanent teeth erupt
- It determines space available vs. space required
- The analysis is based on a correlation of tooth size
- It is performed during the mixed dentition
- It is performed with a boley gauge, study models and a prediction table
- All of the above are true concerning a mixed dentition analysis

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• **All of the above are true concerning a mixed dentition analysis**

Procedure for mixed dentition analysis:

1. Measure the mesial-distal diameter of the mandibular incisors and add them together
2. Measure the space available for the mandibular incisors
3. Subtract # 1 from # 2

***A **negative** number **indicates crowding** in the incisor region

4. Measure the space available for the canine and premolars on each side of the arch
5. Calculate from the prediction table the size of the canine and premolars
6. Subtract # 5 from # 4 on each side

***Once again, a **negative** number **indicates crowding**

***At this point, there will be **3 numbers**

• The number for incisor crowding or excess space
• The number for the right canine and premolar crowding or excess space
• The number for the left canine and premolar crowding or excess space

***Add the three numbers:** A **negative** number = crowding
A **positive** number = space

Note: For the **maxillary arch,** use the mandibular incisors to predict the size of the maxillary canines and premolars. Follow the same steps as described for **mandibular** teeth.

Which space maintainer is most often used when the primary first molar needs to be prematurely extracted?

- "Band and loop" space maintainer
- "Distal shoe" space maintainer
- "Lingual arch" appliance
- "Nance" appliance

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- **"Band and loop" space maintainer**

Space maintainers which replace one prematurely missing primary tooth include:

- The **"band and loop"** → used after unilateral loss of a primary first molar. It consists of a band which is cemented usually to a primary second molar. Attached to this band is a loop, which extends to the distal surface of the canine. The loop prevents the mesial migration of the primary second molar.

***Note:** Limited strength allows only single tooth-space maintenance.

This photograph shows an example of a fixed unilateral appliance on the maxillary left side for a seven-year-old patient. The photograph demonstrates the appliance after cementation. This appliance is referred to as a **band and loop** space maintainer and is a favorite among many clinicians.

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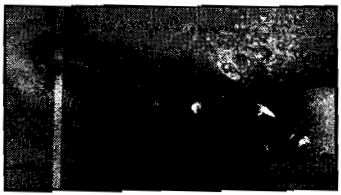

This appliance is called a **distal shoe** space maintainer or a distal extension space maintainer. It is used to prevent unerupted first permanent molars from moving mesially with the premature loss of second primary molars. The example shown is a crown with a distal extension segment soldered to the crown. The distal segment is extended into the tissue against the unerupted first permanent molar. The distal extension, also called a distal shoe, is used when the second primary molars are lost prior to the eruption of the first permanent molars *(i.e., very premature loss)*.

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A nine year old patient had an extraction of the primary mandibular first molar. The ideal treatment at this time is:

- Place a fixed bridge
- Place a space maintainer
- Place a removable partial denture
- Do nothing and observe
- Place an implant

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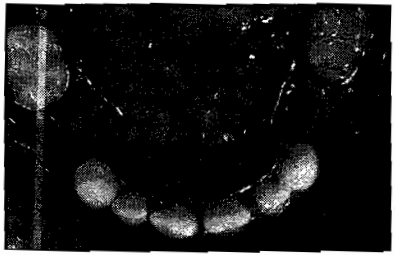
• **Place a space maintainer**

Although this can be done with either fixed or removable appliances, fixed appliances are preferred in most situations because they eliminate the factor of patient cooperation. If the space is unilateral, it can be managed by a unilateral fixed appliance *("band and loop" space maintainer).* If molars on both sides have been lost and the permanent incisors have erupted, it is usually better to place a **"lingual arch"** space maintainer *(See picture below).*



The **lingual arch** appliance, as demonstrated in the photograph, would be the appliance of choice for some clinicians in a situation where both primary first molars have been lost in the primary dentition. The bilateral appliance is very stable since it is anchored to two teeth.

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Notes:

1. Premature loss of a **primary maxillary second molar** usually produces a Class II molar relationship on the affected side. A **distal shoe** space maintainer may help alleviate this potential problem. This appliance extends backwards from a crown on the primary first molar and subgingivally to the mesial line of the unerupted first permanent molar, **thus preventing** mesial migration.
2. With the **"lingual arch"** space maintainer, the primary second molars or permanent first molars are banded. Typically, the "lingual arch" space maintainer is comprised of two bands which are cemented to the primary second molars or permanent first molars with a **loop** of **wire** that rests on the cingula of the incisors.

An 8 year old child with a pulpally involved **primary second molar** tooth comes into your office. Which of the following **ideally** is the best approach to manage the case?

- Prescribe antibiotics only
- Treat the pulp and retain the tooth as a space maintainer
- Extract the tooth. Space maintainer is not needed at this age
- Extract the tooth and construct a "band and loop" space maintainer
- Extract the tooth and construct a "distal shoe" space maintainer

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• **Treat the pulp and retain the tooth as a space maintainer**

The **key point** here is that no prefabricated space maintainer is as good as the natural tooth. Obviously in this case proper pulpal therapy followed by a restorative procedure would be needed on this tooth for it to function as a space maintainer.

The **natural tooth** will **preserve arch length and integrity** better than any prefabricated space maintainer. If a **primary tooth is lost,** an orthodontic evaluation is indicated to determine whether or not space maintenance is necessary. The decision is based on the patient's skeletal and dental development. For example, if a child is **dental age 10** and loses the primary first molar, no treatment is **usually** needed.

Remember: The permanent first premolar usually erupts between 10-12 years old.

Permanent Teeth Eruption Chart		
	Tooth	**Eruption**
Maxillary	Central	7-8 years
	Lateral	8-9 years
	Canine	11-12 years
	First premolar	10-11 years
	Second premolar	10-12 years
	First molar	6-7 years
	Second molar	12-13 years
	Third molar	17-21 years
Mandibular	Central	6-7 years
	Lateral	7-8 years
	Canine	9-10 years
	First premolar	10-12 years
	Second premolar	11-12 years
	First molar	6-7 years
	Second molar	11-13 years
	Third molar	17-21 years

In predicting the time of the pubertal growth spurt, while treating jaw mal-relationships in a growing child, the orthodontist can get the most valuable information from:

- A wrist-hand radiograph
- Height-weight tables
- Presence of secondary sex characteristics
- Stage of dental development

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• **A wrist-hand radiograph**

The **physiologic age or developmental age** can be judged by finding out the skeletal development. The wrist-hand radiograph offers the best aid for this purpose. By looking at the ossification and development of the carpal bones of the wrist, the metacarpals of the hands and the phalanges of the fingers the orthodontist can have an idea about the chronology of skeletal development. Comparing the overall pattern observed in the hand-wrist radiograph, with age standards in a reference atlas, does this.

Remember: The ulnar sesamoid or hamate bones are considered as landmarks to obtain an estimate of the timing of the adolescent growth spurt. Wrist-hand radiographs in the dental office can be obtained by using a standard cephalometric cassette and dental x-ray.

The state of physical maturity or skeletal development **co-relates** well with the jaw growth. Orthodontists use this information to predict how much jaw growth can be expected. **Note:** After sexual maturity much less growth is expected and therefore growth modification is not attempted.

Remember: Hand-wrist radiographs are less useful in evaluating whether growth has stopped or is continuing *(patient's position on growth curve)*. Serial Cephalometric radiographs are used for this purpose.

The anomaly depicted in the picture below is called a:

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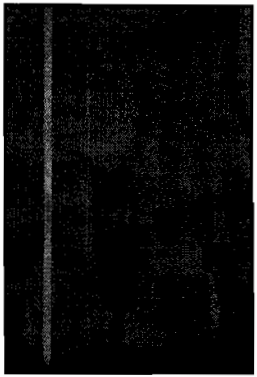
• **Mesiodens**

A midline supernumerary tooth *(mesiodens)* in the mandibular arch is shown. Mesiodens usually occur in the maxillary arch. However, you will see them occasionally in the lower arch. Note the crowding of the mandibular permanent incisors. Extraction of the mesiodens is the treatment of choice. Important: For the best therapeutic result, orthodontic treatment to close the space may be necessary.



A midline supernumerary tooth *(mesiodens)* is present. Note that the maxillary right permanent central incisor is *(slightly)* rotated and that the direction of the roots of the central incisors is more flared. The central incisors most likely were deflected from their normal paths of eruption by the mesiodens. The mesiodens should be extracted.

To localize a supernumerary tooth or impacted tooth and its relationship to other teeth, you should take two or more periapical x-rays at different angles and an occlusal view film.

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Conditions associated with **multiple** supernumerary teeth:

- Gardener's syndrome
- Down's syndrome
- Cleidocranial dysplasia
- Sturge-Weber syndrome

Note: Oligodontia / absence of one or more teeth, is more common in females than males. It is often associated with smaller than average tooth-size ratio.

A **contraindication** for serial extraction is:

- Class I skeletal pattern
- Deep bite
- Mixed dentition stage of dental development
- Severe crowding

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• **Deep bite**

Serial extraction is the orderly removal of selected primary and permanent teeth in a predetermined sequence. It is indicated primarily in **severe Class I malocclusion** in the mixed dentition that has insufficient arch length. This procedure primarily benefits children who demonstrate an arch-length discrepancy.

Stages in serial extraction: The primary canines are the first to be removed, followed by the primary first molars, and then the permanent first premolars *(usually)*. Six to fifteen months is the interval between extractions. To aid in support and retention during this time, a lingual arch should be used in the mandible and a Hawley appliance in the maxilla. This is usually followed by full orthodontic treatment. **Note:** The key to success is extraction of the first premolars before the permanent canines erupt.

In serial extraction procedures, concerns about **eruption sequence** are usually related to the eruption pattern of the **permanent mandibular canines and first premolars. Note:** After extraction of the maxillary first premolar in a serial extraction procedure, the maxillary canines path of eruption will usually be downward and backward.

Remember: Severe arch space deficiency in the permanent dentition *(over 10 mm)* **will almost always require extractions to properly align teeth.**

Arrange the following procedures into their proper sequence for **molar uprighting** of a tooth requiring both restorative and periodontal treatment?

- Band
- Complete restorative treatment
- Complete periodontal surgery
- Separate
- Upright

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- 1) Separate
  2) Band
  3) Upright
  4) Complete periodontal surgery
  5) Complete restorative treatment

A common dental condition that can benefit from orthodontic treatment prior to prosthetic treatment is the **long-term loss of a mandibular permanent first molar.** The loss of the first molar results in tipping, migration and rotation of the adjacent teeth into the edentulous space. **Note:** The best way to upright a second molar that had drifted mesially is by **tipping its crown distally** and opening up space for a pontic to replace the missing first molar, rather than attempting to move the second molar mesially to close the space.

Important: A high mandibular plane angle is one of the **most significant** complications of molar uprighting, because if the molar is uprighted unsuccessfully, it can lead to an increased open bite and loss of anterior guidance.

A **normal angulation** of a molar is desirable since it:
- Improves the direction and distribution of occlusal forces
- Decreases the amount of tooth reduction required for parallelism of the abutments
- Decreases the possibility of endodontic, periodontic or more complex prosthodontic procedures
- Increases the durability of the restorations, due to better force distribution
- Improves the periodontal environment by eliminating plaque-retentive areas
- Improves the alveolar contour
- Improves crown-to-root ratio

The **most rapid losses** in the A-P distance of the arch is usually due to a:

- Distal tipping and rotation of the permanent second molar after removal of the permanent third molar
- Mesial tipping and rotation of the permanent first molar after removal of the primary second molar
- Mesial tipping and rotation of the permanent canine after removal of the primary lateral incisor
- Distal tipping and rotation of the permanent second premolar after removal of the permanent first molar

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- **Mesial tipping and rotation of the permanent first molar after removal of the primary second molar**

Very Important: When the primary second molar is lost, always maintain space until the arrival of the second premolar.

Notes:

1. If a **permanent first molar is extracted** on a child **before** the eruption of the **permanent second molar,** the **best approach** is to allow the eruption of the second molar and the mesial drifting to occur naturally. This will fill in the space most of the time.
2. A space maintainer can be removed as soon as the permanent tooth begins to erupt through the gingiva. *(See picture below).*
3. The most reliable indicator of readiness of eruption of a succedaneous tooth *(and the need for a space maintainer)* is the extent of root development determined by radiographic evaluation. No space maintainer is required if eruption of the succedaneous tooth is imminent.

This photograph demonstrates a tooth partially erupting between the wires of a space maintainer. The space maintainer now can be removed.

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The displacement of a tooth from the socket in the **direction of eruption** is referred to as:

- Tipping
- Translation
- Extrusion
- Intrusion
- Torque
- Rotation

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• **Extrusion**

Six types of tooth movement that can be accomplished with orthodontics:

1. **Tipping** → the crown moves in one direction while the root tip is displaced in the opposite direction due to rotation or pivoting of the tooth around the axis of resistance or axis of rotation *(located somewhere in the apical one-third of the root)*. Most readily accomplished with a removable appliance. **Accomplished most easily with anterior incisor teeth.**
2. **Translation** *(bodily movement)* → coupled force is applied to the crown to control root movement in the same direction as crown movement *(force is applied through the tooth's center of resistance)*. **Very difficult to accomplish.**
3. **Extrusion** → displacement of the tooth from the socket in the direction of eruption.
   Intrusion → movement into the socket along the long axis of the tooth. **Very difficult to accomplish.**
4. **Torque** → controlled root movement labiolingually or mesiodistally while the crown is held relatively stable *(mesial-distal root movement is also termed "uprighting")*.
5. **Rotation** → revolving the tooth around its long axis. Recurring tooth rotations after orthodontic correction occur because of the persistence of the elastic supracrestal gingival fibers *(mainly free gingival and transseptal fibers)*. **Need adequate retention to prevent relapse.**

Note: On the side **toward** which the tooth is being moved, you will find **"osteoclasts"** *(break down bone)* and on the side of the root **from which the** tooth moves, you will find **"osteoblasts"** *(bone-forming cells)*.

Post-orthodontic **circumferential supracrestal** fibrotomy is most often performed in which situation?

- An intruded mandibular second molar
- A rotated maxillary lateral incisor
- An extruded maxillary second premolar
- A mandibular first molar that is in crossbite

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• **A rotated maxillary lateral incisor**

One of the most important aspects of orthodontic therapy is **retention.** After malposed teeth have been moved into the desired position, they must be mechanically supported until the hard and soft tissues have been thoroughly modified — both in structure and in function — to meet the demands of the new position. Once the desired occlusal results are achieved and the hard tissues are in normal function, the next step is to maintain or to modify the soft tissues in the retention phase. **Important:** Most clinicians believe that the **collagen fibers in the supra-alveolar tissue** are significantly responsible for the relapse of orthodontically **rotated teeth** as well as the redevelopment of spaces between orthodontically moved teeth.

Remember: Collagen fibers are the primary components of the attached gingiva. When teeth are orthodontically moved, the fibers stretch like rubber bands to adjust to the new position. **However,** like rubber bands, they have a strong tendency to return to their former position, pulling teeth with them as they go.

The **circumferential supracrestal fibrotomy** is a minor surgical procedure. A simple incision in the sulcus is made to the crest of the bone. This incises all of the collagen fibers that are inserted into the root of the tooth. By cutting the collagen fibers, **two** things are accomplished:

1. Eliminate the potential for relapse due to collagen fiber retraction.
2. Allow new fibers to form that will help retain the tooth in its new position.

The rationale for **retention** in orthodontics *(accomplished with fixed or removable retainers)* is to:

- Allow for reorganization of the gingival and periodontal tissues
- Minimize changes due to growth
- Maintain teeth in unstable conditions
- All of the above

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• **All of the above**

Maintaining the treatment result following orthodontic treatment is one of the most difficult aspects of the entire treatment process. Retention is necessary in orthodontics for the following reasons:

1. **The gingival and periodontal tissues are affected** by orthodontic tooth movement and require time for reorganization when the appliances are removed.
2. **Changes produced by growth may alter** the orthodontic treatment result.
3. **The teeth may be in an inherently unstable position** after the treatment, so that the soft tissue pressures constantly produce a tendency for relapse.

In the last situation, gradual withdrawal of an orthodontic appliance is of no value. The only possibilities are accepting relapse or using permanent retention. **Fortunately,** only the first two reasons apply to most orthodontic patients, and maintaining the position of the teeth until remodeling of the supporting tissues is completed and growth has essentially ceased allows a stable orthodontic result without further retention.

Note: Retention is accomplished with either fixed or removable retainers.

Remember:

- **Anterior crossbite** is easily retained after orthodontic correction by the overbite achieved during treatment.
- **Supracrestal fibers** are commonly associated with relapse following orthodontic rotation of teeth.

Lateral cephalometric radiographs of a typical growing patient were taken several years apart, and superimposed on the anterior cranial base. The result showed that during growth the maxilla and mandible move:

- Downward and backward
- Downward and forward
- Upward and backward
- Upward and forward

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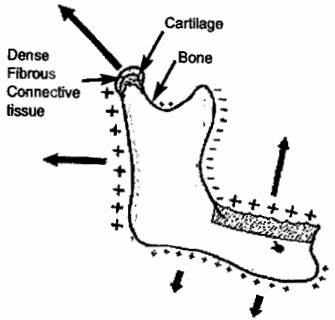
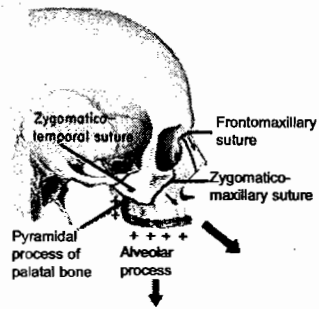
- **Downward and forward**



Mandibular growth is in the downward and forward directions. Growth in the **condyle** increases the anterior-posterior *(downward and forward pattern of growth)* dimension of the mandible. Anterior-posterior dimension of the mandible is also increased by resorption of bone on the anterior border of the ramus and apposition of bone on the posterior border of the ramus. Appositional growth of alveolar bone increases the superior-inferior dimension of the mandible.



This graphic shows the **maxillary** growth sites. The primary centers of growth contributing to the downward and forward direction of the maxilla are the following:

- Growth at the spheno-occipital and sphenoethmoidal junctions
- Growth at the nasal cartilaginous septum

The following sutures are considered secondary or accommodating growth sites for the primary centers of growth:

- Frontomaxillary suture
- Zygomaticotemporal suture
- Pyramidal process of palatal bone
- Alveolar process

The **flat bones** of the skull and part of the clavicle are formed by:

- Intramembranous ossification
- Endochondral ossification
- Erythropoiesis
- Epiphyseal formation

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• **Intramembranous ossification**

Bone formation begins in the embryo where **mesenchymal cells** differentiate into either fibrous membrane or cartilage. This leads to **two paths** of bone development:

1. **Intramembranous ossification** is so called because it takes place in membranes of connective tissue. **Osteoprogenitor** cells in the membrane differentiate into osteoblasts; a collagen matrix is formed which undergoes ossification.
   Note: The **maxilla** and **mandible** are formed this way.

2. **Endochondral ossification** is how the remainder of the skeleton forms and takes place within a hyaline cartilage model. Cartilage cells are replaced by bone cells *(osteocytes replace chondrocytes)*, organic matrix is laid down and calcium and phosphate are deposited. This type of ossification is principally responsible for the formation of short and long bones. **Note:** The ethmoid, sphenoid and temporal bones form this way.

Remember: Once bone is formed, it **grows by appositional growth** *(which is growth by the addition of new layers on those previously formed).*

Hyaline cartilage **differs** from bone in that hyaline cartilage may grow:

- By appositional growth
- By interstitial growth
- Neither of the above

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- **By interstitial growth**

Growth of cartilage occurs in two ways:

1. **Appositional** by the recruitment of fresh cells, chondroblasts, from perichondral stem cells and the addition of new matrix to the surface.
   Note: The perichondrium consists of a fibrous outer layer and chondroblastic inner layer.

2. **Interstitial** by the mitotic division of, and deposition of more matrix around, chondrocytes already established in the cartilage. Examples of sites that grow by interstitial growth include the **mandibular condyle,** nasal septum and spheno-occipital synchondrosis.

Growth of bone:
Appositional → below the covering periosteal layer of bone. Periosteum consists of a fibrous outer layer and a cellular inner layer of osteoblasts, which lay down bone. **Because of its rigid structure, interstitial growth is not possible.**

(Do not confuse bone growth with bone formation. Bone forms by either endochondral ossification or Intramembranous ossification).

In a young child, which structure grows in **height and length** to accommodate the developing dentition?

- The tuberosity
- The ramus
- The condyles
- The alveolar process

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• **The alveolar process**

The bone of the alveolar process **exists only to support the teeth.** If a tooth fails to erupt, alveolar bone never forms in that area; and if a tooth is extracted, the alveolus resorbs after the extraction until finally the alveolar ridge completely atrophies.

The **space between the jaws** into which the teeth erupt is generally considered to be provided by **growth at the mandibular condyles** *(especially the molars)*. The condyle is a **major site** of vertical growth in the mandible. Many arguments have been made about the condyle's function in mandibular growth. Most authorities agree that soft-tissue development carries the mandible forward and downward, while **condylar growth** fills in the resultant space to maintain contact with the base of the skull.

In infancy, the ramus is located at about the spot where the primary first molar will erupt. Progressive posterior remodeling creates space for the second primary molar and then for the sequential eruption of the permanent molar teeth. More often than not, however, this growth ceases before enough space has been created for eruption of the third permanent molar, which becomes impacted in the ramus. **Note: After age 6, the greatest increase in size of the mandible occurs distal to the first molars.**

Remember: Resorption occurs along the anterior surface of the ramus *(creates space for mandibular molars)*. **Apposition** occurs along the posterior surface of the ramus.

Bone deposition in the _____region is responsible for the lengthening of the maxillary arch.

- Palate
- Tuberosity
- Incisor
- Zygomatic

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• **Tuberosity**

The maxillary arch elongates, moves in a posterior direction, and increases in height. Bone deposition in the **tuberosity region** is responsible for the lengthening of the arch *(elongation)*. The movement in a posterior direction is a result of resorption of the labio-alveolar surface and apposition of the lingual surface. **Alveolar growth** is responsible for an increase in height of maxillary bones.

Posterior movement **predominates** in the area of the tuberosity. The principal movement of the alveolar region and palate is downward, the nasal region moves forward and the zygomatic process moves posteriorly and laterally.

Growth of the maxilla and its associated structures occurs from a **combination** of growth at the sutures and direct remodeling of the surface of the bone.

Mandibular anterior crowding occurs in normal young adults as a result of :

- Third molars
- Late mandibular growth
- Maxillary tooth-size excess
- Trauma

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• **Late mandibular growth**

The current concept is that **late incisor crowding** develops as the mandibular incisors, and perhaps the entire mandibular dentition, move distally relative to the body of the mandible late in mandibular growth.

Late incisor crowding **does occur** in individuals with no third molars at all, and so the presence of these teeth is not a critical variable → the extent of late mandibular growth is a critical variable.

Remember: The **mandible** can and does undergo **more growth** in the late teens than does the maxilla.

What percentage of **6 year-old children** have a median *(maxillary)* diastema?

- 98%
- 78%
- 49%
- Less than 25%

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• **98%**

***It is prevalent in 49% of 11 year-old children.

The cause of a median diastema could be any of the following: a tooth-size discrepancy, a mesiodens, an abnormal frenum attachment, or a normal stage of development.

The **spaces tend to close** as the permanent canines erupt. The greater the amount of spacing, the less the likelihood that a maxillary central diastema will totally close on its own. As a **general guideline,** a maxillary central diastema of 2 mm or less will probably close spontaneously, **while** total closure of a diastema initially greater than 2 mm is unlikely. If the space is 2 mm or less and the maxillary laterals are in a good position, it is most likely the result of a normal developmental process.

If it is caused by an **abnormal frenum,** it is best to align the **teeth orthodontically and then do a frenectomy.** Usually this is not done until the permanent canines erupt.

Accepted methods of closing a diastema:
- Using a lingual arch with finger springs
- Using a Hawley appliance with finger springs
- Using cemented orthodontic bands with inter-tooth traction

In which direction do the permanent teeth **move** during eruption?

- Mesially and occlusally
- Occlusally and buccally
- Buccally and mesially
- Occlusally and lingually

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• **Occlusally and buccally**

Permanent teeth move **occlusally and buccally** while erupting. Also, during active tooth eruption there is apposition of bone on all surfaces of the alveolar crest and on the walls of the bony socket.

Remember: The maxillary arch is **slightly longer** in length compared to the mandibular arch. The reason is the sum of the M-D diameter of the **maxillary permanent teeth is approximately 128 mm,** whereas the sum of the M-D diameter of the **mandibular permanent teeth is approximately 126 mm.**

The most commonly impacted teeth are:

- Maxillary canines
- Maxillary central incisors
- Mandibular first premolars
- Mandibular lateral incisors

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• **Maxillary canines**

Failure of a permanent tooth to erupt **may cause** damage to roots of other teeth and also **create** a severe orthodontic problem. Orthodontic consultation is indicated when first observed on an x-ray. An impacted canine or other tooth in a teenage patient can usually be brought into the arch by orthodontic traction after being surgically exposed. **In older patients, there is an increasing risk that the impacted tooth has become ankylosed.** Even adolescents have a risk that surgical exposure of a tooth will lead to ankylosis.

In treatment planning for an **impacted tooth,** three principles should be followed:

1. The prognosis should be based on the **extent of displacement** and the **surgical trauma** required for exposure.
2. During **surgical exposure, flaps** should be reflected so that the tooth is ultimately pulled into the arch **through keratinized** tissue, **not through alveolar mucosa.**
3. **Adequate space** should be provided in the arch before attempting to pull the impacted tooth into position.

Note: Research suggests the association of impacted canines with missing lateral incisors or shortened roots of lateral incisors. The distal aspect of the root of lateral incisors guides the eruption of canines.

Treatment of the **ectopic eruption** of a permanent maxillary first molar consists of:

- Disking the distal of the primary first molar
- An appliance incorporating a finger spring to move the primary second molar mesially
- A brass wire placed between the primary second molar and permanent first molar
- Extraction of the primary second molar

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• **A brass wire placed between the primary second molar and permanent first molar**

This **separating device** *(brass wire)* will cause the permanent first molar to be tipped distally.

Ectopic eruption occurs when a tooth **erupts in the wrong place.** It is most likely to occur in the eruption of maxillary first molars and mandibular incisors. Its occurrence is much more common in the maxilla and is often associated with a developing skeletal Class II pattern. It is seen in about 2-6% of the population and spontaneously corrects itself in about 60% of cases.

If the eruption path of the **maxillary first molar** carries far too mesially at an early stage, the permanent molar is unable to erupt and the root of the primary molar may be damaged. The mesial position of the permanent molar means that the arch will be crowded unless the child receives treatment. **Remember:** This mesially inclined position of the permanent molar makes it susceptible to decay. If it shows signs of caries, extract the adjacent primary second molar immediately. The resultant space can then be maintained as part of orthodontic treatment.

Ectopic eruption of **mandibular lateral incisors,** which occurs more frequently than mandibular first molars, may lead to transposition of the lateral incisor and canine. A poor eruption direction of the canine, sometimes leading to impaction, is observed often but usually is due to the eruption path being altered by a lack of space.

The time required to **upright a molar** can vary from:

- 2-3 weeks
- 1-2 months
- 6-12 months
- 2-3 years

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• **6-12 months**

***A severely tipped molar or one that requires mesial movement to shorten the pontic space requires a longer treatment time.

A **fixed edgewise orthodontic appliance** is usually used for molar uprighting. The bracket slot size of 0.022 inch allows a wide range of wire sizes to be used. The alternate slot size is 0.018 inch, which can also upright the molar, but limits the wire sizes available. The **tipped second molar should be banded** because of the considerable posterior masticatory forces produced can easily shear off bonded brackets.

Facts about molar uprighting:

- A severely lingually tipped mandibular molar is **more difficult** to control and upright properly.
- Molar uprighting treatment in **high angles cases** will tend to result in excessive bite opening *(increases vertical dimension of occlusion).*
- **Stabilization** should last until the **lamina dura and PDL reorganize.** This ranges from approximately 2 months *(simple uprighting)* to 6 months *(uprighting plus osseous surgery, grafts, etc.).*
- **Retention** *(stabilization)* can be provided by an appliance or by a well-fitting provisional restoration,which will stabilize the tooth positions. This will allow for reorganization of the PDL.

Slow progress in molar uprighting in an adult patient is **most likely** due to occlusal interferences.

Match the dental arch relationships on the left with the correct depiction of the relationship on the right.

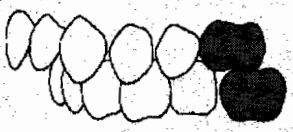
Normal occlusion

Class I malocclusion

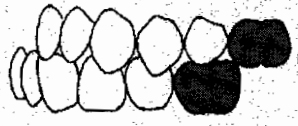
Class II malocclusion

Class III malocclusion

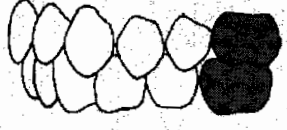
A



B



C



D

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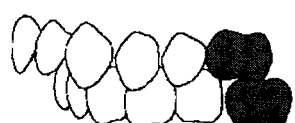
Normal occlusion = C

Mesiobuccal groove of the mandibular first permanent molar aligns with the mesiobuccal cusp of the maxillary first permanent molar.

Class I malocclusion = D

Class I malocclusion has the normal molar relationship but the incorrect line of occlusion.

Class II malocclusion = A



Class II malocclusion has the mandibular molar placed behind the maxillary molar.

Class III malocclusion = B

Class III malocclusion has the mandibular molar placed forward to the maxillary molar.

Malocclusion is most often:

- Caused by trauma
- Acquired from a friend
- Hereditary
- Caused by antibiotics
- Caused by bad habits

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• **Hereditary**

There may be a disproportion between the size of the maxilla and mandible or between the jaws and tooth size resulting in overcrowding of teeth or in abnormal bite patterns. Supernumerary teeth, malformed teeth, impacted or lost teeth and teeth that erupt in an abnormal direction may contribute to malocclusion. Less frequent causes of malocclusion include habits such as thumb sucking or tongue thrusting.

Signs of incipient malocclusion:

- The lack of interdental spacing in the primary dentition
- The crowding of the permanent incisors in the mixed dentition
- The premature loss of the primary canines, particularly in the mandibular arch

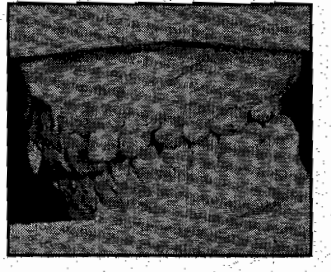
Notes:

1. The significance of the lack of spacing relates to the increased mesio-distal width of the permanent teeth.
2. Since arch perimeter increases after the eruption of the incisors and is small in the maxilla and essentially non-existent in the mandible, arch growth can not usually contribute to further dental alignment.
3. The premature loss of the mandibular primary canine reflects insufficient arch size in the anterior region. As such, the crowns of the lateral incisors, during eruption, impinge on the roots of the primary canines causing them to resorb. When the canine is shed, the midline will shift in the direction of the lost tooth. You will have lateral and lingual migration of the mandibular incisors.

Remember: An **anterior crossbite** in a primary dentition usually indicates a **skeletal** growth problem.

Which Angle Classification does the arrangement of the anterior teeth in this frontal view of study casts most likely represent?

- Class I
- Class II, Division 1
- Class II, Division 2
- Class III



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• **Class II, Division 2**

Class II, Division 2 is a **malocclusion** in which the body of the mandible and its superimposed dental arch are also in **distal relationship** to the maxilla, and the molar and canine occlusion are the same as Class II, Division 1 type. The **distobuccal cusp** of the maxillary first molar occludes in the buccal developmental groove of the mandibular first molar, and the maxillary canines occlude mesial to the mandibular canines. The big difference between Division I and Division II is in **Division II** the maxillary laterals have tipped labially and mesially.

Remember: Class II, Division 1 = maxillary incisors *(centrals and laterals)* are in **extreme labioversion.**

Notes:

1. There is **no set rule as** to when a malocclusion should be treated. The age at which it is treated depends on the problem involved.
2. Malocclusions are more identifiable in children **7 to 9 years old** because the eruption of permanent incisors reveals tooth-arch length discrepancies.

Which type of malocclusion listed below is most often associated with mouth breathing?

- Dental open bite
- Skeletal open bite
- Dental cross bite
- Skeletal cross bite

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• **Skeletal open bite** → sometimes called the "Long Face Syndrome"

The following factors **are associated** with chronic mouth breathing:

• Narrow face

• Narrow oropharyngeal space

• Chronic rhinitis → inflammation of the mucous membranes of the nose

• Chronic tonsillitis

• Allergies

• Deviated nasal septum

Note: The **earliest possible diagnosis** of this open bite is essential because the condition is **not self-correcting** and usually worsens with time. **Anterior open bites** can be classified as a form of **apertognathism** *(which means open bite deformity).*

Which cusp of the **maxillary first molar** serves as a **reference point** in identifying Angle's Class I, II and III occlusions?

- Distobuccal
- Mesiobuccal
- Mesiolingual
- Distolingual

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• **Mesiobuccal**

Classification of Human Occlusion (Angle's)

Class I → most common *(about 70 % of the population).* The mesiobuccal cusp of the maxillary first molar lines up **approximately with** the buccal groove of the mandibular first molar. The maxillary central incisors overlap the mandibulars. Maxillary canine lies **between** the mandibular canine and first premolar. Class I is associated with an orthognathic facial profile where the nose, lips, and chin are harmoniously related. It is most commonly caused by a discrepancy between tooth structure and the amount of supporting bone length.

Class II → less common *(about 25 %).* The mesiobuccal cusp of the maxillary first molar falls approximately **between** the mandibular first molar and the second premolar. The lower jaw and chin may also appear **small and withdrawn.** The mandibular incisors occlude even more posterior to the maxillary incisors so they may not touch at all. Maxillary canine is **mesial** to mandibular canine. Class II is associated with a retrognathic facial profile.

Class III → the **least common** *(less than 5 %).* The mesiobuccal cusp of the maxillary first molar falls approximately **between** the mandibular first molar and second molar. The chin may also protrude like a **bulldog's does.** The mandibular incisors overlap **anterior** to the maxillary incisors. The maxillary canine is **distal** to mandibular canine. Class III is associated with a prognathic facial profile.

Planes of space used to classify malocclusion:

- Antero-posterior
- Transverse
- Vertical

What cephalometric analysis measurement is characteristic in **Class I** malocclusions?

- SNA angle of > 84°
- SNB angle of < 78°
- ANB angle of < 4°
- None of the above

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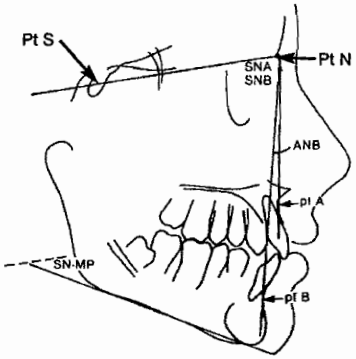
• **ANB angle of < 4°**

*** **Remember:** An **SNA** Angle of > 84° indicates maxillary prognathism, An **SNB** angle of < 78° indicates mandibular retrognathism and an **ANB** angle of < 4° indicates a harmonious skeletal profile. **Note:** The ANB angle describes the relation of the maxillary and mandibular denture bases.

Severe malocclusion may compromise all aspects of oral function. There may be difficulty in masticating if only a few teeth meet, and jaw discrepancies may force adaptive alterations in swallowing. It can be difficult or impossible to produce certain sounds in the presence of severe malocclusion, and speech therapy may require some preliminary orthodontic treatment. Referral to a speech therapist is **helpful** because both patient and parents are likely to benefit from the counseling. Even less severe malocclusions tend to affect mastication, swallowing and speech; not so much by making the function impossible as by requiring physiologic compensation for the anatomic deformity.



Class II malocclusion is usually associated with which facial profile?

- An orthognathic profile
- A retrognathic profile
- A prognathic profile
- None of the above

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• **A retrognathic profile**

The **convexity** is due to the relative prominence of the maxilla compared to the mandible. The mandibular incisors will most likely be tipped forward.

An **orthognathic** profile is one in which the nose, lips and chin are harmoniously related. This relationship is usually accompanied by a **Class I dental occlusion.**

A **prognathic** profile is one in which the mandible is markedly forward of the maxilla giving a **concave** midfacial appearance. This is often indicative of a **Class III malocclusion.** The maxillary incisors will most likely be tipped lingually.

Important: A **bimaxillary dentoalveolar protrusion** means that in both jaws the teeth protrude. This condition is seen in facial appearances in 3 ways: excessive separation of the lips at rest *(incompetence),* severe lip strain *(needed to bring the lips into closure),* and prominence of lips in the profile view.

Note: As children **mature** their profiles become **less convex.**

Class II malocclusion is also referred to as:

- Retrognathism or overbite
- Prognathism or underbite
- Neither of the above

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- **Retrognathism or overbite**

Those malocclusions in which there is a **"distal"** relationship of the mandible to the maxilla make up **Class II.**

Divisions are as follows:

- **Class II, Division 1** → a distal relationship of the buccal groove of the mandibular first permanent molar to the mesiobuccal cusp of the maxillary first permanent molar **along with the maxillary incisors** *(centrals and laterals)* in extreme labioversion *(protruded).*

- **Class II, Division 2** → a distal relationship of the buccal groove of the mandibular first permanent molar to the mesiobuccal cusp of the maxillary first permanent molar **along with the maxillary laterals being tipped labially and mesially** *(sometimes actually overlapping the centrals).* The maxillary centrals are usually retruded somewhat.

***Subdivisions** → when the distoclusion occurs on one side of the dental arch only, the unilateral distoclusion is referred to as a subdivision of its division. For example:

- **Class II, Division 1 Subdivision** → **one side** of the maxillary arch is in a Class II relationship with its occluding mandibular quadrant while the **other side** is in a Class I relationship. The protruded maxillary incisors *(centrals and laterals),* maxillary overjet, and other anterior aberrations, are usually confined to one side of the maxillary arch.

Which of the following is the **least** common?

- Class I malocclusion
- Class II malocclusion
- Class III malocclusion
- Normal occlusion

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• **Class III malocclusion**

Class III malocclusions are those in which the body of the mandible and its superimposed dental arch are in a **mesial** relationship to the skull base and maxilla. The maxillary first molar therefore occludes **distal** to the mandibular first molar, while the maxillary canine is an exaggerated distal relationship to the mandibular canine. The mandibular incisors are usually tipped lingually and **forward** to the maxillary incisors. Also characteristic of the **"true"** Class III malocclusion is the **prognathic mandible.** Class III **subdivision** is a Class III relationship of the teeth on **one side** with a Class I relationship on the other side.

A **psuedo-class III** malocclusion is one in which the mandibular incisors are forward of the maxillary incisors when in centric occlusion, however, the patient has the ability to bring the mandible back **without strain** so that the mandibular incisors can touch the maxillary incisors *(this ability is often considered diagnostic).* This type is therefore a **milder form of the "true"** Class III malocclusion and more amenable to conservative orthodontic movement than the "true" Class III malocclusion which often requires surgical correction.

Which Angle Classification is shown in this set of study casts?

- Class I
- Class II, Division 1
- Class II, Division 2
- Class III

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• **Class II, Division 1**

In most cases **Class II, Division 1 malocclusions,** the body of the mandible and its superimposed dental arch are in a **distal relationship** to the maxilla and the maxillary incisors are usually in a labial axial inclination. In addition, the relationship of the maxillary first molars and canines to their mandibular counterparts is such that the **distobuccal cusp** of the maxillary first molar occludes in the buccal developmental groove of the mandibular first molar and the **maxillary canines occlude mesial to the mandibular canines.** Besides the labial axial inclination of the maxillary incisors *(overjet),* various aberrations in the individual alignment of the teeth *(for example, crowding)* can be superimposed upon this class.

Class II, Division 1 Subdivision includes malocclusions, which have **one side** of the maxillary arch in a Class II relationship with its occluding mandibular quadrant, while the other side is in a Class I relationship. The maxillary overjet or other anterior aberrations are usually confined to **one side** of the maxillary arch.

Note: Relative to a heterogeneous population, the incidence of malocclusion in a homogeneous population generally is lower.

The existence of a **forward shift** of the mandible during closure to avoid incisor interference is found in:

- "True" Class III malocclusions
- "Pseudo" Class III malocclusions
- "Sunday bite"

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• **"Pseudo" Class III malocclusions**

In order to **avoid** the interference of teeth, a patient may adopt a **jaw position** on closure, which is **forward to normal.** This may look like a Class III position in the absence of a true skeletal Class III relationship. Hence termed "pseudo Class III malocclusion". In most cases they have an edge-to-edge bite. Mandibular incisors are forward of maxillary incisors in centric occlusion but the patient can bring the mandible back without strain so that the mandibular incisors touch the maxillary incisors. This is a milder form of the true class III malocclusion and is more amenable to conservative orthodontic movement, while true class III malocclusion often requires surgical correction.

Treatment involves the **elimination of the CO-CR discrepancy** early in the course of treatment to avoid abnormal wear and abnormal growth influences. Occlusal interferences and anterior cross bites are dealt with subsequently.

To **figure out whether a patient** has a Class III or Pseudo Class III malocclusion. You would gently take the patients chin and try to push the mandible back into the retruded position or centric position. If the patient has a pseudo class III malocclusion, when you push the patients jaw back into the retruded position you will find that the patients anterior teeth will be "edge on edge." If, however, you try to push the jaw back into the retruded position or centric relation and it doesn't go back at all, then you know that you have a true class III malocclusion.

Note: The **"Sunday bite"** is a term given to the forward postural position of the mandible which is adopted by people with class II profiles in an effort to improve their esthetics.

Difficulty pronouncing "f" and "v" sounds is **most likely associated** with which type of malocclusion?

- An anterior open bite
- A Class II Division I malocclusion
- A Class II Division II malocclusion
- A Skeletal Class III malocclusion

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- **A Skeletal Class III malocclusion**

***Speech is affected in severe malocclusions along with other oral functions *(i.e., swallowing and mastication).*

The cephalometric analysis of the **Class I occlusion** would indicate an ANB angle of less than 4 degrees signifying a harmonious skeletal profile and sagittal harmony between the maxillary and mandibular dental arches. The **most common cause** of Class I malocclusion is a discrepancy between tooth structure and the amount of supporting bone (length). Perhaps the **most prevalent** characteristic of Class I malocclusion is **crowding** *(i.e., insufficient alveolar arch length to accommodate all teeth in ideal alignment and in a good sagittal position).*

When a diagnosis is made that **crowding does exist** and this crowding exceeds 4 mm in the mandibular arch, extractions are often required to attain an excellent, stable result. **However,** the decision whether to extract teeth depends greatly on a space analysis performed on the mandible. **The patient should be referred to the orthodontist for this analysis.**

In general:

- When the space lacking is **less than 4mm,** in most cases it can be obtained by carefully stripping some interproximal enamel from each of the anterior teeth.
- A space deficiency **exceeding 4mm** usually indicates extraction for correction of the malocclusion.

All of the following are **extra** oral components of the headgear **except:**

- Neck strap
- Chin cup
- Face-bow
- Head cap

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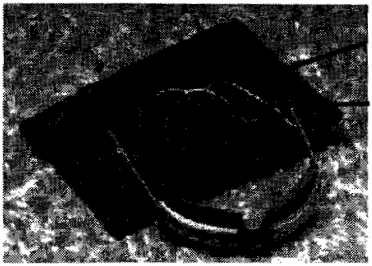
• **Face-bow**

The face-bow is the intra-oral component of the headgear. It has an outer and inner bow. The length and position of outer bow is set according to the inner bow. The inner bow relates to the center of resistance of the tooth and effects anchorage and / or traction.



Headgear is used for the maintenance of anchorage extra-orally. But it can also be employed for extra oral traction. Any one of the four basic types *(cervical-pull, straight pull, high pull, and reverse pull)* is selected by identifying the required direction of force *(pull)* needed.

Remember: For use with URA, the direction of force above the occlusal plane aids in retention.

The approximate required-force for anchorage → 250g for 10hr / day; for traction → 500g for 14-16hr / day.

Note: One of the greatest advantages of using extraoral anchorage *(i.e., headgear)* is that it permits posterior movement of teeth in one arch without adversely disturbing the opposite arch.

All of the following are fixed orthodontic appliances **except:**

- Lingual archwires
- Whip-spring appliances
- Palate-separating devices
- Frankel's appliances
- Edgewise mechanisms
- Light-wire appliances

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• **Frankel's appliances**

Frankel's appliance is a **removable functional appliance** and is employed in cases of abnormal *(hyper-active)* soft tissue patterns. **Whip-spring appliances** are used to de-rotate one or two teeth.

Fixed orthodontic appliances offer **controlled** tooth movement in all 3 planes of space. **Examples include:** lingual archwire, fixed space maintainers, palate-separating devices, the edgewise mechanism, light-wire appliances as well as other fixed appliances *(i.e., twin-wire appliance, universal appliance).*

Removable orthodontic appliances are generally restricted to **tipping** teeth.

Examples include:
• **Attached** removable appliances
1. **Active** appliances
    a. Extra-oral traction devices → head gears, face masks, chin cups
    b. Lip bumpers
    c. Active plates → Schwartz appliance, anterior spring aligners
    d. Vacuum formed appliances
2. **Passive** appliances
    a. Bite planes, occlusal splints, retainers
• **Loose** removable appliances → functional appliances, functional jaw orthopedic appliances

Note: For an orthodontic appliance to be effective in translating the roots of teeth, it must be capable of exerting a torque.

Remember: The 4 basic components of fixed appliance include: bands, brackets, archwires, and auxilliaries *(elastics or ligatures to hold the archwire in brackets).*

Which of the following alloys are used for archwires in orthodontics?

- Stainless steel
- Chromium-cobalt
- Titanium
- All of the above

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• **All of the above**

The properties of stainless steel wires used for archwires can be controlled over a wide range by varying the amount of cold working annealing during manufacturing. Steel is softened by annealing and hardened by cold working *(work hardening).*

Chromium-cobalt alloys have the advantage that they can be supplied in a softer and therefore more formable state, and that they can be hardened by heat treatment after being shaped. The heat treatment increases strength significantly.

Titanium alloys offer a highly desirable combination of strength and springiness and reasonably good formability.

The properties of an **ideal wire material** for orthodontic purposes can be described largely in terms of the following criteria. It should possess: **1)** high strength, **2)** low stiffness, **3)** high range and **4)** high formability. In addition, the material should be weldable or solderable, so that hooks or stops can be attached to the wire. **Loops** and **helices** are incorporated into archwires to **increase the activation range.**

The **stiffness** of orthodontic wires is a function of the length of the wire, the diameter of the wire and the alloy composition.

Which appliance is probably the most widely used today by orthodontists?

- The Begg appliance
- The edgewise appliance
- The universal appliance
- None of the above

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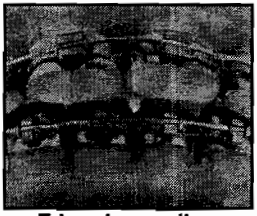
• **The edgewise appliance**

In its **essential form,** this mechanism consists of bands on all the teeth, tubes on the last molar and brackets on all other teeth. One labial arch is used at a time. The ultimate labial arch wire is .0125 x .028 in diameter and the narrow dimension or edge fits precisely into the bracket slot, which is .022 inches wide from top to bottom. **It finds its greatest application in the treatment of comprehensive malocclusions of the adolescent permanent dentitions.**

Variations of the **basic edgewise appliance** include the use of double or tandem brackets *(Siamese twin brackets)* and narrow slotted brackets, .018 from top to bottom. A straight wire appliance is a version of the edgewise appliance with several features that allow placement of an ideal rectangular archwire without bends.

Components of an edgewise appliance:

- **Siamese twin bracket** → for use on maxillary anterior teeth
- **Broussard buccal tube** → to allow for the use of the segmented arch technique, which is necessary to intrude teeth
- **Straight wire bracket**
- **A bracket with a .022 x .028 rectangular slot.** The bracket slot size of .022" allows a wide range of wire sizes to be used. The alternate slot size is .018", which can also upright molars but limits the wires sizes available. Tipped second molars should be banded because considerable posterior masticatory forces produced can easily shear off bonded brackets.



Edgewise appliance

Notes:

1. A **first order bend** in an orthodontic wire is in the **horizontal plane.**
2. The **Begg appliance** uses round wires which fit loosely in the vertical slot of the bracket.

What is the **most common** removable retainer used in orthodontics? A picture of this type of retainer is shown below.

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- **Hawley retainer → It incorporates clasps on molar teeth and a characteristic outer bow with adjustment loops, spanning from canine to canine.**

The palatal coverage of a removable plate like a Hawley retainer makes it possible to incorporate a bite plane lingual to the upper incisors to control the bite depth. This design consideration is important for any patient who once had an excessive overbite. This **palatal coverage** *(acrylic)* **is the major source of anchorage in the Hawley appliance.**

A Hawley retainer can be made for the **upper or lower** arch. The lower retainer is somewhat fragile and may be difficult to insert because of undercuts in the premolar region. A patient may have **difficulty pronouncing** linguoalveolar consonants for a few days after receiving a maxillary Hawley appliance **until the tongue** adapts to the palatal coverage.

Major components of a removable appliance:

1. **Retentive component** → retains the appliance in function: consist of various clasps. The best example is Adam's crib
2. **A framework or baseplate** → usually acrylic. This provides anchorage
3. **Tooth-moving elements** → typically either springs or screws
4. **Anchorage component** → resists force of active components *(e.g., Acrylic base-plate)*
5. **Active components or tooth moving components** → springs, screws or elastics

Indications for removable appliances:

- **Retention** after comprehensive treatment
- Limited **tipping** movements
- **Growth modification** during the mixed dentition

Note: Components of anchorage can also bring about *(desired or undesired)* tooth movements.

Which condition is appropriately treated at an **early** age?

- Deviated midline in the absence of a functional shift
- Mild crowding of lower permanent incisors
- Two deciduous molars nearly in crossbite
- Posterior crossbite with a functional shift

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• **Posterior crossbite with a functional shift**

Posterior crossbite:
- Should be corrected as soon as possible
- Should be thoroughly diagnosed as to whether it is of a dental, functional or skeletal origin
- May be corrected with palatal expansion
- May be associated with a mandibular shift

It is important to **correct posterior crossbites** *(which are related to the transverse plane of space)* and mild anterior crossbites in the **first stage** of treatment, even if the permanent first molars have not yet erupted. **Severe anterior crossbites,** in **contrast,** are usually **not** corrected until the **second stage** of conventional treatment.

Important: The **most common type** of active tooth movement in the primary dentition is to correct a **posterior crossbite** *(transverse problem)*.

Remember: A **skeletal** crossbite, as contrasted with a **functional** crossbite, usually demonstrates a **smooth closure** to centric occlusion.

After palatal expansion, the following are observed:
- Diastema formation between central incisors
- Expansion of the nasal floor

Note: Tooth movement and **skeletal expansion** are **inevitable** when the midpalatal suture is widened.

Displaced teeth related to functional shifts are usually seen in which two of the following circumstances?

- Posterior crossbite after prolonged thumb sucking
- Class II, Division I malocclusion
- Anterior crossbite in mildly prognathic children
- An anterior open bite after prolonged thumb sucking

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- **Posterior crossbite after prolonged thumb sucking**
- **Anterior crossbite in mildly prognathic children**

Prolonged sucking habits often **produce** a mildly narrow maxillary arch and a tendency toward bilateral crossbite. Children with this condition usually shift the mandible to one side on closure to gain better function, which can guide permanent molars, or later, premolars into a crossbite relationship.

A young child who has a tendency **toward a Class III malocclusion** will have end-to-end contact of the primary incisors. A **true anterior crossbite** in the primary dentition is **quite rare** because mandibular growth lags behind maxillary growth. The primary incisors wear down rapidly, and an anterior shift of the mandible to escape occlusal interferences rarely occurs until the permanent incisors begin to erupt. A pattern of anterior displacement of the mandible may develop when the permanent incisors come into contact, **however,** producing an anterior crossbite from the shift.

Notes:

1. An **anatomic** crossbite *(skeletal)*, as **contrasted** with a **functional** crossbite *(from thumb sucking)*, usually demonstrates a smooth closure to centric occlusion.
2. A **functional** crossbite is usually caused by thumbsucking and **does not** demonstrate smooth closure into CO.
3. A corrected anterior crossbite is **best retained** by the normal incisor relationship that is achieved through treatment **(the overbite),** not appliances.

Crossbites may be associated with all of the following **except:**

- Jaw-size discrepancy
- Heredity
- Reverse Over-jet
- Scissor-bite
- Tongue thrusting

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• **Tongue thrusting**

A **crossbite** occurs when some of the teeth wind up on the "wrong side of the track". A crossbite can be unilateral *(on one side)* or bilateral *(on both sides)*. It also can occur anteriorly or posteriorly.

Orthodontic treatment to correct a crossbite in children should begin as early as possible. The first step, **"maxillary expansion"**, broadens the maxilla with an appliance called an "expander". Fixed to the roof of the mouth, the expander is widened each night for about 1 to 2 months with the turn of a key. The expander remains in the mouth for about 3 more months to allow the bone to harden in its new position.

Note: Braces may be put on the maxillary teeth while expansion is going on to eventually close the "gap-tooth grin" that will develop as the maxilla is being expanded. Once the expansion is complete, the child may need to wear a full set of braces for 1 to 2 years to achieve an ideal occlusion.

Remember:

- **Reverse overjet** is associated with Class III skeletal patterns with more than two maxillary anterior teeth in linguoversion.
- **Scissor bite or bilateral lingual crossbite** results from a narrow mandible or a wide maxilla. Milder cases may involve only maxillary first premolars. The severe cases may need either contraction of the maxilla or expansion of the mandible.

The classic symptoms of a **sucking habit** may include all of the following **except:**

- Anterior open bite
- Crossbite
- Expanded maxillary arch
- Proclination of the maxillary incisors
- Retroclination of the mandibular incisors
- A Class II malocclusion

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• **Expanded maxillary arch**

***Constriction of the maxillary arch occurs, **not** expansion.

Anterior open bite is the **most common** sequelae of a digital sucking habit. Unilateral cross bites can also occur. **Increased pressure** exerted by the buccinator muscles during sucking, on the maxillary arch, results in its constriction. The other mechanical forces cause the maxillary incisors to procline and the mandibular incisors to retrocline. As the hand rests on the chin, it retards growth of the mandible resulting in a Class II profile.

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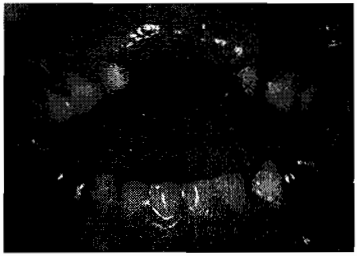

This patient demonstrates anterior openbite. The patient was a long-term vigorous thumb sucker, which resulted in the openbite. When you encounter cases of overjet and/or openbite, checking for a thumb, digit, or pacifier habit is recommended.

Notes:

1. Most of the time the anterior open bite is **asymmetrical** with normal posterior occlusion.
2. Anterior open bites are **much more common** in African Americans than Caucasians, whereas deep bites are much more common in Caucasians.

Remember: An open bite is a malocclusion, or an abnormal bite, in which some teeth, usually the front teeth, cannot be brought into contact with the opposing teeth.

An anterior crossbite in the primary dentition is often indicative of which two of the following?

- Impacted permanent maxillary canines
- A skeletal growth problem
- A developing Class II malocclusion
- A developing Class III malocclusion

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- **A skeletal growth problem**
- **A developing Class III malocclusion**

It can be the result of:
- A labially situated supernumerary tooth
- Traumatic injury
- Arch length discrepancy

Anterior crossbite of **one or more** of the permanent incisors, however, may be evidence of a localized discrepancy and a condition that almost always should be treated in the mixed dentition state or **as soon as** it's discovered. It is most often associated with prolonged retention of a primary tooth. Delayed treatment can lead to serious complications, such as **loss of arch length.** The most essential factor related to correction of anterior crossbite is the space available mesiodistally. **It is easily retained once it is corrected.**

Important: A corrected anterior crossbite is **best retained** by the normal incisor relationship that is achieved from the treatment *(the overbite),* not from appliances.

Note: The **premature exfoliation of a primary canine** may indicate an arch length deficiency. The premature loss of a primary mandibular canine may cause a lingual collapse of the mandibular anterior teeth.

Which of the following would be indicative of **maxillary retrognathism?**

- An SNA angle of approximately 82°
- An SNA angle greater than 82°
- An SNA angle less than 82°

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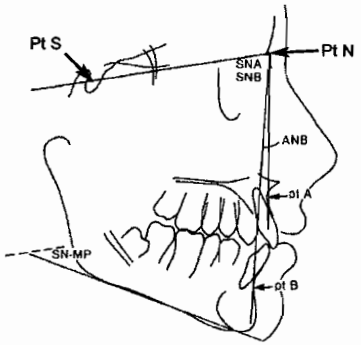
• **An SNA angle less than 82°**

The relative position of the maxilla to the cranial base is obtained by drawing two lines; one from the sella turcica ***(S)*** to nasion ***(N)*** and one from nasion to **Point A.** The angle made at the intersection of these two lines is the so-called **SNA** angle. Steiner indicates that in a good skeletal pattern this SNA angle should approximate 82°.

- SNA greater than 82° = maxillary prognathism
- SNA less than 82° = maxillary retrognathism



The **SNB** angle created by the intersection of line **SN** and **NB** defines the sagittal location of the mandibular denture base. Steiner considers an 80° angle compatible with skeletal harmony.

- SNB greater than 80° = mandibular prognathism
- SNB greater than 80° = mandibular retrognathism

A third critical angle emphasized by Steiner analysis is the **ANB** angle. Steiner's norm for this angle is 2° or the difference between the norm for SNA and SNB.

- A Class I skeletal profile ANB angle = 2°
- ANB greater than 4° = Class II skeletal profile
- ANB less than 0° = Class III skeletal profile

Note: Dental arch form is ultimately determined by the interaction of environmental influences on the genetic pattern

A **steep** mandibular plane angle correlates with:

- Short anterior facial vertical dimensions and anterior open bite malocclusion
- Long anterior facial vertical dimensions and anterior open bite malocclusion
- Short anterior facial vertical dimensions and anterior deep bite malocclusion
- Long anterior facial vertical dimensions and anterior deep bite malocclusion

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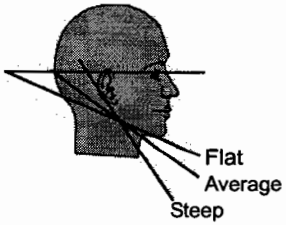
• **Long anterior facial vertical dimensions and anterior open bite malocclusion**

The **mandibular plane angle** can be visualized clinically by placing a mirror handle or other instrument along the border of the mandible.



Important: A **flat mandibular plane angle** correlates with **short** anterior facial vertical dimensions *(height)* and **anterior deep bite** malocclusion.

The angle between the mandibular plane *(Go-Me line)* and the maxillary plane *(ANS-PNS line)* is called the **maxillary-mandibular plane angle** *(MMPA)*. Its normal value is: 27° (+/- 4°). The **greater** value indicates a **longer** anterior face height.

There is also an interaction between face height and the anteroposterior position of the mandible; all other things being equal, a **long face** predisposes the patient to **Class II** malocclusion, a **short** face to **Class III** malocclusion.

What is needed so that **soft tissues** are clearly visible on a lateral cephalometric radiograph?

- Adjustment in kilovoltage
- Adjustment in millamperage
- A soft tissue shield
- A hard tissue shield
- Nothing must be done to make soft tissues visible

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• **Soft tissue shield**

The **lateral head radiograph** *(cephalometric x-ray)* must be compared with the "normal" lateral radiographs form an accepted norm. **Linear and angular measurements** are obtained utilizing known anatomical landmarks in the lateral head radiography of the patient. These measurements are then compared with those considered within normal limits and in that way enable the orthodontist to assess aberration in the dentition and jaw structures, which result in malocclusion.

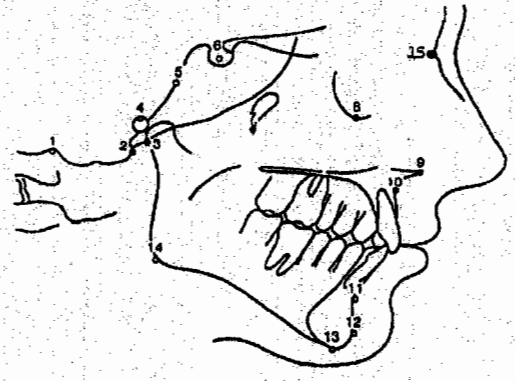
Analysis of cephalometric radiographs is **not limited** to the hard structures such as bone and teeth, but also includes measurements of soft tissue structures such as the nose, lips and soft tissue chin.

Superimposition in longitudinal cephalometric studies is generally on a reference plane and a registration point. This will best demonstrate the growth of structures furthest from the plane and the point. The most stable area from which to evaluate craniofacial growth is the **anterior cranial base** because of its early cessation of growth.

Cephalometrics is **useful in assessing** tooth-to-tooth, bone-to-bone and tooth-to-bone relationships. Serial cephalometric films can show the amount and direction of growth.

Note: A lateral cephalograph usually shows magnification with up to 7-8% magnification considered acceptable. The resulting double shadows are traced and the average is used for measurements.

Identify and define the following cephalometric landmarks labeled 1–15.



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1. **Bolton** *(Bo)* → highest point in the upward curvature of the retrocondylar fossa of the occipital bone.
2. **Baison** *(Ba)* → lowest point on the anterior margin of the foramen magnum, at the base of the clivus.
3. **Articulare** *(Ar)* → the intersection of three radiographic shadows, the inferior surface of the cranial base and the posterior surfaces of the necks of the condyles of the mandible.
4. **Porion** *(Po)* → midpoint of the upper contour of the metal ear rod of the cephalometer.
5. **Sphenooccipital synchondrosis** *(SO)* → junction between the occipital and basisphenoid bones.
6. **Sella** *(S)* → midpoint of the cavity of sella turcica.
7. **Pterygomaxillary fissure** *(Ptm)* → point at base of fissure where anterior and posterior walls meet.
8. **Orbitale** *(Or)* → lowest point on the inferior margin of the orbit.
9. **Anterior nasal spine** *(ANS)* → tip of the anterior nasal spine.
10. **Point A** *(Subspinale)* → innermost point on contour of premaxilla between anterior nasal spine and incisor tooth.
11. **Point B** *(Supramentale)* → innermost point on contour of mandible between incisor tooth and bony chin.
12. **Pogonion** *(Pog)* → most anterior point of the contour of the chin.
13. **Menton** *(Me)* → most inferior point on the mandibular symphysis, the bottom of the chin.
14. **Gonion** *(Go)* → lowest most posterior point on the mandible with the teeth in occlusion.
15. **Nasion** *(Na)* → anterior point of the intersection between the nasal and frontal bones.

The **Frankfort-Horizontal plane** is constructed by drawing a line connecting **porion and orbitale.** This has been adopted as the best representation of the natural orientation of the skull.

A **seven-year-old child** with an otherwise good occlusion has a lingually locked maxillary permanent central incisor. There is sufficient room for the tooth. In order to treat this condition properly, the dentist should do what?

- Wait until all permanent teeth have erupted
- Surgically reposition the central incisor
- Correct the condition immediately with a simple appliance
- None of the above

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• **Correct the condition immediately with a simple appliance**

Ideally, this anterior crossbite **should have been corrected** before it reached the occlusal plane *(while it was erupting).* The most probable etiologic factor for this happening is prolonged retention of the primary maxillary incisors.

Cross-elastics from the maxillary lingual to the mandibular labial can be used to correct a single-tooth crossbite. A maxillary removable appliance can also be used. When elastics are used to move teeth they should be attached directly to the appliance components.

Anterior crossbite, particularly **crossbite of the incisors,** is rarely found in children who do not have a skeletal Class III jaw relationship. A crossbite relationship of one or two anterior teeth, **however,** may develop in a child who has good facial proportions. The maxillary lateral incisors tend to erupt to the lingual and may become trapped in that location, especially in the presence of severe crowding. In this situation, extracting the adjacent primary canines usually leads to spontaneous correction of the crossbite. It is important to evaluate the space situation before attempting to correct any anterior crossbite. If enough space is available to accomplish the movement, a maxillary removable appliance is usually the best mechanism to correct a simple anterior crossbite that requires a tipping movement. **Remember: Anterior crossbite in a primary dentition usually indicates a skeletal growth problem.**

Which of the following is usually used as an **etching agent** before direct bonding of orthodontic brackets?

- 10–15% unbuffered phosphoric acid
- 35–50% unbuffered phosphoric acid
- 75–85% unbuffered phosphoric acid
- 100% unbuffered phosphoric acid

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• **35–50% unbuffered phosphoric acid** → for 1 minute

Remember: After etching, the tooth surface should have a frosted appearance.

The tooth surface **must not be** contaminated with saliva, which promotes immediate remineralization, until bonding is completed; otherwise re-etching is required. Topical fluoride **should not be used** before etching because fluoride decreases the solubility of enamel.

Indications for **using bands** instead of bonded brackets:
- To provide **better** anchorage for greater tooth movement
- For teeth that will need **both** lingual and labial attachment
- Teeth with **short clinical crowns**
- Tooth surfaces that are **incompatible** with successful bonding

Cementation of bands: Glass ionomer cements *(resin or non-resin based)* because of their fluoride releasing properties and retentive strengths are fast replacing Zinc phosphate cement. The cold slab is used for mixing regardless of which of the two cements is used *("frozen slab technique").* This allows the addition of a greater amount of powder into the cement liquid and thus produces a stronger cement.

An active finger spring of a removable appliance usually touches the tooth with a point contact. What is the most likely type of tooth movement produced in this situation?

- Tipping
- Extrusion
- Intrusion
- Translation

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• **Tipping**

The best method for tipping maxillary and mandibular anterior teeth is with **finger springs.**These finger springs are **attached** to a removable appliance. The most common problems associated with these simple removable appliances are lack of patient cooperation, poor design leading to lack of retention, and improper activation. An undesirable common side effect of a finger spring is the tendency for the root apex to move in the direction opposite from the crown.

Z-springs can also be used **but** they deliver excessively heavy forces and lack range of motion.

Maxillary incisor rotation is **not commonly treated** during the stage of mixed dentition. It is best treated after all permanent teeth have erupted *(early permanent dentition)*. This is usually accomplished with a simple removable appliance. However, if the incisor is in crossbite, it should be corrected as soon as possible *(while it is erupting)*.

Notes:

1. When using **buccal coil springs** to try and regain space by pushing a tooth mesially or distally, be careful because what commonly occurs is rotation of that tooth instead of actual movement.
2. The force generated in the spring is directly proportional to the distance that an orthodontic spring is deflected and the radius *(r)* of the wire. It is inversely proportional to the length of the spring.

Precisely: $F \alpha \frac{d}{r^4}$

Which of the following are **potentially** detrimental conditions that can occur during use of orthodontic appliances?

- Cause irritation to the gingiva
- Act as plaque harbors
- Make proper oral hygiene difficult to perform
- All of the above

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• **All of the above**

Orthodontic appliances **may be irritating** or interfere with the performance of good oral hygiene. Prolonged orthodontic treatment has long been associated with the causation of inflammatory periodontal diseases. **However,** if meticulous oral hygiene is maintained at all times during orthodontic treatment, the periodontal health can be maintained.

Note: When a patient *(young or old)* is in **active** orthodontic treatment and the gingiva is inflamed, the dentist should encourage better oral hygiene. It may be useful to recommend the use of water irrigation devices to help flush food debris away from the brackets.

Which of the following types of headgear produces a **distal and upward** force on the maxillary teeth and maxilla?

- Straight-pull headgear
- Reverse-pull headgear
- Cervical-pull headgear
- High-pull headgear

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• **High-pull headgear**

High-pull headgear consists of a **head cap** connected to a face-bow. This appliance places a distal and upward force on the maxillary teeth and maxilla. These types of headgear have a more direct effect on the anterior segment of the arch. **Indications:** Class II, Division 1 malocclusions that have an open bite.

Cervical-pull headgear is made up of a **neck strap** connected to a **face-bow.** This appliance produces a distal and downward force against the maxillary teeth and the maxilla. A major disadvantage of treatment using cervical headgear is possible extrusion of the maxillary molars. Likely results include: opening the bite, first molars will move distally and forward growth of the maxilla will decrease. **Indications:** Class II, Division 1 malocclusions.

Straight-pull headgear is similar to the cervical-pull headgear. However, this appliance places a force in a straight distal direction from the maxillary molar. Like cervical-pull headgear, the **indications** are Class II, Division 1 malocclusions *(when bite opening is undesirable).*

Reverse-pull headgear unlike all of the other headgears above, has an **extraoral component** that is supported by the chin, cheeks, forehead or a combination of these structures. **Indications:** Class III malocclusions *(where protraction of the maxilla is desirable).*

The generalized **causes** of the failure of tooth eruption or delayed tooth eruption include all of the following **except:**

- Hereditary gingival fibromatosis
- Down's syndrome
- Rickets
- Hyperparathyroidism

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- **Hyperparathyroidism**

***This condition can result in the **premature exfoliation** of primary teeth.

The generalized eruption failure or "primary failure of eruption" is caused by the failure of the eruption mechanism itself. Bone resorption proceeds normally, but involved teeth simply do not follow the path that has been cleared. The involved teeth do not erupt spontaneously and are not amenable to any orthodontic recourse. This condition is rare.

The **localized** causes of failure of eruption or the delayed eruption of the teeth include:
- Congenital absence
- Abnormal position of the crypt
- Lack of space in the arch *(crowding)*
- Supernumerary teeth
- Dilacerated roots

A **"Poor man's Cephalometric Analysis"** is performed via a:

- Dental cast analysis
- Facial profile analysis
- Photographic analysis
- Full face analysis

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• **Facial profile analysis**

The facial profile analysis delineates the same information as that obtained through lateral cephalometric radiographs. The difference lies in the detail obtained through the latter method, however, the former is considered a vital diagnostic technique for primary evaluation. It is a quick and simple *(also cheap)* technique which readily gives the following information:

1. Anteroposterior position / proportion of the jaws relative to each other
2. Lip posture *(competent / incompetent)* and incisor prominence
3. Vertical facial proportions
4. Inclination of the mandibular plane angle

Note: Within the lower third of anterior face height the mouth should be about one-third of the way between the nose and the chin.

All of the following are advantages of the **indirect method** of bonding brackets to a tooth over the direct method **except:**

- Reduced chair-side time
- More precise location of brackets possible in the laboratory
- Controlled thickness of the resin between the tooth and the bracket interface
- No need for custom impression trays and silicone or rubber impressions
- Less technique sensitive
- Easier clean-up during bonding and de-bonding

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• **Less technique sensitive**

***The indirect bonding technique is **more complex** and technique sensitive and requires extra precautions.

The procedure involves the following steps:

1. An accurate impression with alginate is taken and poured up with orthodontic model stone to be used as a working model.
2. Vertical lines are drawn on the teeth to aid in bracket placement and a separating media is applied.
3. The brackets are then loaded with a filled resin paste and cured.
4. After its initial set, individual positioning of a tray with silicone is prepared by applying it over the bracketed teeth on plaster model.
5. This whole set-up then is placed in warm water to dissolve the separating media.
6. The silicone tray is then removed from the plaster model with brackets embedded in it.
7. Brackets are cleaned under running water making sure that pads have cured resin.
8. Enamel is etched, conditioned and unfilled resin is applied. Unfilled resin is also applied to cured resin on the base of the bracket pads.
9. The silicone tray with embedded brackets is then positioned on the teeth being bonded and held in position till the initial set of the unfilled resin is reached.

Notes:

1. The control of **"flash"** *(excess of resin)* not only makes clean up easy but also the controlled thickness of it accurately expresses the built-in prescription of the appliance.
2. Also, in situations where visibility is a problem *(e.g., lingual appliance)*, this technique is almost always employed.

All of the following statements regarding the effect of environmental influences during growth and development of the face, jaws and teeth are true **except:**

- Patients who have excessive overbite or anterior open bite usually have posterior teeth that are infra- or supra-erupted respectively
- A non-nutritive sucking habit leads to malocclusion only if it continues during the mixed dentition stage
- Negative pressure created within the mouth during sucking is not considered a cause of constriction of the maxillary arch
- "Tongue thrust swallowing" leads to an open-bite
- "Adenoids" which lead to mouth breathing, cannot be indicted with certainty as an etiologic agent of a long-face pattern of malocclusion because studies show that the majority of the long-face population have no nasal obstruction

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• **"Tongue thrust swallowing" leads to an open bite** → this is **not** true

Recent studies indicate that "tongue thrust swallowing" can not be blamed for an open bite as it was shown that there is no tongue-force on the teeth during swallowing even though the tip of the tongue is placed forward.

The tendency to place the tongue forward between the teeth *(in cases of anterior open bite)* appears to originate from the need to attain an oral seal during swallowing. So, the forward position of the tongue during swallowing is due to the anterior open bite, the reverse is not true → **"A tongue thrust swallow therefore should be considered the result of displaced incisors, not the cause".**

All of the following are **functional** appliances **except:**

- Frankel
- Bionator
- Clark's Twin Block
- Herbst
- Activator
- Quad-Helix

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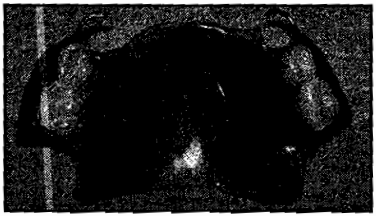
- **Nance appliance**

Note the small acrylic button that will rest against the palatal tissue with this appliance. Some clinicians object to the button since it can create tissue irritation. Therefore, it is important that patients and parents be instructed to make sure that the patient meticulously flosses under the acrylic button. The Nance appliance is used in situations where **premature bilateral loss of maxillary primary teeth** has occurred.



This photograph shows a maxillary removable appliance, in this case a maxillary removable bilateral space maintainer. As suggested previously, removable appliances are not commonly used because of problems with the appliance not being worn and the frequent incidence of breakage and loss.

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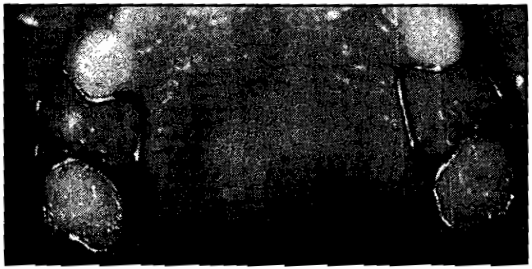

This photograph shows two band and loop space maintainers, an example of the bilateral use of fixed unilateral band and loop space maintainers. These are very common types of unilateral space maintainers, and they often are used bilaterally.

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PROSTHODONTICS

Legend

Major Topic	Abbreviation
Acrylic Resins	Acrylic Resins
Complete Dentures	Complete Dent
Crown & Bridge	Crn & Bridge
Gypsum Products	GP
Impression Materials	Impr Mat
Miscellaneous	Misc.
Occlusion	Occlusion
Porcelain	Porc
Removable Partial Dentures	RPD

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Several polymer systems are used in dentistry. Which of the following systems is used **most frequently?**

- Bis-GMA
- Polyether
- Methyl methacrylate
- Polyvinyl

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• **Methyl methacrylate**

***Methyl methacrylate is the liquid **monomer** and is abbreviated **MMA,** polymethyl methacrylate is the powder **polymer** and is abbreviated **PMMA.**

Heat-cured materials → heat is used as an accelerator to decompose **benzoyl peroxide** *(the initiator)* into **free radicals.** These free radicals initiate the polymerization of MMA into PMMA. The polymerization process continues as new PMMA is formed as a matrix around residual PMMA powder particles.

Self-cured *(auto-cured, cold cured)* **materials** → a chemical activator such as **dimethyl-p-toluidine** *(which is a tertiary amine)* is added to the monomer *(MMA)*. This chemical activator causes decomposition of the **benzoyl peroxide** *(the initiator)* into **free radicals.** These free radicals initiate the polymerization of MMA and PMMA. The polymerization process continues the same as in heat-curing above.

Notes:

1. The **polymerization range** is the temperature range, approximately 60°C *(140°F)* to 77°C *(170°F)*, at which the major part of polymerization occurs in a heat-cured resin.
2. The **heat-cured resins** have less residual monomer and a higher molecular weight than the self-cured resins; therefore, **they are stronger.** They also have **superior color stability.**

Other monomers *(liquids)* used are ethyl methacrylate, vinyl ethyl methacrylate, and epimine resins. **These all are less irritating to the pulp.**

The **powder** used in mixing acrylic resin is referred to as the:

- Dimer
- Initiator
- Polymer
- Monomer

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- **Polymer**

Components of Acrylic Resins

- **Powder: Polymethyl methacrylate** *(PMMA)* polymer, benzoyl **peroxide** initiator, and pigments.
- **Liquid:** Pure **methyl methacrylate** *(MMA)* monomer, **Hydroquinone** inhibitor, cross-linking agents, and **chemical activator** *(dimethyl-p-toluidine).* **Note:** This activator is only present in self-cured resins to bring about polymerization.

Remember → mechanical properties of resins are influenced by the following:
- Molecular weight of the polymer
- Degree of **cross-linking** *(need difunctional monomers which contain two areas for reaction)*
- Composition of monomers used to prepare the polymer

***Both** PMMA and the newer bisacryl composite resins *(e.g., Iso-temp)* may be used to fabricate a successful provisional restoration. PMMA resins are strong, easy to repair and trim, and have adequate working time, however, the materials' liquid monomer component results in an increased allergic reaction rate. Their long-term color stability is poor and their exothermic setting reactions may affect the pulp deleteriously. The newer bisacryl composite resins perform as well as the PMMA resins. They are easier to handle and provide color stability for longer periods of time. The quality of contouring, marginal finishing, and polishing is an operator-dependent variable and does not appear to be related statistically to the type of resin material.

Notes:

1. **Acrylic resins** will expand when immersed in water and become distorted when dried out.
2. **Excessive shrinkage** may occur if too much monomer *(liquid)* is added to the polymer *(powder).*
3. The polymerization reaction of methyl methacrylate is **exothermic** → gives out heat.
4. **Inhibitors** are added to the monomers to aid in preventing polymerization during storage.
5. **Cross-linking** contributes greatly to the strength of the polymer.

The shape and amount of the **distobuccal extension** of a complete **mandibular edentulous** impression is determined during border molding by the:

- Ramus of the mandible
- Position and action of the masseter muscle
- Lateral pterygoid muscle
- Tone of the buccinator muscle
- Size and location of the buccal frena

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• **Position and action of the masseter muscle**

When border molding a **mandibular custom tray** that will be used for a final denture impression:

- The **distobuccal extension** is determined by the position and action of the **masseter** muscle.
- The **distolingual extension** is limited by the action of the **superior constrictor** muscle.

Important: The most important consideration in checking custom trays for accurate border molding is **stability and lack of displacement.**

Note: The **custom tray** for a final mandibular or maxillary complete denture impression should have a **spacer with stops** to insure that the tray will be seated in proper relationship to the arch and that there will be adequate room for the impression material. The space is created with wax covered by aluminum foil over the master cast prior to forming the tray.

The **primary difference** between border molding with a ZOE impression material and border molding with modeling plastic is that the ZOE impression material **must be border molded during one insertion** and within the setting time of the material → as opposed to two insertions with modeling compound.

Remember: The palatoglossus, superior pharyngeal constrictor, mylohyoid, and genioglossus muscles are influential in molding the **lingual border of the mandibular impression** for an edentulous patient.

The **primary reasons** for obtaining the most extensive areal coverage for a mandibular complete denture are:

- To increase the capacity of underlying structures to withstand the stress due to biting force and to improve appearance
- To provide balanced occlusion and to increase tongue space
- To increase the capacity of the underlying structures to withstand the stress due to biting force and to increase the effectiveness of the seal
- To improve retention and to increase tongue space

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- **To increase the capacity of the underlying structures to withstand the stress due to biting force and to increase the effectiveness of the seal**

*** **Key point** → underextension of the peripheral border of a complete mandibular denture decreases tissue-bearing surfaces, thereby affecting denture stability. **Marked ridge resorption** will occur if a mandibular complete denture base terminates short of the retromolar pad.

The **underlying basal bone** *(beneath the retromolar pad)* is resistant to resorption. Coverage of this area will also provide some border seal. An **overload of the mucosa** will occur if the bases covering the area are too small in outline.

Remember: Mandibular dentures **do not rely on suction** from a peripheral seal for retention *(as do maxillary dentures)* **but rather** on denture stability in covering as much basal bone as possible without impinging on the muscle attachments. The **active border molding** performed by the lips, cheeks, and tongue **determines the peripheral areas** of a mandibular arch, thus establishing maximal base bone coverage.

Note: Thin mucosa is found in the following areas:
- In the mylohyoid area → mandibular denture
- On the midline of the palatal vault → maxillary denture
- Over a torus palatinus → maxillary denture
- Over a mandibular tori → mandibular denture

Immediate dentures should be scheduled for **relines** at:

- 1 month and 3 months post extraction
- 4 months and 7 months post extraction
- 5 months and 10 months post extraction
- 1 year and 2 years post extraction

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• **5 months and 10 months post extraction**

Recontouring of the healing ridge **progresses rapidly for four to six months** and does not become stable in form until 10 –12 months post extraction. Due to this, immediate dentures become progressively more ill-fitting. They should be relined five months and ten months after delivery in order to compensate for contour changes. **Note:** This is a **general timeline;** each case needs to be evaluated monthly and, if necessary, relines performed.

A reline **is indicated** on any denture when the diagnostic information indicates that a reline will effectively solve the patient's chief complaint → when the denture base adaptation is the major defect in the prosthesis. A reline **is contraindicated** when there is excessive overclosure of the vertical dimension → a large decrease in vertical dimension. **In this case,** new dentures are indicated at the proper vertical dimension.

Note: When a patient wears a complete maxillary denture against the six mandibular anterior teeth, it is **very common** to have to do a reline every so often due to the loss of bone structure in the anterior maxillary arch → evidenced by a flabby maxillary anterior ridge.

All **new dentures** should be evaluated:

- 3 hours after delivery
- 12 hours after delivery
- 24 hours after delivery
- 48 hours after delivery

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• **24 hours after delivery**

This is done for the purpose of correcting undetected errors. Tissue trauma attributed to denture function manifests as hyperemia, inflammation, ulceration, and pain.

The **basic sequence** of the clinical procedure for a 24 hour recall appointment is:

1. Remove the dentures from the mouth.
2. Thoroughly examine the mouth.
3. Ask the patient about the areas of tissue trauma which have been observed.
4. Permit the patient to describe additional complaints.

***After collecting all of the diagnostic information, the dentist can determine the source of the problem and the cure.

Remember: During the first few days following the insertion of complete dentures, the patient should expect some difficulty in masticating most foods and excessive saliva → which is due to reflex parasympathetic stimulation of the salivary glands. Over time this will subside and become normal.

Important: Occlusal disharmony can be **most accurately** corrected on the articulator after patient remounting procedures.

Posterior teeth that are set **edge to edge** may cause:

- Gagging
- Cheek biting
- Reduced taste
- Speech aberrations

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- **Cheek biting**

Common Causes of Cheek Biting with New Dentures	
Cause	**Treatment**
Posterior teeth edge to edge	Reduce the facial surfaces of mandibular molars to create proper horizontal overlap
Inadequate vertical dimension of occlusion	Reline at corrected VDO, patient remount, fabricate new denture
Biting corners of the mouth	Reset cuspids/bicuspids

Notes:

1. **Lip biting** may be due to reduced muscle tone and / or a large anterior horizontal overlap.
2. **Tongue biting** may be caused by having posterior teeth too far lingually.

Maxillary anterior teeth in a complete denture are usually arranged:

- Facial to the ridge
- Lingual to the ridge
- Exactly over the ridge

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• **Facial to the ridge** → for best esthetics

Setting anterior teeth **directly over the ridge** usually causes poor esthetics of dentures. Also, it is important to have accurate adaption of the border seal and adequate bulk of the maxillary facial flange for good esthetics. Vertical dimension of occlusion affects the lip support as well.

For **most patients,** the labial surface of the central incisor should be approximately 8 mm anterior to the center of the **incisive papilla.** The labioincisal one-third of the maxillary central incisors should support the lower lip when the teeth are in occlusion.

Important: The long axes of the maxillary central incisors should be perpendicular to the occlusal plane; the long axes of the maxillary lateral incisors should have an asymmetric mesiodistal inclination.

Remember: Maxillary central incisors are the **most important teeth** when esthetics is under consideration. Their placement controls the midline, speaking line, lip support and smiling line composition. **Note:** Placement of maxillary anterior teeth in complete dentures too far superiorly and anteriorly might result in difficulty in pronouncing "f" and "v" sounds.

Some of the common errors in the arrangement of teeth include:
- Setting mandibular anterior teeth too far forward to meet the maxillary teeth
- Failure to make canines the turning point of the arch
- Setting the mandibular first premolars buccal to the canines
- Establishing the occlusal plane by an arbitrary line on the face
- Not rotating anterior teeth enough to give an adequately narrower effect

A patient who wears a complete maxillary denture **complains of a burning sensation in the palatal area of his / her mouth.** This is indicative of too much pressure being exerted by the denture on the:

- Incisive foramen
- Palatal mucosa
- Hamular notch
- Posterior palatal seal

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• **Incisive foramen**

Notes:

1. A **burning sensation** in the mandibular anterior area is caused by pressure on the mental foramen.
2. A patient having **trouble swallowing** may have insufficient interocclusal space → decreased freeway space caused by excessive vertical dimension of occlusion.
3. The best dietary advice for an **elderly denture patient** is to eat foods rich in protein and vitamins A, C, D, and B complex.

Important: Learning to chew satisfactorily with new dentures requires at least 6-8 weeks. This time is spent on establishing new memory patterns for both facial and masticatory muscles.

Residual ridges can be ruined by the use of denture adhesives and home-reliners. Therefore, patients should be specifically warned about their uses. These agents can modify the position of the denture on the ridge and result in change of both vertical and centric relations.

The treatment plan for a patient indicates that **both** mandibular and maxillary immediate dentures are to be fabricated. The **ideal** way to do this is:

- Fabricate the maxillary immediate denture first
- Fabricate the mandibular immediate denture first
- Fabricate the maxillary and mandibular immediate dentures at the same time

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• **Fabricate the maxillary and mandibular immediate dentures at the same time**

The main reason for this is to **avoid setting the maxillary teeth** to the likely malpositions of the remaining mandibular teeth.

Important: If the **master casts are altered** in an immediate denture procedure *(e.g., elimination of gross undercuts)*, it is advisable to construct a second denture base that is transparent *(called a surgical stent or template)*. This surgical stent is placed over the ridge after the teeth are extracted. Pressure points and undercuts are readily visible and surgical ridge correction can be performed.

Remember: The duplication of the master cast used for the construction of the surgical template to be used at the time of immediate denture insertion is **best made** after wax elimination and after the cast is trimmed.

Note: A **major advantage** with immediate dentures is being able to **duplicate the position of the natural teeth.**

The **first step** in the treatment of abused tissues in a patient with existing dentures is to:

- Fabricate a new set of dentures
- Reline the dentures
- Educate the patient
- Excise the abused tissues

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• **Educate the patient**

Important: The patient should understand both the cause of the tissue deterioration and the eventual outcome if the process is not arrested.

If the constant wear of **unacceptable dentures** is the cause of the tissue abuse, the most efficient preliminary treatment is removal of the dentures. However, business and social commitments may not permit removal for extended periods. In such patients, **resilient tissue conditioning materials** may be used to assist in the tissue recovery program.

Other procedures recommended as aids in the treatment of abused tissues include massage and warm saline rinses.

The most important **benefit** of an overdenture *(root-retained denture)* is:

- The psychological comfort of avoiding the loss of all teeth
- The continuous functional feedback for the neuromuscular system from proprioceptors in the periodontal membrane
- The preservation of the alveolar ridge
- The improved support and stability for the denture
- The increased retention of the denture

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• **The preservation of the alveolar ridge**

A **complete overdenture** is a denture whose base is constructed to cover all of the existing residual ridge and selected roots. Retained roots help to prevent resorption of the alveolar ridges. These roots also improve retention and afford the patient some proprioceptive sense of "naturalness" in function of the dentures.

It is **not always necessary** to cover a root beneath an overdenture, however, if a root is not covered, the **exposed surfaces are highly susceptible to decay.** The oral hygiene of the patient must be impeccable to prevent the decay of these roots.

Note: Retained roots are the most common findings when taking routine panoramic radiographs of patients who wear complete dentures *(not necessarily overdentures).*

The incisal edges of the maxillary anterior teeth should just touch the wet/dry line of the lower lip when enunciating which sound(s)?

- "Th" sound
- "F" and "V" sounds
- "P" and "B" sounds
- "S" sound

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• **"F" and "V" sounds**

"S" sound → the mandibular incisal edges should be even with or just behind the incisal edges of the maxillary teeth. Most people form the "s" sound with the tip of the tongue approaching the anterior palate and lingual surfaces of the maxillary teeth.
Note: These sounds bring the mandible and maxilla close together.

"Th" sound → when forming the "th" sound, the tongue should **protrude slightly** between the maxillary and mandibular anterior teeth. It should normally protrude 2-4 mm.

"F" and "V" sounds → are formed by the incisal edges of the maxillary teeth and the lower lip. The incisal edges of the maxillary teeth **should just touch** the wet / dry line of the lower lip.

"P" and "B" sounds → are formed totally by the lips.

Words with the sibilant sound *(hissing sounds)* are pronounced correctly with the incisal edges of maxillary and mandibular almost touching. These sounds are usually produced between rest and the occluding position.

Note: The two most probable causes of a patient complaining that whenever he / she tries to make an "s" sound, it sounds like "th" are:
• Incisor teeth are set too far palatally
• Palate is made too thick

*** If the teeth are positioned too far lingually, the **"t"** will tend to sound like a "d." If the teeth are positioned too far labially, the **"d"** will sound more like a **"t."**

*** An increased occlusal vertical dimension can result in clicking of teeth.
*** A high palatal vault or a constricted palate can cause whistling sounds.

The primary role of **anterior teeth** on a denture is:

- To incise food
- Occlusion
- Esthetics
- Stability of the denture

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• **Esthetics**

Spaces, lapping, rotation, and color changes can be judiciously used to create a natural appearance. **Note:** Proper lip support is provided by the facial surfaces of teeth and simulated attached gingiva.

Setting the anterior teeth either too far lingually or facially to satisfy esthetic concerns **should not be done.** When selecting teeth, pre-extraction records are very valuable. Maxillary and mandibular anterior teeth **should not** contact in centric relation.

The outline of anterior teeth should **harmonize** with the form of the face:

- Convex profile faces should have a similarly convex labial surface of anterior teeth.
- Broader contact areas of teeth look more natural on dentures as they seem more compatible with advanced age.

Whistling when a patient **speaks with dentures** *(complete or partial which replaces the incisors)* may be caused by any of the following:

- **Vertical** overlap is **not enough.**
- Horizontal overlap is **too much.**
- The area palatal to the incisors is **improperly contoured.**

Note: In general, **functional needs** overshadow those of esthetics when selecting **posterior teeth. Do not set** mandibular molars over the ascending area of the mandible because the occlusal forces in the area will dislodge the mandibular denture.

A patient has worn a complete maxillary denture for 8 years against mandibular anterior teeth *(the remainder of the mandibular teeth are missing)*. She complains of looseness of the denture. Examination of the mouth shows an excessive amount of hyperplastic tissue at the anterior part of the maxillary ridge. The maxillary denture teeth do not show below the upper lip. Radiographs show poor bone structure in the anterior part of the maxillae. The principal cause of difficulty with her maxillary denture is:

- Fibrous tuberosities
- Too great a vertical dimension of occlusion
- A lack of posterior occlusion
- The maxillary denture teeth that were used are too short

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- **A lack of posterior occlusion**

The patient's chief complaint will be **looseness** of the maxillary denture. They will also state that they can no longer see their upper teeth on the denture. These signs and symptoms are **caused** by a lack of posterior occlusion.

Important: A patient wearing a **maxillary complete denture** and a **mandibular bilateral distal-extension** removable partial may show:

- Decreased vertical dimension of occlusion
- A prognathic facial appearance

Note: When a complete maxillary denture **opposes** natural mandibular anterior teeth, the **maxillary anterior ridge** often becomes very flabby.

Remember: The best impression technique for an edentulous patient with loose, hyperplastic tissue in the maxillary anterior region is to register the tissue in its **passive position.**

The **primary indicator** of the accuracy of border molding is:

- Adequate coverage of tray borders with the material used for border molding
- Contours of the periphery similar to the final form of the denture
- Stability and lack of displacement of the tray in the mouth
- Uniformly thick borders of the periphery

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• **Stability and lack of displacement of the tray in the mouth**

The **ease** and **accuracy** of the border molding depends upon:

1. An accurately fitting custom tray
2. Control of bulk and temperature of the modeling compound
3. A thoroughly dried tray

The custom tray fabricated on the preliminary cast is trimmed approximately 2 mm short of the mucosal reflection and frenae. This is done by first checking the borders in the mouth and then trimmed down. This will allow a uniform thickness of 2 mm of modeling compound when borders are molded. Proper border molding results in contours resembling the final form of the denture. However, the primary indicator of the **accuracy of border molding** is the stability and lack of displacement of tray in the mouth.

Border molding is completed in two stages. In the **first stage** the molding should approximate the borders but should be slightly overextended. Excess compound is trimmed from inside and outside of the tray. The remaining modeling compound is then refined by repeating the process. The final form of the border molding should represent an accurate impression of the peripheral tissues. The modeling compound should have a smooth, almost polished appearance.

After border molding is completed, some areas of the modeling compound should be relieved because the tissues are extremely displaceable and have probably been distorted during the border molding process. These areas include around the maxillary labial frenum and over the retromolar pad areas.

Remember: Modeling compound *(plastic)* has a relatively **low thermal conductivity.**

You are in the process of making a complete **maxillary** denture for a patient. Which of the following structure(s) will be the **secondary** support area(s)?

- Residual ridges
- Palatal rugae
- Incisive papilla
- Maxillary tuberosity
- Buccal vestibule

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- **Palatal rugae**

***The **primary** support areas of the maxillary complete denture are the **residual ridges.**

In the **mandibular arch,** the primary support area is the buccal shelf because of its bone structure and its right angle relationship to the occlusal plane. The residual ridges if large and broad can also be considered as the primary support areas.

Remember:

- The **secondary peripheral** seal area for a mandibular complete denture is the anterior lingual border.
- If you are fabricating a mandibular complete denture for a patient with a **knife-edge ridge,** you need maximal extension of the denture to help **distribute the forces of occlusion** over a larger area.

Important: The most important factor for providing retention for complete dentures is the **peripheral seal.**

An **overextended distobuccal corner** of a mandibular denture will push against which muscle during function?

- Zygomaticus
- Orbicularis oris
- Temporalis
- Masseter

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• **Masseter**

This is a **very common area** of overextension and should be checked very well when delivering the mandibular denture.

The **buccinator muscle** lies under the denture flange in this area but the fibers run anteroposterior in a horizontal plane and their action is weak; the **anterior fibers** of the masseter muscle pass outside the buccinator at the distobuccal corner of the mandibular denture and **will push against** the buccinator during function causing dislodgement.

Important: When the **posterior maxillary buccal space** is entirely filled with the denture flange, the **coronoid process may interfere** with the denture upon opening of the mouth. This will cause dislodgement of the maxillary denture.

Notes:

1. The superficial layer of the **masseter muscle** originates from the zygomatic process of the maxilla and inserts at the angle and lower lateral side of the ramus of the mandible.
2. The **pterygomandibular raphe** lies between the buccinator and superior constrictor muscles.

After **border molding** the mandibular custom tray, it is important to check for dislodgement in order to detect areas of:

- **Underextension** of the tray
- **Overextension** of the tray
- **Thickness** of the tray
- None of the above

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- **Overextension of the tray**

Check for dislodgement **using** the following techniques:

- Pull gently upward on the patient's cheek.
- Pull the lower lip gently forward in a horizontal direction.
- Have the patient open widely.
- Have the patient move the tongue into the right and left buccal vestibules.
- Have the patient protrude the tongue to touch the lower lip. Have the patient move the tip of the tongue from one corner of the mouth to the other.

Dislodgement indicates **overextension** and the border molding process should be refined in the offending area. Common areas of overextension of the **mandibular** impression are the **labial** and the **buccal.** This is suspected when the impression raises as the mouth is opened.

The **most critical area** in the border-molding procedure for a **maxillary** denture is the **mucogingival fold** above the maxillary tuberosity area. This area is extremely important for **maximal retention.** Other critical areas are the labial frena in the midline and the frena in the bicuspid area. Overextension in these areas **often leads to** decreased retention and tissue irritation.

Before an accurate face-bow transfer record can be made on a patient, which of the following must be determined?

- The inclination of each condyle
- Vertical dimension of occlusion
- Centric relation
- Location of the hinge axis point

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• **Location of the hinge axis point** → axial center of opening-closing

A **face-bow** is a caliper-like device used to record the patient's maxilla / hinge axis relationship *(opening and closing axis)*. It is also used to transfer this relationship to the articulator during the mounting of the maxillary cast. If the face-bow transfer procedure **is properly done,** the arc of closure on the articulator should duplicate that exhibited by the patient. This hinge-axis face-bow transfer enables alteration in vertical dimension on the articulator. When **altering vertical dimension** *(either through restorations or with dentures)*, casts should be **mounted on the hinge axis.**

When the maxilla / hinge axis relation is transferred to the fully adjustable articulator, it may be necessary to obtain the precise tracing of the paths followed by the condyles. A **pantograph** is an instrument which carries out this task with the help of two face-bows. One is attached to the maxilla and the other to the mandible using a clutch that attaches the teeth in their respective arches.

A plaster index is used to:

- Maintain the vertical dimension of occlusion
- Maintain bite registration
- Preserve the face-bow transfer
- All of the above

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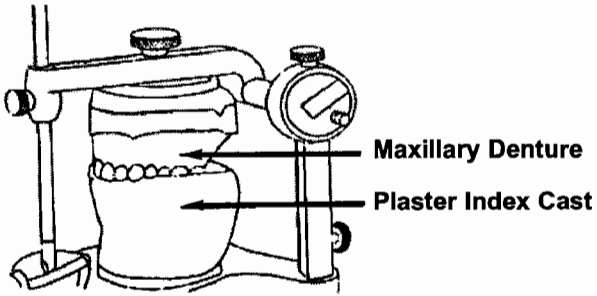
• **Preserve the face-bow transfer**

When fabricating dentures, there are **two methods** used to preserve the face-bow transfer:

1. **Taking a plaster index** of the occlusal surfaces of a maxillary denture before removing the denture from the articulator and cast *(see picture below)*.
2. **Placing a piece of 10x wax** on the occlusal surfaces of the mandibular teeth and closing the articulator in centric relation. Chill the wax, drop the incisal guide pin to touch the incisal guide table (*do not change)*.

The plaster index method is the **preferred method** due to possible distortion of wax.



A **generalized speech difficulty** with complete dentures is usually caused by which **two** of the following?

- Faulty tooth position
- Excess vertical dimension of occlusion
- Faulty palatal contours
- Faulty occlusion

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- **Faulty tooth position**
- **Faulty palatal contours**

Speech problems due to faulty tooth position can be avoided by placing the denture teeth **as close as possible** to the position of the natural teeth. **Note:** The most effective time to test for phonetics is at the time of the wax try-in of the trial denture *(this is usually the fourth appointment)*. Faulty palatal contours can be corrected by trial and error. Add wax to increase contours and reduce as needed to improve articulation of sounds. **Note:** Patients who have been **edentulous for many years** often have more distorted speech than those who have been edentulous for a short time. This is usually due to a **loss of tonus** of the tongue musculature.

Problems Related to Phonetics

Complaints	Causes	Treatments
Whistle on "S" sounds	Air stream passes unimpeded or with inadequate impedance between the dorsal surface of the tongue and the anterior palate	Increase the palatal resin convex contours lingual to the maxillary central incisors to impede the air stream passing between the tongue and palate. Create rugae if necessary
Lisp on "S" sounds	The air stream passing between the tongue and anterior palate is excessively impeded, usually by rugae or excessive resin contour	Reduce occlusal vertical dimension until premolars no longer contact during speech
Maxillary & Mandibular incisors or premolars contact during sibilant *(s, sh, z, ch)*	Occlusal vertical dimension too great	Reduce occlusal vertical dimension until premolars no longer contact during speech
Clinician observes that incisal edges of maxillary incisors contact the lower lip 1 mm or more labial to the wet/dry junction of lower lip when "F" & "V" sounds are made	Maxillary teeth may be set too far labially	Evaluate lip support and overall appearance of anterior teeth as they are positioned. Reset to a more lingual position as needed. Incisal edge of maxillary incisiors should contact the wet/dry junction just lingual to it during production of the "F" & "V" sounds

At the first appointment after insertion of complete dentures, the presence of generalized soreness on the crest of the mandibular ridge is most likely due to:

- The newness of the denture
- Defective tissue registration
- Premature occlusal contacts
- Incomplete polymerization of the denture base

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• **Premature occlusal contacts**

At the **first appointment** after insertion of complete dentures, the presence of generalized soreness on the crest of the mandibular ridge is **most likely due** to improper occlusion *(premature occlusal contacts)*. To identify these, the best method in the mouth is to use disclosing wax that is slightly warmed. Insert the wax **bilaterally** and have the patient close into centric. The prematurities will show up as windows in the wax. Once centric is complete, be sure to check eccentric movements.

Important: Acrylic spicules, inaccurate denture bases and trapped food can all cause ulcers as well. If an acrylic spicule is found, it should be reduced. If an inaccurate denture base is suspected, it should be relined.

Notes:

1. After relining dentures, if a patient constantly returns for adjustments due to sore spots on the ridge, **check the occlusion.** The relining procedure may have changed the centric relation contacts.
2. Errors in occlusion may be checked **most accurately** by remounting the dentures on the articulator using remount casts and new interocclusal records.

The **inferior surface** of the maxillary occlusion rim should be parallel to:

- Frankfort's plane
- Camper's line
- Fox plane
- Horizontal condylar inclination

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- **Camper's line** → which is the line running from the inferior border of the ala of the nose to the superior border of the tragus of the ear.

Occlusion rims are the resultant product after adding **base plate wax to a record base** in order to approximate the tooth position and arch form expected in the completed denture.

Occlusion rims are used to:

- Determine and establish the vertical dimension of occlusion
- Make maxillo-mandibular jaw records
- Establish and locate the future position of the artificial teeth

Notes:

1. A good starting point for determining the vertical length of the maxillary occlusion rim is a point approximately 2 mm below the upper lip when it is relaxed.
2. When recording **centric relation** for a removable partial denture, the occlusion rim should be **attached to** the completed partial denture framework instead of a record base as used with a complete denture.
3. If at the tooth try-in appointment the teeth need to be adjusted to correct the centric occlusion, the **best way** to do this is to take a new centric relation record and remount.

The **most frequent cause** of porosities in a denture is:

- **Insufficient pressure** on the flask during processing
- **Insufficient material** in the mold
- A **rapid elevation** in temperature to 212°F causing vaporization of the liquid

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- **Insufficient pressure on the flask during processing**

Acrylic resin used for denture repairs should be under **20-30 psi air pressure** while being processed to help eliminate porosities. These porosities, if present, will usually occur in the **thickest part** of the denture. Self-cured resins are generally used for repairs instead of heat-cured resins because the risk of distorting the denture is less.

Notes:

1. When there is a rapid elevation in temperature causing vaporization of the liquid, the vapor is then trapped as **gas bubbles.**
2. Porosities will also occur if the **packing** and **processing** of the powder and liquid resin is **too plastic** *(stringy or sandy)*. This permits the liquid to vaporize and, at the same time, does not allow sufficient pressure during closure of the flask.

All of the following are disadvantages to immediate denture therapy. Which one is considered to be the **major disadvantage** to immediate denture therapy?

- Increased post-insertion care
- Increased post-insertion soreness
- Not being able to have an anterior tooth try-in to evaluate esthetics
- Greater complexity of clinical procedures
- A higher cost of treatment

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- **Not being able to have an anterior tooth try-in to evaluate esthetics**

Other drawbacks of immediate dentures:

- **Increased post-insertion care,** including relining or remaking the dentures. Contour changes occur in the healing residual ridge for 8-12 months.
- **Increased post-delivery soreness.** The combination of post-extraction pain and denture related trauma often produces greater discomfort during the first few days following insertion.
- **Greater complexity of clinical procedures.** For example, border molding and final impressions are more difficult when natural teeth remain.
- **Higher total cost of treatment.** There is an increased expense due to the need for relines and repeated equilibration of the occlusion.

Recommended two-step schedule of tooth removal:

- **First step** → extract all posterior teeth **except** a maxillary first premolar and its opposing tooth. This leaves a posterior **"stop"** in order to maintain the vertical dimension of occlusion.
- **Second step** → after the posterior residual ridges exhibit acceptable clinical healing, the second phase of treatment, that of denture fabrication, can begin. The anterior teeth will be extracted at the time of denture insertion.

The disadvantage (s) of an immediate complete denture is (are):

- Esthetics is compromised
- Speech adaption is difficult
- Extraction sites are susceptible to trauma
- Relining / rebasing of the denture is required in 8-12 months
- Masticatory function is lost during the healing phase
- All of the above

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- **Relining / rebasing of the denture is required in 8-12 months**

***Relining though simple has to be carried out within 8-12 months depending upon the rate of alveolar ridge resorption. Also increased post-delivery soreness for a few days can be encountered.

Advantages of immediate dentures:

- **Continuously acceptable esthetics.** Immediate dentures are esthetically advantageous in that the patient is never without either natural or artificial teeth.
- **Improved speech adaption.** Immediate dentures require only one period of speech adaptation, whereas conventional denture treatment requires two; one after the teeth are extracted and another after the dentures are delivered.
- **Protection of the extraction sites from trauma.** Dentures act as a type of bandage over the clot filled sockets.
- **Continuously acceptable masticatory function.** The patient retains some semblance of chewing ability during the healing process.
- **Prevention of tongue enlargement**. When natural teeth are lost and not replaced, the tongue tends to expand into the available space.

To help the patient get through the first day of wearing immediate dentures, instruct him to do the following:

- **Do not** remove the dentures
- **Eat** soft foods
- Return in **24 hours**

All of the following statements are true concerning a face-bow or a face-bow transfer **except:**

- The face-bow is a caliper-like device used to record the patient's maxilla / hinge axis relationship *(opening and closing axis)*
- If the transfer is done properly, the arc of closure on the articulator should duplicate that exhibited by the patient
- The face-bow transfer is a maxillo-mandibular record
- The face-bow transfer is used to transfer the maxilla / hinge axis relationship to the articulator during the mounting of the maxillary cast

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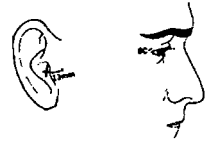
• **The face-bow transfer is a maxillo-mandibular record**

***This is **false;** it is a record used to **orient the maxillary cast to the hinge axis on the articulator.**



T = Tragus of ear **OC =** Outer canthus of the eyes

Several varieties of arbitrary face-bows are available. All are based on an average location of the hinge axis and will yield an error of 2 mm or less in the majority of patients. Arbitrary rotational centers are generally located over measured points on the face or by some type of earpiece. One average measurement *(above picture)* places the rotational point 13 mm anterior to the distal edge of the tragus of the ear, along a line from the superior-inferior center of the tragus to the outer canthus of the eye. The condylar styli of the face-bow are then placed directly over the dots.

The **posterior palatal seal** for a maxillary denture:

- Is placed 3 mm posterior to the vibrating line
- Is not necessary when fabricating a complete denture on a patient with a flat palate
- Is not necessary if a metal base is used
- Will vary in outline and depth according to the palatal form of the patient

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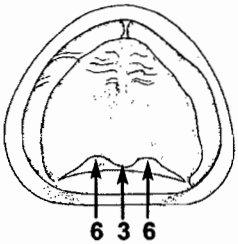
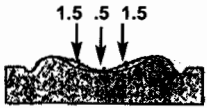
• **Will vary in outline and depth according to the palatal form of the patient**

Posterior Palatal Seal





The figure demonstrates the following depths along the posterior denture border:

- .5 mm extending 3 mm on **both sides of the midline.**
- 1.5 mm lateral *(to the .5mm extending 3 mm on both sides of the midline)* extending up to the medial boundary of the pterygomaxillary notches.
- The **width of the seal anteriorly** is characterized by a concave surface, 3 mm in the midline and 6 mm in the mid-lateral areas.

Note: These measurements are **generalizations.**

Remember: The placement of the posterior palatal seal is the responsibility of the **dentist** and **should never** be delegated to a lab technician.

Which of the following landmarks is **least relevant** to the location of the posterior palatal seal area?

- Pterygomaxillary notch
- Vibrating line
- Hamular process
- Fovea palatinae

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- **Hamular process**

Landmarks for Posterior Palatal Seal

- The **posterior outline** is formed by the **"ah"** line or **vibrating** line and passes through the two pterygomaxillary *(hamular)* notches and is close to the fovea palatini.
- The **anterior outline** is formed by the **"blow"** line and is located at the **distal extent of the hard palate.**

Note: Excessive depth of the posterior palatal seal will **usually result** in unseating of the denture.

Remember: The posterior palatal seal **will vary** in outline and depth according to the palatal form of the patient.

Functions of the Posterior Palatal Seal:

- Completes the border seal of the maxillary denture
- Prevents impaction of food beneath the tissue surface of the denture
- Improves the physiologic retention of the denture
- Compensates for shrinkage of the denture resin during processing

In metal-ceramic restorations, **failure** or **fracture** usually occurs:

- In the porcelain
- At the porcelain-metal interface
- In the metal

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• **In the porcelain**

One of the major reasons for the acceptance of porcelain fused to metal restorations is its greater strength and resistance to fracture. The combination of porcelain and metal, fused together, is stronger than porcelain alone. Because true adhesion occurs, the bond strength is such that failure or fracture will occur **in the porcelain** farther than at the porcelain-metal interface.

Important points concerning the metal-ceramic crown:

- The **necessary thickness** of the metal substructure is **0.5** mm
- The **minimal** porcelain thickness is **1.0-1.5 mm**
- Based on the above points, the tooth reduction necessary for the metal-ceramic crown is **approximately 1.5-2.0 mm.** The labial shoulder width is ideally **1.5 mm.**
- The **most frequent cause** of porosity in the porcelain is inadequate condensation of the porcelain.
- The effectiveness of condensing porcelain powder to reduce shrinkage is determined by the **shape and size** of the particle.

Remember: Porcelain is much stronger under compressive forces than it is when subjected to tensile forces by the opposing teeth. Porcelain fracture in all-ceramic restorations can be avoided by keeping the angles of the preparation rounded.

The strength of a solder joint is proportional to its:

- Porosity
- Thickness
- Surface area
- All of the above

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• **Surface area**

Soldering is used in dentistry to **connect** bridgework and in fabricating orthodontic appliances. Gold solders are generally used for fixed bridgework and silver solders for orthodontic appliances. It is important that the **solder melt at least 150°F below the fusion temperatures** of the metals or alloys being solders *(for obvious reasons).*

A **good solder joint** between 2 castings requires **clean surfaces** and **free electrons** present on the surfaces.

Commonly used **dental solders** include:
• Au/Cu/Sn/Zn
• Ag/Cu/Sn

Note: The bonding of the solder is contingent upon **wetting** of the joined surfaces by the solder, and not upon melting of the metal components.

Cleanliness is the **most important prerequisite** of soldering, since the soldering process depends upon wetting of the surfaces to achieve bonding. **Fluxing** is the oxidative cleaning of the area to be soldered. Fluxes *(most commonly* **borax***)* are used to dissolve surface impurities and to protect the surface from oxidation while heating. **Note:** Fluxing is also performed on molten metal alloys during the casting of a crown or partial denture framework.

Which of the following **are indications** for fixed bridgework or important considerations to think about when contemplating the fabrication of fixed bridgework for a patient?

- A limited number of **edentulous areas** which would not otherwise be more satisfactorily restored with a removable partial denture
- The need to **prevent** the over-eruption of opposing teeth and the drift of teeth neighboring the edentulous space
- The **presence of** suitable abutment teeth → favorable crown / root ratio, adequate alveolar support, absence of apical pathology, etc.
- Esthetics
- Patient motivation, including time availability
- Clinical and technical ability
- All of the above

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• **All of the above**

Contraindications for fixed bridgework:

- Poor oral hygiene
- High caries rate
- Multiple spaces in the arch or teeth likely to be lost in the near future
- Space not detrimental to the maintenance of arch stability or dental health
- Unacceptable occlusion
- Bruxism

Notes:

1. If the clinical and technical skills of the dentist **do not match** the demands of the case, fixed bridgework should **not be undertaken** because a failed bridge is likely to be more detrimental to dental health than a failed removable partial denture.
2. Unless **specifically contraindicated,** fixed restorations **are always** the treatment of choice.
3. Fixed bridgework can be used in conjunction with removable partials. **Example:** A patient with a couple of missing anterior teeth and no posterior teeth. Treatment could be fixed bridgework in the anterior and a partial denture replacing posterior teeth.
4. Although somewhat controversial, the literature recommends that you should **not splint natural teeth and implants in a fixed partial denture.** Implants have no periodontal ligament and so do not have the same capacity to absorb shocks as do natural teeth *(they have different mobilities).* When this bridge is subject to occlusal loading, the difference has been shown to be detrimental to the natural teeth as well as cause bone loss around the implants.

Endodontically treated teeth that have been restored with a **cast post and core, and crown,** are subjected to the high incidence of:

- Periodontal disease
- Recurrent caries
- Vertical root fracture
- The need for an apicoectomy

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- **Vertical root fracture**

***The **main symptom** will almost always be **pain when biting.** The radiograph usually appears normal.

Advantages of using a **post and core** as opposed to a **post crown** when restoring endodontically treated teeth:

- The **marginal adaption** and fit of the restoration is independent on the fit of the post.
- The restoration **can be replaced** at some time in the future, if necessary, without disturbing the post and core.
- If the **endodontically treated tooth** is to serve as a bridge abutment, it is not necessary to make the root canal preparation parallel with the line of draw of other preparations → it can be treated as an independent abutment.

The **post and core,** when used, is made separate from the final restoration. The crown is then fabricated and cemented over the core just as a restoration would be placed over a preparation done in tooth structure.

For teeth with **little or no clinical crown** that have roots with adequate length, bulk, and straightness, a post and core can be utilized. For posterior teeth with less extensive destruction of coronal tooth structure, or for those possessing less favorable root configurations, a pin retained amalgam or composite core can be used.

All of the following are **indications** for porcelain veneers **except:**

- **Coverage of labial surface defects** → hypoplasia of the enamel
- **Masking of discolored teeth** → tetracycline staining, discoloration following loss of vitality
- **The severe imbrication of teeth**
- **Repair of structural damage** → fractured incisal edges
- **Improvement of tooth contour** → peg-shaped lateral incisors
- **Reduction of spacing in cases** when orthodontics would be inappropriate

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- **The severe imbrication of teeth** → this is a contraindication to porcelain veneers

***Other contraindications to porcelain veneers include:** traumatic occlusal contacts, unfavorable morphology, insufficient tooth structure, and insufficient enamel.

Technique for Insertion of Porcelain Veneers

- The veneer should be **tried in wet** with either a drop of water or glycerine to check for fit.
- A reliable estimate for the possible post-cementation appearance with try-in pastes can also be performed.
- The **veneer fit surface should be cleaned** to remove any saliva contamination or try-in composite.
- If the fit surface **has not previously been treated with silane** and protected with light-cured unfilled resin, **this should be done at this stage.**
- The enamel surface should be cleaned with **pumice and water.**
- While protecting adjacent teeth with matrix strips, **the enamel is acid-etched.** The etched surface is washed and dried and a layer of unfilled bond resin is applied and thinned with **oil-free air.**
- An appropriate shade of light-cured composite is applied to the **fit surface of the veneer** which is "puddled" into place on the tooth surface.
- **Gross excess of composite** should be removed and light-curing completed.
- Remaining excess composite is removed with finishing diamond burs, discs, strips, etc., and the margins finely polished.
- The patient should be seen in approximately one week.

The **absolute maximum** number of posterior teeth, which can be safely replaced with a fixed bridge, is:

- One
- Two
- Three
- Four

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- **Three** →→→ and three teeth should be replaced only under **ideal conditions.** Any bridge replacing more than two teeth should be considered high risk.

Important: One factor that limits the length of the pontic span is the abutment teeth's ability to accept the additional occlusal load while providing adequate support to the cemented fixed partial denture. Ante's law states that the root surface area of the abutment teeth supported by bone must equal or surpass the root surface area of the teeth being replaced with pontics.

An edentulous space involving **four adjacent teeth** other than four incisors is usually **best treated with a removable partial denture.** If more than one edentulous space exists in the same arch, even though each of them could be individually restored with a bridge, it may be desirable to restore them with a removable partial denture. This is especially true if the spaces are bilateral and each one involves two or more missing teeth.

Third molars can rarely be used as abutments, since they frequently display incomplete eruption; short, fused roots; and a marked mesial inclination in the absence of a second molar.

Remember:
- Splinting adjacent abutment teeth in a fixed bridge is primarily done to improve the distribution of the occlusal load.
- In order to maintain and protect the health of the gingival tissues and prevent recession, the correct contour of the crown's gingival one-third to one-fifth and interproximal areas are most important in the final restoration.
- An anterior fixed bridge is **contraindicated** when there is considerable resorption of the residual bridge. A removable partial denture would be **indicated** in this case.
- **Horizontal loads** *(or forces)* on natural or abutment teeth are **most destructive** to the periodontium.

A **reverse ¾ crown** is most frequently fabricated for a:

- Maxillary premolar
- Mandibular premolar
- Mandibular molar
- Maxillary molar

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• **Mandibular molar**

This design **preserves the lingual surface** and is indicated for restoring mandibular molars with damaged buccal surfaces and intact lingual surfaces. It is also useful on teeth with severe lingual inclinations where large quantities of tooth structure would be destroyed if a full veneer crown were to be used.

The standard **three-quarter crown** is a partial veneer crown in which the **buccal surface** is left uncovered. It is the **most commonly** used form of the partial veneer crowns.

A patient with a **high caries index,** short clinical crowns, and minimal horizontal overlap **would not** be a candidate for partial veneer crowns. The restoration of choice would be a full metal-ceramic crown.

Note: Retention and **resistance forms** in full coverage preparations on short molars can be enhanced by placing several vertical grooves.

High-gold noble alloys, which are used in fabricating metal-ceramic restorations, are:

- 50% gold, platinum, and palladium
- 75% gold, platinum, and palladium
- 98% gold, platinum, and palladium
- 100% gold, platinum, and palladium

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- **98% gold, platinum, and palladium**

Remember: Noble alloys *(gold, platinum, and palladium)* **do not** oxidize on casting. This feature is important in a metal substrate so that oxidation at the **metal-porcelain interface** can be controlled by the addition of trace elements to the metal *(silicon, indium, and iridium).*

Classification of PFM Alloys

- **High-gold noble alloys** → 98% gold, platinum, and palladium with trace elements. *****This is the best to use.**
- **Palladium-silver alloys** → 50-60% palladium and 30-40% silver *(which is not a noble metal and thus oxidizes on casting).*
- **Nickel-chromium alloys** → 70-80% nickel and 15% chromium. These base metal alloys readily oxidize and can create porcelain-to-metal interface problems.

Porcelain adheres to metal primarily by a **chemical bond.** A covalent bond is established by sharing O_2 with the elements present in the porcelain and the metal alloy. These elements include silicon dioxide *(SiO_2)* in the porcelain and oxidizing elements such as silicon, indium, and iridium in the metal alloy.

Periodontal health of the gingival tissues is a major concern when planning any fixed prosthodontic treatment. For **optimum periodontal health,** restoration finish lines should be:

- Within the sulcus at least 1.0 mm and away from the free gingival margin without encroaching on the biologic width
- Terminated at the free gingival margin
- Supragingival whenever possible *(at least 0.5 mm from the free gingival margin)* to allow for hygienic cleansing
- As far as possible subgingivally into the attachment apparatus

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- **Supragingival whenever possible *(at least 0.5 mm from the free gingival margin)* to allow for hygienic cleansing**

***There is general agreement among dentists and researchers that optimum fixed prosthetic restorations will display supragingival finish lines.

Such positioning is **quite often not possible** because of **esthetic** or caries considerations. Subsequently, the margin must be placed subgingivally. If a margin needs to be placed subgingivally, the major concern is **not to extend** the preparation into the attachment apparatus. If the margin does extend into the attachment apparatus, a constant gingival irritant has been constructed and **ultimately the crown will fail.** In this case, the tooth should have had crown lengthening performed on it **prior** to final crown preparation.

The **most important criterion** for a gingival margin on a crown preparation is that its position is easily discernible → must be able to recognize it easily. **Note:** The most common complaint of lab technicians regarding a PFM prosthesis is improper margins in the impression.

Remember: The **optimum margin** for a casting is an **acute edge** with a nearby bulk of metal. This acute edge or angle can be easily burnished to improve its fit.

Note: A **butt joint,** as typified by a shoulder, is the **poorest type** of finish line that can be used with cast metal restorations.

When casting **conventional gold alloys,** which type of investment material is used?

- Silica-bonded investments
- Phosphate-bonded investments
- Gypsum-bonded investments

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- **Gypsum-bonded investments**

A **dental investment** is a refractory material that is used to surround the wax pattern during the procedure of fabricating the metallic permanent restoration. It **forms the mold** into which the alloy is cast after the wax has been eliminated.

An investment material to be used for a casting mold should expand on setting and heating to compensate for the shrinkage of molten metal as it solidifies. Metal casting alloys have different melting ranges - only pure metals and alloys of eutectic composition have a melting point. The melting range of gold casting alloys *(approx 900°C)* is lower than that of Co-Cr alloys *(approx 1350°C)*. Therefore, investment materials used for gold casting alloys are sometimes different from those used for Co-Cr alloys. The investment material should be of a suitable consistency for adaptation to the wax model and have a reasonable setting time. To withstand the temperatures required for the casting process there should be no distortion, no decomposition; the investment should not fragment or disintegrate under the impact of the molten metal; the material should be porous to allow the escape of air and gases and the investment should be easily removed from the casting after cooling.

Classification of Dental Investment Materials

- **Gypsum-bonded** investments → **binder** is **gypsum** *(calcium sulfate hemihydrate)*. Used when casting **conventional gold alloys.**
- **Phosphate-bonded** investments → **binder** is a metallic oxide and a **phosphate.** Used when casting **base metal alloys** for metal-ceramic crowns.
- **Silica-bonded** investments → **binder** is a **silica gel.** Used when **casting base metal alloys** for partial denture frameworks.

The **refractory material** for these investments is either quartz or cristobalite. This material provides the thermal expansion for the investment. **Note:** The expansion of the investment provides a larger mold **to compensate** for the subsequent contraction of **the alloy**.

All of the following statements regarding metal-ceramic restorations are true **except:**

- The metal and porcelain must have compatible melting temperatures as well as compatible coefficient of thermal expansions
- The metal's melting temperature should be at least 300-500°F higher than the fusing temperature of the porcelain
- The metal coping should preferably have sharp surfaces to prevent shrinkage of the porcelain
- The absolute minimum required thickness of the porcelain is 0.7mm and the metal's thickness may vary from 0.3 to 0.5mm for noble alloys and 0.2mm for base metal alloys
- In function, glazed porcelain on the occlusal surface removes 40 times as much of the opposing tooth structure than gold

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• **The metal coping should preferably have sharp surfaces to prevent shrinkage of the porcelain**

***This is **false;** the metal coping must have all of its surfaces smooth and rounded to prevent porcelain shrinkage.

Note: The **purpose** of the metal coping is to ensure the fit of the crown and to maximize the strength of the porcelain veneer.

Important points to remember concerning the metal coping or substructure of a metal-ceramic crown:

1. The metal must have **proper thickness** *(0.5 mm for high-gold noble alloys and 0.2 mm for base metal alloys)* so as not to distort during the firing of the porcelain. It should be **reinforced in load-bearing areas,** such as the interproximal space and can be strengthened in areas where the metal exists alone, such as the lingual collar.
2. The outer junction of porcelain to metal should be at a **right angle** *(to avoid burnishing of the metal and subsequent fracture of the porcelain).*
3. All of the porcelain should be **supported by metal.**

Important: If your margins were **all closed** at the metal try-in appointment and when the crown came back from the lab they **are all open,** check the contacts. They are probably **too tight** *(over-bulked porcelain).*

Notes:

1. Porcelain that is baked onto a high-fusing gold alloy may exhibit a green discoloration due most likely to contamination of the metal by copper traces.
2. The **best measure** of the potential clinical performance of a casting alloy is its **ADA certification.**

When preparing a tooth for either a full-or partial-veneer casting, the functional cusp bevel is an integral part of the occlusal reduction phase. The functional cusp bevel:

- Enhances resistance form when buccal-to-lingual forces are applied
- Serves as a positive stop when the casting is seated during cementation
- Relieves the functional cusp from additional stresses when the restoration is loaded in the long axis of the tooth
- Provides space for restorative material of adequate thickness in an area of heavy occlusal contact

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- **Provides space for restorative material of adequate thickness in an area of heavy occlusal contact**

The **functional cusp bevel** is an area of reduction over the functional cusps that allows for extra thickness of metal in this area of heavy occlusal contact in centric occlusion as well as in lateral movements. The **functional cusps** are those that oppose the central fossae of the teeth in the opposite arch *(buccal cusps on mandibular teeth, lingual cusps on maxillary teeth).*

The **primary reason** for choosing a $^{3}/_{4}$ crown over a full cast crown is **tooth structure is spared.**

Other **advantages** to the use of partial veneer restorations *(three-quarter & seven-eighths crowns)*:

- A great deal of the margin is in an area **accessible** to the dentist for finishing and to the patient for cleaning.
- Less of the **restoration margin** is in close proximity to the gingival crevice, thus lessening the opportunities for periodontal irritation.
- Can be more **easily seated** completely during cementation.
- With at least part of the **margin visible,** complete seating of a partial veneer crown is more easily verified by direct vision.
- If it is ever necessary to conduct an electric pulp test on the tooth, a portion of enamel is unveneered and accessible.

Notes:

1. The path of insertion of an **anterior** three-quarter crown parallels the incisal 1/2 to 2/3 of the labial surface, not the long axis of the tooth. For a **posterior** three-quarter crown it parallels the long axis of the tooth.
2. A pin modified three-quarter crown can preserve the facial surface and one proximal surface. This is preferred in cases which require repairing of severe lingual abrasion on incisors and canines, avoiding other more destructive options like full veneer metal-ceramic restorations.

The preparation for a **full gold crown** involves circumferential and occlusal reduction of between:

- 0.5 and 1.0 mm
- 2.0 and 2.5 mm
- 3.0 and 3.5 mm
- 4.0 and 4.5 mm

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• **0.5 and 1.0 mm**

This reduction is done to eliminate undercuts and create space for sufficient metal to ensure **adequate strength** of the crown.

Remember: In preparing a tooth for a **metal-ceramic crown,** it is necessary to create space for 0.5 mm of metal **plus at least** 1.0 mm of porcelain *(preferably 1.5 mm)* to ensure adequate strength and optimum esthetics of the ceramic material. **Supporting cusps require 2 mm of the reduction.** The opposing walls should converge no more than 10 degrees. A chamfer finish line and all margins should be placed supragingivally when possible.

The same amount of overall tooth reduction is needed for a **metal-ceramic crown** as for an **all-ceramic crown** *(1.5-2.0 mm).* However, for **all-ceramic restorations,** the preparation needs to be well-rounded with **no sharp angles** to avoid porcelain fracture.

Note: The **most frequent** cause of failure of a crown *(regardless of which type)* is the lack of attention to tooth shape, position, and contacts. **Important:** For gingival health, the correct contour of interproximal gingival areas and the gingival third are most important.

Important: Gold is regarded as a **more favorable material** for the occlusal surface as its wear characteristics are more in harmony with enamel; porcelain is considered to be **the cause of accelerated wear** of the opposing dentition. **Gold** would certainly be preferred for the restoration of occlusal surfaces in the presence of a **tooth-grinding habit.**

Which of the following best describes **"strain hardening"** or **"work hardening"?**

- Hardening *(or deformation)* of a metal at room temperature
- Hardening *(or deformation)* of a metal at a very high temperature
- Softening a metal by controlled heating and cooling
- Softening a metal at room temperature

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• **Hardening *(or deformation)* of a metal at room temperature**

In polycrystalline metal, dislocations *(defects)* tend to build up at the grain boundaries. Also, the barrier action to slip at the grain boundaries causes the "slip" to occur on other intersecting slip planes. Point defects increase and the entire grain may eventually become distorted. Greater stress is required to produce further "slip" and the metal becomes stronger and harder. The process is known as **strain hardening** or **work hardening**. The latter term is derived from the fact that the process is a result of cold work, i.e., deformation at room temperature, in contrast to the effect of working at a higher temperature, such as in forging. The **ultimate result of strain hardening,** with further increase in cold work, is **fracture.**

The phenomenon of cold work and strain hardening is familiar to everyone. For example, one way to cut a wire is to bend it back and forth rapidly between the fingers. When all the slip possible has occurred, the wire fractures.

Important: The surface hardness, strength, and proportional limit of the metal are **increased** with strain hardening, whereas the ductility and resistance to corrosion are **decreased.** However, the elastic modulus is **not changed** appreciably.

Notes:

1. Under a microscope, elongated grains in the microstructure of a wrought wire indicate that the wire has been cold worked or strain hardened.
2. A slip is a deformation process requiring the simultaneous displacement of an entire plane of atom A , relative to the plane B, below it.

Which of the following best describes the term **"quenching"?**

- A metal is elevated to a temperature above room temperature and held there for a length of time
- A metal is rapidly cooled from an elevated temperature to room temperature or below
- Softening a metal by controlled heating and cooling
- None of the above

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- **A metal is rapidly cooled from an elevated temperature to room temperature or below**

This is **usually done** for one of the following two reasons:

1. To preserve at room temperature a phase ordinarily stable only at elevated temperatures.
2. To rapidly terminate a process that only occurs at elevated temperatures .

Important: To achieve a **softened condition** for a Type III dental gold alloy, the casting should be **quenched** in water within 30-40 seconds of being made.

Notes:

1. **Heat treatment** is the subjection of metals and alloys to controlled heating and cooling after fabrication to relieve internal stresses and improve their physical properties. Methods include annealing, quenching, and tempering.
2. **Annealing** is controlled cooling of a material to **increase ductility and strength.** The process involves first heating a material *(usually glass or metal)* for a given time at a given temperature, followed by slow cooling.
3. **Fritting** is a process of manufacturing low and medium fusing porcelains. It involves raw constituents of porcelain to be fused, quenched, and ground back to an extremely fine powder. This "frit" can be added over by other metallic substances to produce color in porcelain.

Gypsum bonded investment material can be used for:

- Dowel crowns to be cast in silver-palladium alloys
- Titanium crowns and copings
- The substructure for metal ceramic crowns
- Type IV gold alloys
- None of the above
- All of the above

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• **Dowel crowns to be cast in silver-palladium alloys**

Dowel cores **do not require** as much expansion as do crowns. So even though they are cast with Ag-Pd alloys *(alloys that require a high temperature for expansion)*, a gypsum bonded mold is used and heated to only 1200°F. Type I, II, and III Gold alloys can also be cast in gypsum bonded investment material.

The substructures for metal ceramic crowns and Type IV Gold requires heating above 2100°F. These are invested in phosphate bonded material. Any alloy with a casting temperature in excess of 2100°F *(1150°C)* should be cast in an investment with a binder other than gypsum. High temperatures cause decomposition of calcium sulfate in the gypsum binder with the resultant release of contaminating sulfur into the mold.

Magnesium phosphate reacts with primary ammonium phosphate to produce magnesium ammonium phosphate which gives the investment its strength at room temperature. At higher temperatures, silicophosphates are formed which give the investment its great strength.

The **metal-ceramic alloys** must have a high melting range so that the metal is solid well above the porcelain baking temperatures to minimize distortion *(sag)* of the casting during porcelain procedures. A high sag factor will lead to distortion of bridge spans when the porcelain is fired. **Remember:** When casting a certain alloy, make sure you use a crucible that **has not been used** for other alloys.

The **path of insertion** for an anterior ¾ crown should:

- Be perpendicular to the incisal one-half of the labial surface rather than the long axis of the tooth
- Be parallel to the incisal one-half to two-thirds of the labial surface rather than the long axis of the tooth
- Be parallel to the long axis of the tooth
- Be parallel to the cervical one-third of the labial surface rather than the long axis of the tooth

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- **Be parallel to the incisal one-half to two-thirds of the labial surface rather than the long axis of the tooth**

***Important:** If the path of insertion is made parallel to the long axis of the tooth, the labio-incisal corner will be sacrificed and an **unnecessary display of gold** will result.

Two factors that must be dealt with **successfully** to produce an anterior ¾ crown with a minimal display of gold:

1. **Path of insertion and groove placement**
2. **Placement and instrumentation of extensions**
   - Proximal extensions must be done with thin diamonds and hand instruments from a lingual approach to minimize the display of gold. They should be extended facially to a cleansable area without destroying the facial contour of the tooth.

Note: The **anterior** three-quarter crown **is not used** as frequently today as it once was. Unsightly and unnecessary displays of gold in poor examples of this restoration have made it less popular with the public and dentists alike. However, the **standard three-quarter crown** on a maxillary anterior tooth need not show large quantities of gold if prepared correctly.

When treatment planning for fixed prosthodontics, all of the following information can be obtained by studying diagnostic casts **except:**

- The length of the abutment teeth can be accurately gauged
- The true inclination of the abutment teeth will be evident
- The presence of periodontal pockets and the crown-to-root ratio of potential abutment teeth
- Mesial / Distal drifting, rotation, and faciolingual displacement of potential abutment teeth can be clearly seen

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- **The presence of periodontal pockets and the crown-to-root ratio of potential abutment teeth**

***Important:** The presence of periodontal pockets and the crown-to-root ratio of potential abutment teeth **cannot** be determined by studying diagnostic casts. You need to do an exam and have x-rays in order to obtain this information.

More information that can be obtained by studying the diagnostic casts:

- It allows an **unobstructed view** of the edentulous areas and an accurate assessment of the span length, as well as its occlusogingival dimension.

- The **curvature of the arch** in the edentulous region can be determined, so that it will be possible to predict whether the pontic*(s)* will act as a lever arm on the abutment teeth.

- A thorough **evaluation of wear facets,** their number, size, and location is possible when they are viewed on casts. Excessive wear on occluding surfaces of teeth usually results from a disharmony between centric occlusion and centric relation.

All of the following statements concerning pontics are true **except:**

- With regard to the ease of cleaning and good tissue health; proper pontic design is **more important** than the choice of material used in fabricating the pontic
- The **contour** and **nature** of the pontic contact with the ridge is very important
- The area of contact between the pontic and the ridge should be **small**
- The portion of the pontic approximating the ridge should be as **concave** as possible
- The pontic should exert **no pressure** on the ridge *(passive contact with no blanching of the tissue)*

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• **The portion of the pontic approximating the ridge should be as concave as possible**

***This is **false;** the portion of the pontic approximating the ridge should be as **convex** as possible.

Pontic design and selection **directly impact** periodontal health. Pontics should contact keratinized attached tissue and rest passively, free of pressure, to prevent ulcerations and plaque buildup. Pontic designs with concavities *(such as the saddle-shaped pontic),* are difficult to clean because the hollow or depression on their inner surface is inaccessible to conventional methods of oral hygiene. Egg- or bullet-shaped pontics are easiest to clean because they are convex in all aspects and contact the residual ridge at a single point.

Most important: Whatever pontic is used, it must be properly designed to **prevent** an unhealthy response to the underlying ridge mucosa. **The pontic must:**

- Be **nonporous, smooth,** and have a polished surface
- Make passive **pinpoint contact** with the gingival tissue
- **Not be concave** in two directions
- Be **readily cleanable** by the patient
- Be **narrower** at the expense of the lingual aspect of the ridge
- Be on as **straight a line** as possible between the retainers to prevent any torquing of retainers or abutments.

Note: Excessive tissue contact has been cited as one of the major causes of failure of fixed bridges.

Glazed porcelain, polished gold, unglazed porcelain, and polished acrylic are preferred in that order for their acceptability to the soft tissue.

The **ideal** crown-to-root ratio of a tooth to be utilized as a bridge abutment is:

- 3:1
- 2:1
- 1:2
- 1:1

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• **1:2**

This high a ratio is **rarely achieved,** however, and a ratio of 2:3 is a more realistic optimum. A ratio of 1:1 is the minimum ratio that is acceptable for a prospective abutment under normal circumstances.

The crown-to-root ratio alone is **not adequate** criteria for evaluating a prospective abutment tooth. **Root configuration** is an important point in the assessment of an abutment's suitability from a periodontal standpoint. Roots that are **broader labio-lingually** than they are mesiodistally **are preferable** to roots which are round in cross section. **Multi-rooted** posterior teeth with **widely separated roots** will offer **better periodontal support** than roots which converge, fuse, or generally present a conical configuration. Single-rooted teeth with an irregular configuration or with some curvature in the apical third of the root are preferable to the tooth which has a nearly perfect taper. **Root surface area** of the prospective abutments should also be evaluated.

All of the following are **factors** in fixed bridgework design:

- Root configuration
- Crown-to-root ratio
- Axial alignment of teeth
- Length of the lever arm *(span)* **Note: Replacing three teeth is maximum!!!**

Remember: Parallelism of abutment preparations is **best determined** by the long axis of the preparations.

Which agent is most commonly added to the flux *(used for soldering stainless steel or cobalt chromium alloys)* with a specific purpose to dissolve the passivating film *(chromium oxide film)* that may prevent wetting of the metal with the solder?

- Sodium pyroborate
- Boric acid
- Silica
- Potassium fluoride

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• **Potassium fluoride**

***Other **constituents of the flux** are sodium pyroborate *(55%)*, boric acid *(35%)*, and silica *(10%)*.

In addition to the usual reducing and cleaning agents incorporated in a flux, a flux used for soldering stainless steel or cobalt-chromium alloys also contains a **fluoride** to dissolve the passivating film supplied by the chromium *(chromium oxide film)*. The solder will not wet the metal when such a film is present. Potassium fluoride is the most common agent.

Soldering is the joining of metal components by a filler metal, or solder, which is fused to each of the parts being joined. To be biologically and mechanically acceptable, a solder joint should be circular in form and occupy the region of the contact area. The **strength of the solder joint** is increased by increasing the height of it *(as opposed to the width)*.

Cleanliness is the **prime prerequisite** of soldering. Corrosion products, such as oxides and sulfides that are present as a result of the casting process, interfere with bonding. **Flux** is placed on the surfaces to be soldered before they are heated. When it melts, the flux displaces gases and removes corrosion products by either combining with them or **reducing them.** The flux in turn is displaced by the solder, which can now form an interface with and bond to the surface being soldered.

Note: Antiflux is a material used to outline the area to be soldered in order to restrict the flow of solder. The most common antiflux is a **soft graphite pencil.**

What type of pontic design would you use in a patient with a high esthetic demand when preparing teeth numbers 9 –11 for a fixed partial denture?

- The ridge lap or saddle pontic
- The sanitary or hygienic pontic
- The modified ridge lap pontic
- An ovate pontic
- Bullet-shaped pontic

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• **The modified ridge lap pontic**

The pontic is the **suspended member** of a fixed bridge that replaces a missing tooth. This tooth substitute **must provide** patient comfort, convenient contours for hygiene, and be esthetic, if indicated.

Most Common Pontic Designs

- The **sanitary pontic** design leaves a space between the pontic and the ridge. Is most commonly used where esthetics **are not important** *(nonappearance zone)*. Convex in all areas.
- The **saddle pontic** design looks most like a tooth. Covers the ridge labiolingually with a large concave contact. Impossible to clean, **should not be used.**
- The **modified ridge lap pontic** design uses a ridge lap for minimal ridge contact. Gives the illusion of being a tooth, but possesses **all convex surfaces** for ease of cleaning. This design is the one of choice for pontics in the **appearance** zone *(where esthetics are important)* for both maxillary and mandibular bridges.

Conical pontic → rounded *(top)* and conical *(bottom)*. Suited for thin mandibular ridges in non-appearance zone.

Ovate pontic → a sanitary substitute for saddle design. Set in the concavity of the ridge *(which is either present or surgically made)* that gives the appearance that it is growing from the tooth.

Remember: The **faciolingual dimension** of the occlusal portion of pontics is determined by the **faciolingual position** of the opposing centric holding contact areas.

Which of the following **luting materials** should be the material of choice to cement a ceramic crown and can provide the strongest bond?

- Zinc polycarboxylate cement
- Glass ionomer cement
- Zinc phosphate cement
- ZOE cement
- Composite resin

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- **Composite resin**

***Ceramic crowns** are bonded with composite resin *(after etching the internal surface of the crown)* and are shown to be better in bonding strength than other available materials.

Zinc phosphate cement can also be used. It has a good compressive strength *(14000 to 16000 psi)*. Its high pH is a problem → two layers of varnish must be applied in order to protect the pulp.

On teeth whose preparations **possess adequate length** and retentive features, or when the depth of the preparation raises some concern for the vitality of the pulp, a more biologically compatible cement *(i.e., zinc polycarboxylate or zinc oxide-eugenol cement)*, should be used. Also, **zinc polycarboxylate** and **zinc oxide-eugenol cements** cements exhibit better resistance to solubility than does zinc phosphate.

Important:

- Cements **do not add** to the retentive characteristics of a crown.
- Zinc polycarboxylate cements **adhere to** calcified dental tissue *(as to glass ionomer cements)* and have superior biologic compatibility when compared to zinc phosphate cements.
- A tooth should be **wiped dry** before cementation of a crown as opposed to drying the tooth with alcohol and warm air to decrease the possibility of pulp damage.
- Always apply cement to **both** the restoration and tooth.

Which of the following marginal designs is **theoretically** the best finishing margin for **cast gold restorations,** allowing burnishing and adaptation of the gold to the tooth?

- Shoulder
- Shoulder with a bevel
- Chamfer
- Bevel or feathered edge

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• **Bevel or feathered edge**

However, in practice this finishing line is difficult to read on both the impression and die and may lead to inaccurate extension and also distortion of the wax pattern, and subsequent casting, as a result of the thin wax. It also offers the **least marginal strength** to the casting.

The **chamfer** preparation is the **preferred finishing line** for cast gold restorations. The resultant casting has sufficient marginal strength; at the same time it allows the sliding joint at its periphery to minimize the gap between the tooth and preparation, thus reducing the thickness of the cement. A well-prepared chamfer margin combines the advantage of an easily definable margin, on both the impression and die, with minimal tooth preparation.

The **shoulder** preparation is the finishing line of choice for **porcelain jacket** and **all-ceramic crown preparations.** The edge strength of porcelain is low; therefore, a butt joint is required. The shoulder provides resistance to occlusal forces and minimizes stresses in the porcelain. The margin can be easily read on both the impression and die. The **main disadvantage** is that any inaccuracies in the fit of the crown will be reproduced at the margin, resulting in an increased thickness of cement.

The **shoulder with a bevel** allows a sliding fit to occur at the margin and therefore may be used on the proximal box of inlays and the occlusal shoulder of the mandibular three-quarter crowns. It may also be used for the labial margins of metal-ceramic crowns. Providing these margins are placed just in the gingival crevice, little display of metal will be noted.

What is the general rule for **sprue pin diameter** when using a centrifugal type of casting machine?

- The diameter of the sprue pin should be equal to or **greater** than the **thickest** portion of the pattern.
- The diameter of the sprue pin should be equal to or **smaller** than the **thickest** portion of the pattern.
- The diameter of the sprue pin should be equal to or **greater** than the **thinnest** portion of the pattern.
- The diameter of the sprue pin should be equal to or **smaller** than the **thinnest** portion of the pattern.

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- **The diameter of the sprue pin should be equal to or greater than the thickest portion of the pattern**

The **sprue** is a small diameter *(10-12 gauge)* **pin** made of wax or plastic.

A **10 gauge sprue pin** can be used on most patterns, while the 12 gauge is used on small premolar patterns. The sprue should be attached to the wax pattern at its point of greatest bulk and at an angle *(45°)* that will allow the incoming gold to flow freely to all portions of the mold. Spruing at a thin area of the pattern can produce the same result as using a sprue that is too small → **shrink back porosity.** This is caused by turbulence in the flow of the molten metal which in turn creates a shrinkage void, or suck-back porosity.

Note: Low investment permeability and insufficient wind-up of the casting machine may also cause this shrink back porosity.

A **non-rigid** connector is comprised of:

- A key
- A keyway
- A key and a keyway

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• **A key and a keyway**

A **nonrigid connector** is a broken-stress mechanical union of retainer and pontic, instead of the usual rigid, solder joint.

The **most commonly used** nonrigid design consists of a **T-shaped key** that is attached to the pontic and a **dovetail keyway** placed within the retainer. The path of insertion of the key into the keyway should be **parallel** to the pathway of the retainer not involved with the keyway.

Its use is restricted to a **short span bridge,** replacing one tooth. It is indicated when retainers cannot be prepared to draw together without excessive tooth reduction. Prostheses with nonrigid connectors **should not** be used if prospective abutment teeth exhibit significant mobility.

Important: When abutment teeth are in **normal** alignment and have **good bone** support *(canine and first molars),* the **connectors of choice** are solder joints.

The anteroposterior curvature *(in the median plane)* and the mediolateral curvature *(in the frontal plane)* in the alignment of the occluding surfaces and incisal edges of artificial teeth that are used to develop balanced occlusion is called:

- Curve of Spee
- Compensating Curve
- Curve of Wilson
- Curve of Pleasure

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• **Compensating Curve**

The form of the compensating curve is entirely **under the control of the dentist.** For example, if during a try-in evaluation, a dentist notes that a protrusive excursion movement results in the separation of posterior teeth, the problem can be corrected by simply increasing the compensating curve. The **value** of the compensating curve is that it allows the dentist to alter the effective cusp angulation without changing the form of the manufactured denture teeth. The **function** of this curve is to help provide a balanced occlusion. A prominent compensating curve is required when there is a steep condylar path associated with a low degree of incisal guidance.

Orientation of the occlusal plane: The occlusal plane is an imaginary surface which is related anatomically to the cranium and which theoretically touches the incisal edges of the incisors and the tips of the occluding surfaces of the posterior teeth. It is not a plane in the true sense of the word, but represents the mean curvature of the surface. The **anterior point** of the occlusal plane is determined by the position of the anterior teeth. The **posterior determinants** are anatomical landmarks → two-thirds the height of the retromolar pads. Therefore, it is **debatable** as to the extent of control the dentist may exercise over the orientation of the occlusal plane.

Cusp inclination is the angle made by the slopes of a cusp with a perpendicular line bisecting the cusp, measured mesiodistally or buccolingually. This is under **control of the dentist** *(choosing 30º degree teeth or cuspless teeth, etc.).*

Assume that a patient wearing complete dentures for a number of years is given an oral examination and it is determined that the **vertical dimension of occlusion** has been decreased. This would cause:

- An increased vertical dimension that leaves the teeth in a clenched, closed relation in normal positions
- An occluding vertical dimension that results in an excessive interocclusal distance when the mandible is in the rest position
- An insufficient amount of interarch distance because of heavy, bony ridges
- An inability to open the mandible because of temporomandibular joint pathosis

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- **An occluding vertical dimension that results in an excessive interocclusal distance when the mandible is in the rest position**

Interocclusal distance → also called **"freeway space"** is the vertical distance or space between the incisal and occlusal surfaces of the maxillary and mandibular teeth with the mandible in the physiological rest position. The **average** interocclusal distance is **about 3 mm.** Too much interocclusal distance may result in muscular imbalance.

Vertical dimension of occlusion is the vertical length of the face as measured between two arbitrarily selected points, one above and one below the mouth, when the teeth or any substitute material *(occlusion rims)* are in contact in centric relation. **Excessive vertical dimension** may result in **trauma** to the **underlying supporting tissues** *(in a denture patient)* and **straining of the closing muscles** as well as **adversely affecting** the interocclusal distance *(decreased freeway space).*

Vertical dimension of rest is the vertical length of the face as measured between two arbitrarily selected points, one above and one below the mouth, when the mandible is in the **rest position;** in the physiologically healthy individual, there will always be a vertical space between the teeth *(freeway space)* when the mandible is in the rest position. This position is important in complete denture fabrication because it provides a guide to the vertical dimension of the occlusion.

Notes:

1. Vertical Dimension of Occlusion + Interocclusal Distance = Vertical Dimension of Rest.
2. The vertical dimension of rest is **always greater** than the vertical dimension of occlusion.

Centric relation is an example of a (an):

- Tooth-to-tooth relation
- An occlusal relation
- Bone-to-bone relation
- Balanced relation

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• **Bone-to-bone relation**

Centric relation (CR) *(also called the retruded contact position)* is the most unstrained, retruded anatomic and functional position of the heads of the condyles in the mandibular *(glenoid)* fossa of the temporomandibular joints. This is a relationship of the bones of the upper and lower jaws **without tooth contact.**

Important points about centric relation:

- The mandible cannot be forced into centric relation from the rest position because the patient's reflex neuromuscular defense would resist the applied force.
- The mandible should be relaxed and gently guided into centric relation.
- In fixed and removable prosthodontics, centric relation should be established prior to designing the frameworks.
- When a centric relation record is taken in the natural dentition, imprints of the teeth should be confined to cusp tips and the registration material should not be perforated.

The **current concept** about centric relation: it occurs when the condyles are in their most **superoanterior position,** resting on the posterior slopes of the articular eminences with the discs properly interposed.

Why do we need to know this? This position is considered to be an optimum relative position between all of the anatomic components. And more importantly, it is a repeatable reference position to mount the casts on the articulator.

Helpful hint: Having the patient swallow, turning the tongue upward towards the palate, relaxing the jaw muscles, or protruding and retruding the mandible can be effective ways to help in recording centric relation.

Which of the following statements concerning selective grinding in complete denture fabrication for centric relation **is not** true?

- Primary centric holding cusps are the maxillary lingual cusps
- Secondary centric holding cusps are the mandibular buccal cusps
- Selective grinding of the inner inclines of secondary holding cusps can be done if there is a working side interference
- Grind only the cusp tips of the upper buccal and the lower lingual *(B.U.L.L.)* cusps if they are premature in centric, lateral or protrusive movements

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- **Selective grinding of the inner inclines of secondary holding cusps can be done if there is a working side interference** → This is **incorrect,** the selective grinding of the inner inclines of secondary centric holding cusps can be done if there is a balancing side interference.

Selective grinding in centric relation:

- Ideally selective grinding should result in harmonious cusp-fossa contacts of all upper and lower fossa *(and marginal ridges of bicuspids).* Do not grind the upper lingual or lower buccal cusps. A forward slide from centric can be corrected by grinding the mesial inclines of maxillary teeth and distal inclines of mandibular teeth.
- Primary centric holding cusps are the maxillary lingual cusps. **Never grind these cusps.** See note below.
- Secondary centric holding cusps are the mandibular buccal cusps. Grind these cusps only if there is a balancing side interference.
- Only grind cusp tips if they are premature in centric, lateral, and protrusive movements. Check before grinding.

Selective grinding in working-side relation: The rule of selective grinding for interferences in working -side movements is the **Rule of B-U-L-L:**

- **B**uccal cusp inner inclines of **U**pper teeth
- **L**ingual cusp inner inclines of **L**ower teeth

Selective grinding in balancing side relation:

- Grind the inner inclines of the mandibular buccal cusps.
- **Never** grind the maxillary lingual cusps *(primary centric holding cusps).*

Note: For the **National Board Exam** questions, you can reduce the maxillary lingual cusp if it is high **in centric as well as other occlusal positions** → in reality, you should not.

Balancing side *(non-working side)* interferences generally occur on the inner aspects of the:

- Facial cusps of mandibular molars
- Facial cusps of maxillary premolars
- Lingual cusps of mandibular molars
- Facial cusps of maxillary molars

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• **Facial cusps of mandibular molars**

Working side interferences generally occur on the inner aspects of the lingual cusps of maxillary molars.

Protrusive interferences generally occur between the **distal inclines** of the facial cusps of maxillary posterior teeth and **mesial inclines** of the facial cusps of mandibular posterior teeth. The proximity of the teeth to the muscles and the oblique vector of the forces make contacts between opposing posterior teeth during protrusion potentially destructive. The **purpose** of making a record of protrusive relation is to **register the condylar path** and to adjust the condylar guides of the articulator so that they are equivalent to the condylar paths of the patient.

A **centric interference** *(forward slide)* can be corrected by grinding the mesial inclines of maxillary teeth and distal inclines of mandibular teeth.

Where do the **occlusal** contact possibilities occur during a **protrusive movement?**

- On the **maxillary mesial inclines** and **mandibular distal inclines**
- On the **maxillary mesial inclines** and **mandibular mesial inclines**
- On the **maxillary distal inclines** and **mandibular mesial inclines**
- On the **maxillary distal inclines** and **mandibular distal inclines**

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• **On the maxillary distal inclines and mandibular mesial inclines**

Anteriorly the facial surface of the lower incisors will contact the guiding inclines *(lingual)* of the upper incisors and canines.

Protrusive movement is accomplished when the mandible is moved straight forward until the maxillary and mandibular **incisors contact edge-to-edge.** This movement is bilaterally symmetrical in that both sides of the mandible move in the same direction.

In any restorative case involving **all teeth** in the mouth, the protrusive condylar path inclination will have its primary influence on the same inclines *(distal of maxillary and mesial of mandibular)*.

The pathway followed by the **anterior teeth** during protrusion may not be smooth or straight because of contact between the anterior teeth and sometimes the posterior teeth.

The mandibular movements include movements:

- Approximately 9 – 10 mm anteriorly *(protrusive movement)*
- Approximately 50 – 60 mm inferiorly *(opening)*
- Approximately 10 mm laterally
- Approximately 1 mm posteriorly *(retrusive movement)*

Which of the following best describes **Camper's line** *(plane)?*

- It is a line *(plane)* which is **determined by** the occlusal surfaces of the teeth
- It is a line *(plane)* which **extends** from the outer canthus of the eye to the superior border of the tragus of the ear
- It is the line *(plane)* **running** from the inferior border of the ala of the nose to the superior border of the tragus of the ear
- None of the above

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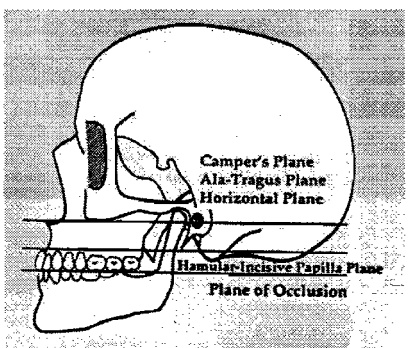
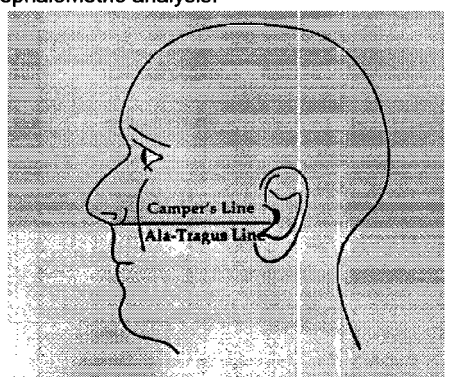
- **It is the line** *(plane)* **running from the inferior border of the ala of the nose to the superior border of the tragus of the ear**

Significance of the Camper's line → the **occlusal plane,** established by the surfaces of the wax occlusion rims, is **parallel to the Camper's line** and the interpupillary line.

Note: The **posterior determinants** of occlusion *(two-thirds the height of the retromolar pads)* have the greatest effect on the setting of the mandibular second molars.

Remember: The **Frankfort horizontal plane** extends from the outer canthus of the eye to the tragus of the ear. It is commonly used in orthodontics for cephalometric analysis.





In the intercuspal position, the **mesiolingual cusp** of a permanent **maxillary first molar** occludes where?

- **Central fossa** of the mandibular **first molar**
- **Central fossa** of the mandibular **second molar**
- The interproximal marginal ridge areas **between** mandibular first and second molars
- The interproximal marginal ridge areas **between** mandibular second and third molars

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• **Central fossa of the mandibular first molar**

Contacts in Intercuspal Position	
Maxillary Lingual Cusps	**Occludes into Area of Mandibular Teeth** *(Class counterpart or counterpart and tooth distal to it)*
First premolar	Distal triangular fossa of first premolar
Second premolar	Distal triangular fossa of second premolar
First molar Mesiolingual Distolingual	 Central fossa of first molar Distal marginal ridge of first molar and mesial marginal ridge of second molar
Second molar Mesiolingual Distolingual	 Central fossa of second molar Distal marginal ridge of second molar and mesial marginal ridge of third molar

During **non-working**, excursive movements, the permanent maxillary first molar's mesiolingual cusp escapes through:

- The lingual groove of the mandibular first molar
- The mesiobuccal groove of the mandibular first molar
- The buccal groove of the mandibular second molar
- The distobuccal groove of the mandibular first molar
- The space between the mesiobuccal and distobuccal cusps of the mandibular first molar

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• **The distobuccal groove of the mandibular first molar**

Examples:

1. The **mesiolingual cusp of the mandibular first molar** opposes the lingual embrasure between the maxillary first molar and second premolar.
2. The **mesiolingual cusp of the mandibular second molar** opposes the lingual embrasure between the maxillary second molar and first molar.

Note: The **distolingual cusp of the mandibular first molar** fits into *(opposes)* the lingual groove of the maxillary first molar.

Remember: The lingual cusp of permanent mandibular first premolars **does not** occlude with anything.

Important: During mandibular movements *(working, non-working, etc.)* the outer aspects of the **lingual** cusps of the mandibular molars will not contact their maxillary antagonists. All other areas of **buccal** and **lingual** cusps may contact during mandibular movements *(this is assuming that all occlusal relationships are normal).*

Note: In **unilateral balanced** occlusion, contact between mandibular buccal cusps and maxillary buccal cusps, along with simultaneous contact between mandibular lingual cusps and maxillary cusps will most likely occur in laterotrusive movements.

Which should **ideally** provide the **predominant guidance** through the full range of movement in **lateral mandibular excursions?**

- Premolars
- First molars
- Incisors
- Canines

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• **Canines**

This is called **canine** or **cuspid protected occlusion.** It is an occlusal relationship in which the vertical overlap of the maxillary and mandibular canines **produces a disclusion** *(separation)* **of all of the posterior teeth** when the mandible moves to either side. All other teeth, once they move from centric relation, **do not contact.** If there is contact of other teeth, it is termed a "working side" or "non-working side" interference depending on which side the mandible moves towards. **Note:** When placing a crown on a maxillary canine, if you change a canine protected occlusion to group function you **increase the potential** for a "non-working side" interference.

Group function *(sometimes called unilateral balanced occlusion)* is an occlusal relationship in which all posterior teeth on a side **contact evenly** as the jaw is moved toward that side *(working side).* All teeth on the non-working side are **free of any contact.** The **group function** of the teeth on the working side distributes the occlusal load. The **absence of contact** on the non-working side prevents those teeth from being subjected to the destructive, obliquely directed forces found in non-working interferences. It also saves the centric holding cusps, i.e., the mandibular buccal cusps and the maxillary cusps, from excessive wear. The obvious advantage is the **maintenance of the occlusion.**

Notes:

1. Some relationships are not conducive to cuspid protected occlusion such as Class II or end-to-end relationships.
2. Some relationships are not amenable to group function such as Class II, deep vertical overlap.
3. Regardless of what lateral concept is used, **no non-working side contacts are a must** because: **(1)** They are damaging **(2)** They are difficult to control due to mandibular flexure and **(3)** They deliver more force to the teeth than other contacts.

When **establishing a balanced occlusion,** the lingual cusps of maxillary posterior teeth on the **balancing side** should contact:

- The **central fossae** of mandibular posterior teeth
- The **lingual** inclines of **facial** cusps of mandibular posterior teeth
- The **lingual** inclines of **lingual** cusps of mandibular posterior teeth
- The **facial** inclines of **lingual** cusps of mandibular posterior teeth

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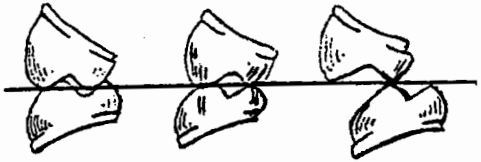
• **The lingual inclines of facial cusps of mandibular posterior teeth**

Remember:



Centric Occlusion Working Side Balancing Side

Notes on contacts in balancing occlusion *(posterior teeth):*

- Ideal Class I → **cusp-fossa** contact in centric occlusion
- Lateral excursion:
  - — Working side → contact of opposing cusps
  - — Balancing side *(non-working)* → contact of maxillary lingual cusps *(lingual inclines)* and mandibular facial cusps *(lingual inclines)*

In an ideal intercuspal position, the **facial cusp tips** of permanent maxillary premolars oppose:

- The facial embrasure **between** their class counterpart and the **tooth mesial to it**
- The facial embrasure **between** their class counterpart and the **tooth distal to it**
- The **opposing central fossae**
- The **opposing mesial marginal ridge**

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- **The facial embrasure between their class counterpart and the tooth distal to it**

Examples:

1. The facial cusp tip of a **maxillary first premolar** opposes the facial embrasure **between** the mandibular first and second premolars *(see note below).*
2. The facial cusp tip of a **maxillary second premolar** opposes the facial embrasure **between** the mandibular second premolar and mandibular first molar.

Note: During lateral excursive movements, the facial cusp ridge of the maxillary first premolar **on the working side** opposes the distal cusp ridge of the first premolar and the mesial cusp ridge of the second premolar.

In the intercuspal position, the **distobuccal cusp** of a permanent mandibular second molar occludes where?

- The interproximal marginal ridge area **between** the maxillary second bicuspid and first molar
- **Central fossa** of the maxillary **first molar**
- **Central fossa** of the maxillary **second molar**
- The interproximal marginal ridge area **between** the maxillary first molar and second molar

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- **Central fossa of the maxillary second molar**

Contacts in Intercuspal Position	
Mandibular Buccal Cusps	**Occludes into Area of Maxillary Teeth** *(Class counterpart or counterpart and tooth mesial to it)*
First premolar	Mesial triangular fossa of first premolar and distal marginal ridge of canine
Second premolar	Mesial triangular fossa of second premolar
First molar	
Mesiobuccal	Mesial marginal ridge of first molar and distal marginal ridge of second premolar
Distobuccal	Central fossa of first molar
Distal	Distal fossa of first molar
Second molar	
Mesiobuccal	Mesial marginal ridge of second molar and distal marginal ridge of first molar
Distobuccal	Central fossa of second molar

In a **retrusive movement,** the condyles of the mandible have moved in a:

- Backward and upward direction
- Downward and forward direction

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• **Backward and upward direction**

— A **protrusive movement** requires the condyles to move **downward and forward direction.**
— In **lateral movements,** the **working condyle** moves down, forward, and laterally.
— In **lateral movements,** the **non-working condyle** moves down, forward, and medially.

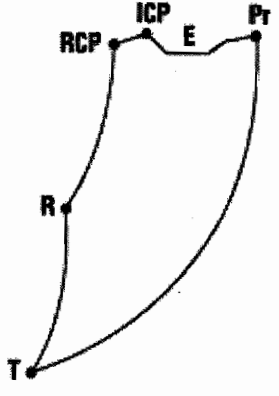
Remember: In **complete dentures,** the path of the condyle during free mandibular movements is **governed primarily** by the shape of the fossa and meniscus *(articular disc)* as well as the muscular influence.

The **inclination of the condylar path** during protrusive movement can vary from steep to shallow in different patients. It forms an average angle of about 30^{0} with the horizontal reference plane. If the protrusive inclination is steep, the cusp height may be obviously longer. Similarly, if the inclination is shallow, the cusp will be shorter. This factor is the **most important** aspect of condylar guidance that affects the selection of posterior teeth with appropriate cusp height.

Anterior guidance *(vertical and horizontal overlap of anterior teeth)* also affects the surface morphology of posterior teeth. The greater the overlap, the longer the cusp height.

In the diagram below of **Posselt's envelope** of mandibular motion *(sagittal section)*, which letter(s) designates **maximum opening?**

- Pr
- E
- T
- RCP
- ICP



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- **T** ***(maximal mandibular opening with full antero-inferior translation of the condylar heads)***

ICP = Intercuspal position *(IP)* or centric occlusion *(CO)*

RCP = retruded contact position or centric relation *(CR)*

PR = Maximum protrusion

E = Edge-to-edge position of incisors

PR-T = the anterior border movement of the mandible

RCP-R = the **rotational movement** of the condyles returning to centric relation *(terminal hinge axis opening)*

R-T = the **translational movement** of the mandible returning to where the condyles are in centric relation

Together these line segments *(RCP-R and R-T)* make up the posterior border movements of the mandible.

During **typical empty mouth swallowing,** the mandible is braced in which jaw position to allow for proper stabilization?

- Intercuspal position *(IP)*
- Retruded contact position *(RCP)*
- Protruded contact position *(PCP)*
- Centric relation *(CR)*

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• **Intercuspal position *(IP)***

***IP** is also called **centric occlusion.**

Remember: When the teeth are in centric occlusion, the position of the mandible in relation to the maxilla is **determined by** the intercuspation of the teeth.

Empty mouth swallowing occurs frequently throughout the day and is an important function that rids the mouth of saliva and helps to moisten the oral structures. The hourly rate of non-masticatory swallowing is apparently related to the amount of salivary flow and, in most instances, may be an involuntary reflex activity.

Notes:

1. The **masseter muscles** contract and the tip of the tongue touches the roof of the mouth during normal swallowing.
2. Tooth contacts are of **longer duration** in swallowing than in chewing, but there is wide variation in frequency and duration from one person to another.

When the mandible is in its **physiologic rest** or **postural position,** the contact of teeth is:

- Maximum
- Not present
- Premature
- Slight

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• **Not present**

This position **results** when the mandible and all of its supporting muscles *(eight muscles of mastication plus the supra - and infrahyoids)* are in their resting posture *(there is a relative muscular equilibrium).* The term used to describe this absence of contact is **"freeway space"** or **"interocclusal distance".** It usually averages between 2-6 mm. This position is a **"muscle-guided"** position. It is the beginning and end point of most mandibular movements.

The **retruded contact position** *(also called centric relation)* is a **"ligament-guided"** position. It is the closing end point of the retruded border movement *(the terminal hinge movement).*

The **intercuspal position** *(also called centric occlusion)* is a **"tooth-guided"** position. When the teeth are in centric occlusion, the position of the mandible in relation to the maxilla is **determined** by the intercuspation of the teeth.

The **protruded contact position** is symmetrical, and the underside of the meniscus *(articular disc)* moves distally relative to the superior surface of the mandibular condyle. The condyle moves forward and carries the disc with it.

Bite registration material used to make an accurate interocclusal record should have what important characteristic?

- Offer a **maximum resistance** to the patient's jaw closure and have **high flow** at mixing
- Offer a **maximum resistance** to the patient's jaw closure and have **low flow** at mixing
- Offer a **minimum resistance** to the patient's jaw closure and have **low flow** at mixing
- Offer a **minimum resistance** to the patient's jaw closure and have **high flow** at mixing

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- **Offer a minimum resistance to the patient's jaw closure and have low flow at mixing**

In recent years, **addition-reaction silicone impression materials** have dominated the **IOR** *(interocclusal record)* market. These materials have **very low flow** when mixed and become rigid after setting.

In the past, an **interocclusal record** was made by placing the IOR material into the mouth and closing the patient's jaws into the material at the desired relationship. Although this concept is acceptable — and may produce a relatively accurate IOR — the act of closing into any material, regardless of its lack of viscosity, often causes a deviation of the mandible away from the desired contact position.

A **more desired** method of obtaining an accurate interocclusal record is as follows:

- Close jaws into centric occlusion *(most interdigitated position)*.
- With teeth occluded tightly, inject the addition-reaction silicone material in between the maxillary and mandibular teeth. Inject material only into areas where teeth have been prepared and not around the entire arch.
- Advise patient to place tongue forward, short of going between teeth, thereby making a matrix for the lingual aspect of silicone.
- Let silicone set for about two minutes.
- Remove IOR and trim with a sharp knife.
- Mount casts with IOR material present only in areas of tooth preparations.

Which of the following materials available for recording centric relation when fabricating a removable partial denture is the **least** satisfactory?

- Modeling plastic
- Wax
- Quick-setting impression plaster
- Metallic oxide bite registration paste
- Silicone impression materials

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• **Wax**

Materials used to record jaw relationships have varied widely over the years. An ideal recording medium would be characterized as easy to handle, uniformly soft while the record is being made, rapid setting, and totally rigid but not brittle when set. Rapid setting plaster, **zinc oxide** and **eugenol pastes,** and modeling plastic all approach the ideal. **Avoid soft waxes** as a recording material. They never become rigid and are likely to be distorted during the cast mounting procedure.

If sufficient natural posterior occlusion exists, the mandibular cast may be mounted in centric occlusion using a zinc oxide-eugenol reinforced wax bite. **In the case of the distal extension partial denture,** base plates and occlusion rims **should be placed on the framework** and the patient closed into softened recording wax or **zinc oxide-eugenol paste** *(preferred).* Whether this record will be in centric occlusion or centric relation will depend upon the individual case and is **dictated** by the presence or absence of any natural posterior occlusion in the patient.

A **reliable method** is to use a record of all remaining occluding surfaces in a wax wafer with the mandible in the terminal hinge position and the teeth **just out** of occlusion.

Primary requirements for making a **centric relation record** when fabricating a removable denture:

- To record the correct horizontal relation of the mandible to the maxilla
- To stabilize the lower record base with equalized vertical pressure
- To retain the record in an undistorted condition until the casts have been accurately mounted on the articulator or until a previous record can be verified

The **lingual** cusps of the **maxillary** posterior teeth are:

- Non-supporting and working
- Supporting and balancing
- Supporting and working
- Non-supporting and balancing

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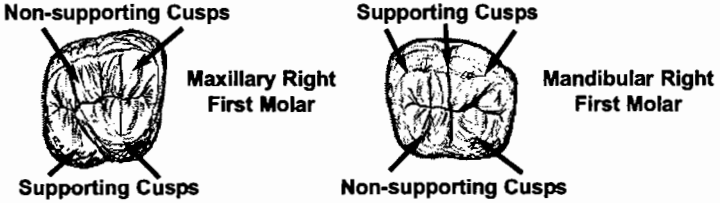
• **Supporting and working**

Five Common Characteristics of Supporting Cusps

1. They **contact** the opposing tooth in the intercuspal position.
2. They support the **vertical dimension** of the face.
3. They are **nearer the faciolingual center** of the tooth than the non-supporting cusps.
4. Their **outer incline** has a potential for contact.
5. They have **broader, more rounded cusp ridges** than non-supporting cusps.

Remember: The **supporting cusps** are the **maxillary lingual** and the **mandibular buccal.** These cusps do grinding work because they occlude in a fossa or marginal ridge and are also called **working** cusps. They are sometimes called **centric** cusps because they hold the occlusion in a middle position *(centric position)*.

The **non-supporting cusps** are the **maxillary buccal** and the **mandibular lingual.** These cusps do not occlude or fit into fossa or marginal ridge areas and are called **balancing or non-centric** cusps. These cusps allow the dentition to move apart, out of occlusion. They allow teeth to "unlock" and move back and forth and side to side.These cusps have **sharper cusp ridges** that serve to shear food as they pass close to the supporting cusp ridges during chewing strokes.



Anterior guidance is the result of:

- Horizontal overlap
- Vertical overlap
- Horizontal and vertical overlap

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• **Horizontal and vertical overlap**

Anterior guidance *(sometimes called anterior coupling)* is a tightly overlapping relationship of the opposing maxillary and mandibular incisors and canines, **which produces disclusion** of the posterior teeth when the mandible protrudes and moves to either side.

Anterior teeth have a mechanical advantage over posterior teeth, due to the fact that they are farther away from the fulcrum *(condyles)*, giving them better leverage to offset the closing musculature. This apparent is apparent when one tries to occlude maximally with anterior teeth as opposed to occluding maximally in the molar region. The further away from the site of muscle action, the less force is exerted.

Important point of all this → if anterior guidance **can be accomplished,** the least amount of force will be placed on the teeth during muscular contraction.

Incisal guidance is a measure of the amount of movement and the angle at which the lower incisors and mandible must move from the overlapping position of centric occlusion to an edge-to-edge relationship with the maxillary incisors.

It is the **second** end-controlling factor in articulator movement. It is, to some degree, under the **control of the dentist.** Influencing factors include: **1)** esthetics, **2)** phonetics, **3)** ridge relations, **4)** arch space, and **5)** inter-ridge space. **Esthetics** and **phonetics** are the primary factors limiting the dentists control of incisal guidance. The incisal guidance on the articulator is the mechanical equivalent of horizontal and vertical overlap.
Note: The right and left condylar mechanisms are the other end-controlling factors in articulator movement.

All of the following concepts related to occlusion are true **except:**

- Optimum occlusion requires minimum adaptation by the patient
- Bilateral balanced occlusion dictates that a minimum number of teeth should contact during mandibular excursive movements
- Unilateral balanced occlusion or "group function" calls for all teeth on the working side to be in contact during a lateral excursion
- Mutually protected occlusion, also called "canine guided" or "organic" occlusion is the one in which anterior teeth protect posterior teeth in all mandibular excursions
- Mutually protected occlusion is the most widely accepted arrangement of occlusion

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- **Bilateral balanced occlusion dictates that a minimum number of teeth should contact during mandibular excursive movements**

Important: In bilateral balanced occlusion the **maximum number** of teeth should contact during mandibular excursions. This concept of occlusal arrangement, though ideal, is very difficult to achieve

The **determinants of occlusion** include the right and left TMJ, the occlusal surfaces of the teeth and the neuromuscular system. The concepts of occlusal arrangement aim to place the artificial teeth in harmony with the TMJ and the neuromuscular system. If this is done properly, it will result in minimum stress on the teeth and only a minimum effort will need to be expended by the neuromuscular system when performing mandibular movements.

There are **four features** of the human dentition which **directly affect** the health of the PDL and its hard tissue anchorage in terms of resisting occlusal force:

1. **Anterior teeth** have slight or no contact in the intercuspal position.
2. The **occlusal table** is less than sixty percent of the overall faciolingual width of the tooth.
3. The **occlusal table** of the tooth is generally at right angles to the long axis of the tooth.
4. **Crowns of mandibular molars** are inclined about 15-20° toward the lingual.

All of the following are the **theoretical determinants** needed for restoring a complete and functional occlusal surface of a tooth **except:**

- The amount of vertical overlap of the anterior teeth
- The contour of the articular eminence
- The height of the pulp horn of that particular tooth
- The amount and direction of lateral shift in the working side condyle
- The position of the tooth in the arch

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- **The height of the pulp horn of that particular tooth**

The **four theoretical determinants** needed for restoring a complete and functional occlusal surface of a tooth are:

1. The amount of **vertical overlap** of the anterior teeth
2. The contour of the **articular eminence**
3. The amount and direction of lateral shift in the **working side condyle**
4. The position of the **tooth** in the arch

However, the jaw relationship most frequently used in the **actual design** of restorations is the **acquired** centric occlusion.

Note: The **anterior determinant of occlusion** is the horizontal and vertical overlap relationship of anterior teeth.

All of the following are considered to be the **basic principles** for occlusal adjustment, **except:**

- The **maximum distribution** of occlusal stresses in centric relation
- The forces of occlusion should be borne as much as possible by the **long axis** of the teeth
- When there is point-to-surface contact of flat cusps, it should be changed to a **surface-to-surface contact**
- Once centric occlusion is established, **never take** the teeth out of centric occlusion

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- **When there is point-to-surface contact of flat cusps, it should be changed to a surface-to-surface contact**

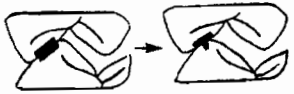
***This is **false;** when there is **surface-to-surface** contact of flat cusps, it should be changed to a **point-to-surface** contact.

<u>The basic principles for occlusal adjustment include:</u>

- **Maximum distribution** of occlusal stresses in centric relation.
- **Forces of occlusion** should be borne as much as possible by the long axis of the teeth *(Picture A)*.
- When there is **surface-to-surface** contact of flat cusps, it should be changed to a **point-to-surface** contact *(Picture B)*.
- **Once centric occlusion** is established, never take teeth out of centric occlusion.

A

B



surface to surface point to surface

In an ideal intercuspal position, the **mesiobuccal cusp** of the permanent **maxillary second molar** opposes:

- The **distobuccal groove** of the mandibular **first molar**
- The **buccal groove** of the mandibular **second molar**
- The **mesiobuccal groove** of the mandibular **second molar**
- The **developmental groove** between the distobuccal and the distal cusps of the mandibular **first molar**

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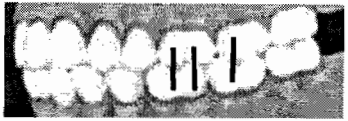
• **The buccal groove of the mandibular second molar**



Important:

- The **mesiobuccal cusp** of the maxillary first molar opposes the **mesiobuccal groove** of the mandibular first molar. This relationship is a **key** factor in the definition of Class I occlusion.
- The **distobuccal cusp** of the maxillary first molar opposes the **distobuccal groove** of the mandibular first molar. **Note:** This **distobuccal groove** also serves as an escapeway for the **ML cusp** of the maxillary first molar during non-working excursive movements.
- The **oblique ridge** of the maxillary first molar opposes the **developmental groove** between the **distobuccal** and **distal cusps** of the mandibular first molar.

Remember: The **maxillary buccal** *(facial)* and the **mandibular lingual** cusps are **guiding cusps.** The inner occlusal inclines leading to these cusps are called **guiding inclines** because in contact movements they guide the supporting cusps away from the midline. Thus, there are the **bucco-occlusal** inclines *(lingual inclines of the buccal cusps)* of the maxillary posterior teeth and the **linguo-occlusal** inclines *(buccal inclines of the lingual cusps)* of the mandibular posterior teeth.

In the **early stages of lateral movements,** the condyle appears to rotate with a slight lateral shift in the direction of the movement. This movement is called the **Bennett movement.** This Bennett movement **refers** to the:

- Non-working side condyle only
- Working side condyle only
- Both the non-working and working side condyles
- Neither of the condyles

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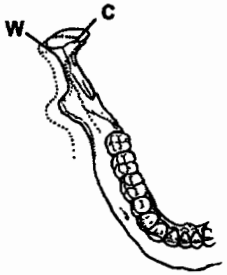
• **Working side condyle only**

In a lateral movement, the **non-working** side condyle moves downward, forward, and medially. The **working** side condyle moves laterally. Since the mandible is a solid bone, the amount that the non-working condyle moves medially determines how far the working side condyle moves laterally. The Bennett movement is sometimes called the **lateral shift** of the mandible or **immediate side shift.** **Important:** This movement influences the lingual concavity of the maxillary anterior teeth and directional placement of the ridges and grooves on the mandibular posterior teeth as well as the mesiodistal position of the cusps of posterior teeth. **Note:** The **Bennett angle** is the angle formed by the sagittal plane and the path of the non-working condyle during lateral movement of the mandible, as viewed in the horizontal plane.



Right Mandibular Movement

On the right side *(working side)*, the condyle moves from C *(centric)* to right working *(W)*. This movement is the **Bennett movement.**

When posterior teeth are in a **normal ideal relationship,** which of the following cusps are considered to be **guiding cusps?**

- Maxillary lingual cusps
- Maxillary buccal cusps
- Mandibular lingual cusps
- Mandibular buccal cusps

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- **Maxillary buccal cusps**
- **Mandibular lingual cusps**

These cusps are also called **balancing, non-supporting, non-centric or shearing** cusps. These cusps do not occlude or fit into fossae or marginal ridge areas on the opposite arch. They allow the dentition to move apart, out of occlusion. They allow the teeth to "unlock" and move back and forth and side to side.

Supporting cusps are the maxillary lingual cusps and the mandibular buccal cusps. These cusps are also called **working, stamp** or **centric cusps. Centric stops** are areas of contact that a supporting cusp makes with opposing teeth. For example, the mesial lingual cusp of the maxillary fist molar *(a supporting cusp)* makes contact with the central fossa *(centric stop)* of the mandibular first molar.

Supporting cusps contact the opposing teeth in their corresponding faciolingual center on a marginal ridge or a fossa. **Non-supporting cusps** overlap the opposing tooth without contacting the tooth.

Note: In **posterior cross-bite situations,** the supporting and guiding cusps are **opposite.** The maxillary buccal and the mandibular lingual would be supporting and the maxillary lingual and the mandibular buccal would be guiding.

The "Glossary of Prosthodontic Terms" defines **balanced occlusion** as:

- An occlusion of the teeth which presents a harmonious relation of the occluding surfaces in **centric only** within the functional range
- An occlusion of the teeth which presents a harmonious relation of the occluding surfaces in **eccentric positions only** within the functional range
- An occlusion of the teeth which presents a harmonious relation of the occluding surfaces in **centric and eccentric positions** within the functional range

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- **An occlusion of the teeth which presents a harmonious relation of the occluding surfaces in centric and eccentric positions within the functional range**

Five significant factors that govern the establishment of balanced articulation are:

- Inclination of the condylar guidance → totally dictated by the patient
- Inclination of the incisal guidance → horizontal and vertical overlap
- Inclination of the occlusal plane → plane of orientation
- Convexities of the compensating curve
- Angle and height of the cusps

} Some of these, if not all, are controlled by the dentist.

Recommended occlusion for complete dentures: Recommended practice is to develop maximum intercuspation of complete dentures to coincide with CR at an acceptable OVD. Failure to achieve that can lead to intolerance, usually because of instability of the dentures or because of pain of the alveolar mucosa as a result of uneven load distribution and high stress concentrations. It is also recommended that a **balanced occlusion** is provided in order to help give occlusal stability.

Balanced centric occlusion in partial dentures is necessary for the **stability** of the appliance. Design of the framework and the relationship of the teeth to the ridges also influences the stability of the partial. **Bilateral eccentric balance** is not an objective in **partial denture construction unless** the partial prosthesis is opposed by a complete denture. The vertical relation for RPD's is usually determined by the remaining natural teeth *(unlike complete dentures)*.

Notes:

1. **Eccentric occlusion** is defined as protrusive and right and left lateral contacts of the inclined planes of the teeth when the jaw **is not** moving.
2. **Articulation** can be defined as the relationship of teeth during movements into and away from eccentric position while the teeth are in contact.

Reducing occlusal interferences *(selective grinding)* should usually be done:

- **After** a fixed bridge or a partial denture is delivered to a patient
- **Before** constructing a fixed bridge or a partial denture for a patient
- After a fixed bridge **but** before a partial denture is delivered to a patient
- After a partial denture **but** before a fixed bridge is delivered to a patient

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• **Before constructing a fixed bridge or a partial denture for a patient**

***This is done to **prevent** duplicating the deflective occlusal contacts in the final restoration.

Note: One common case in which it would be preferable for selective grinding to be completed after the fixed bridge or partial denture is in place is when a fixed or removable partial denture is to be constructed for a space over which the opposing tooth has extruded slightly. The bridge or partial is frequently constructed to the ideal plane of occlusion and the opposing tooth is adjusted after insertion.

The **most common complaint** after cementation of a fixed bridge is sensitivity to hot / cold and **is an indication** of a deflective occlusal contact. The teeth involved may also be sensitive to touch and this may be noticed by the patient while brushing. In these cases, an immediate correction of the occlusion must be made.

The **purpose of selective grinding** is to remove all interferences **without destroying cusp height.** With such an objective in mind, whenever interferences exist **in centric but not** in lateral excursions, the fossa or marginal ridge opposing the premature cusp is deepened. It is important that whenever a prematurity is found, the occlusion be checked in all centric positions before any adjustment is performed. If cusps are found to interfere with one another in excursions, then only the non-holding cusps should be ground to prevent a decrease in vertical dimension.

Important: If you plan on **changing a patient's vertical dimension through crowns,** it is critical to mount a patient's casts **on the true hinge axis** *(use a face bow).*

Prolonged sensitivity to heat, cold, and pressure **after cementation** of a crown or a fixed bridge is usually related to:

- Recurrent decay
- A periodontal problem
- Occlusal trauma
- An open margin

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• **Occlusal trauma**

If centric relation **occlusion is high,** patients will complain of cold sensitivity and pain upon biting down hard. All patients should have an appointment specifically to check the occlusion on all crowns and bridges. Excursive movements should also be evaluated. Many times patients will complain of pain on chewing soft foods, which indicates improper balancing or working contacts. **The occlusion of gold restorations is best checked with silver plastic shim stock.**

Important: Initial sensitivity can be caused by acid irritation accentuated by dehydrated dentin from prolonged drying of the tooth **before** cementation or incorrect liquid / powder ratio of the cement.

Radiographic signs of occlusal trauma include hypercementosis, root resorption, alteration of the lamina dura, and alteration of the periodontal space.

Note: If a **marginal ridge** is left higher than an adjacent marginal ridge, an interference in **retrusive** movement may occur.

In a **posterior** fixed bridge, all of the following are true **except**:

- A pontic **should be** in contact in centric occlusion
- A pontic **may or may not** be in contact in working-side movements
- A pontic **should be** in contact in non-working side movements

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• **A pontic should be in contact in non-working side movements**

***This is **false;** a pontic **should not** be in contact in non-working movements.

Remember: The **success or failure** of a bridge depends mostly on the **design** of the pontic. The design is dictated by function, esthetics, ease of cleaning, patient comfort, and the maintenance by the patient of healthy tissue on the edentulous ridge.

Proper design is more important to cleanability and acceptable tissue well-being than is the choice of materials *(porcelain, gold, etc.)*.

Note: Multiple adjacent pontics on an anterior fixed bridge have reduced facial embrasures to enhance esthetics.

Condylar guidance is a factor which:

- Is totally controlled by the dentist
- Is totally dictated by the patient
- Is partially dictated by the patient but can be adjusted by the dentist if necessary
- Can be adjusted by the laboratory technician

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- **Is totally dictated by the patient**

Condylar guidance is the mechanical device on an articulator which is intended to produce similar guidances in articulator movement **that are produced** by the paths of the condyles in mandibular movements.

It is **important** to realize that condylar guidance is a factor which is **totally dictated by the patient.** It cannot be varied or "adjusted" by the dentist. The inclination of condylar guidance depends on: 1) the **shape and size** of the bony contour of the TMJ *(fossae and disc)*, 2) the **action of the muscles** attached to the mandible, 3) the limiting effects of the **ligaments**, and 4) the **method used** for registration.

Remember:
- The **incline** or **angulation** of the condylar element on the articulator is anatomically related to the slope of the condylar articular eminences *(condylar inclination).*
- When **adjusting** the condylar guidance for protrusive relationship, the incisal guide pin on the articular should be raised out of contact with the incisal guide table. The protrusive record is probably the **least reproducible** maxillomandibular record.

Note: When restoring the entire mouth with crowns, the **protrusive condylar path inclination** influences the mesial inclines of the mandibular cusps and the distal inclines of the maxillary cusps.

Decreased vertical dimension of occlusion refers to an occluding vertical dimension that results in:

- A **loss of interocclusal distance** when the mandible is in the rest position *(decreased freeway space)*
- An **excessive interocclusal distance** when the mandible is in the rest position *(increased freeway space)*
- Neither of the above, vertical dimension of occlusion **does not affect** interocclusal distance

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- **An excessive interocclusal distance when the mandible is in the rest position *(increased freeway space)***

A classic example of a decreased vertical dimension → People with no teeth or people who have worn dentures for a long time present with the lower portion of the face scrunched up or do not show their lips anymore *(poor facial profile)*.

Solution: Make new dentures and **increase** the vertical dimension of occlusion. By doing this, you will **decrease** the interocclusal distance and **decrease** freeway space.

Correct vertical dimension of occlusion is **evaluated** using the following methods:

1. Overall appearance of facial support.
2. Visual observation of space between the occlusal rims at rest.
3. Measurements between dots on the face *(which were placed for this reason)*, when the jaws are at rest and when the occlusal rims are in contact.
4. Observations when the "s" sound is enunciated accurately and repeatedly. This ensures adequate speaking space between the occlusal rims/occlusal plane.

Centric relation *(CR)* is a:

- "Muscle-guided" position
- "Ligament-guided" position
- "Tooth-guided" position

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• **"Ligament-guided" position**

Three Basic Jaw Positions

1) **Centric occlusion** *(CO)* or the intercuspal *(IC)* position is the relationship between maxillary and mandibular occlusal surfaces that provides the maximum contact and or intercuspation. It is a **"tooth-guided"** position.

2) **Centric relation** *(CR) (also called the retruded contact position)* is the most unstrained, retruded anatomic and functional position of the heads of the condyles of the mandible in the mandibular *(glenoid)* fossae of the temporomandibular joints. This is a relationship of the bones of the upper and lower jaws **without** tooth contact. The presence or absence of teeth, or the type of occlusion or malocclusion, **are not factors.** It is a **"ligament-guided"** position. **Note:** The mandible cannot be forced into centric relation from the rest position because the patient's reflex neuromuscular defense would resist the applied force. The mandible should be relaxed and gently guided into centric relation.

3) The **rest position of the mandible** or the postural position **is determined mostly by the musculature.** The usual reflex cited as the basis for the postural position of the mandible is the **tonic stretch reflex** of the mandibular levators *(i.e., the myotatic reflex)*. It is a **"muscle guided"** position.

All of the following statements concerning the **functionally generated pathway technique** are true **except:**

- A prerequisite for the use of this technique for the restoration of a single tooth is the presence of a Class III occlusion
- This technique allows the cuspal movements of the dentition to be recorded in wax intra-orally and transferred to the articulator in the form of a static plaster cast
- This static plaster cast is also called the functional index
- By registering the pathways of the opposing tooth surfaces during mandibular movements, the technique allows a laboratory technician to provide a restoration with an occlusal surface less likely to incorporate occlusal interferences

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- **A prerequisite for the use of this technique for the restoration of a single tooth is the presence of a Class III occlusion**

***This is **false;** a prerequisite for the use of this technique for the restoration of a single tooth is the presence of an **optimal occlusion.**

A **major difficulty** for any dental laboratory technician is to determine the cuspal movements of the dentition using hand-held casts or casts mounted on a simple hinge articulator. The functionally generated pathway technique allows these movements to be recorded in wax intra-orally and transferred to the articulator in the form of a static plaster cast *(the functional index).*

The involved tooth should be immobile and the recording material *(low-fusing hi-fi wax)* retained on the prepared tooth, not moving separately, during the generation of the FGP wax record. The involved tooth should have unprepared teeth anterior and posterior to it to act as **reference** surfaces for checking the complete seating of the functional core of the working cast. There should be **no occlusal interferences pre-operatively** and the opposing surfaces should be properly restored.

Notes:

1. The functional index becomes a **static registration** of all the movements of the opposing cusps.
2. The important consideration in generating this functional pathway is that all motion of the mandible **must be directed** from an eccentric to a centric position, never the reverse.
3. **Full case articulation** is more universally applicable in obtaining occlusal relationships for the fabrication of a wax pattern.

There are four types of gypsum products approved for use in dentistry. Which of the following types is used **rarely** today?

- Type I
- Type II
- Type III
- Type IV

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- **Type I**

Classification of Dental Gypsum Products		
	ADA Specification	**Traditional Terminology**
Type I	Plaster, impression	Impression plaster
Type II	Plaster, model	Model or lab plaster
Type III	Dental stone	Class I stone
Type IV	Dental stone, high strength	Class II stone or improved stone *(die stone)*

Uses of dental stone and plaster:

- **Type I** → rarely used today.
- **Type II** → used for making casts when strength is not important *(ortho)*.
- **Type III** → are preferred for preparing casts upon which dentures are to be processed.
- **Type IV** → used when making stone **"dies,"** which are generally reproductions of teeth with prepared cavities, used for crown and bridge and operative *(inlays and onlays)*.

In mixing dental stone, why should the powder be sprinkled onto the water in the bowl?

- The addition of powder prevents the mix from becoming exothermic
- This is not recommended; the water should be added to the powder
- This process results in better powder mixing and reduced chance for air bubbles
- The powder is added to the water to avoid using more than one bowl

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- **This process results in better powder mixing and reduced chance for air bubbles**

Dental gypsum products are made up of hemihydrate particles whose **size, shape,** and porosity **differ** for each material. These gypsum-based powders require **different amounts of water** for mixing because the different particle shapes produce different packing efficiencies that affect the amount of excess water required for making a suitable mixture.

Dental Plaster and Stone

Mixing:

- **Water / powder ratio** → The water / powder ratio is an important factor in determining physical properties. When a **high proportion of water is used,** the powder particles are farther apart. This results in **less expansion** with a retarded setting time and a weaker product. Dental plasters generally require about twice as much water compared to stones. Plaster has a **higher setting expansion** than does stone.
- **Water temperature** → Generally, the **colder** the water, the **longer** the setting time.
- **Spatulation** → Rapid spatulation for a time equal to normal hand mixing for 1 minute accelerates setting time and produces greatest strength. Do not spatulate to the point where the mixture starts to harden. This will produce a cast that is much weaker.
- **Accelerators and retarders** *(modifiers)*:
  - Accelerators commonly used are **potassium sulfate** and sodium chloride. Alum can also be used.
  - Borax and **sodium citrate** are retarders.

Dental stone and **dental plaster** both have a slightly different type of principal component *(calcium sulfate hemihydrate).* **Dental plaster** has:

- Beta-hemihydrate and dental stone has gamma-hemihydrate
- Alpha-hemihydrate and dental stone has beta-hemihydrate
- Gamma-hemihydrate and dental stone has beta-hemihydrate
- Beta-hemihydrate and dental stone has alpha-hemihydrate

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• **Beta-hemihydrate and dental stone has alpha-hemihydrate**

The **principal constituent** of the dental plasters and stones is the **calcium sulfate hemihydrate.** Depending upon the method of calcination, different forms of the hemihydrate can be obtained → either alpha or beta hemihydrate. The **beta-hemihydrate** is more popularly known as **plaster of Paris,** and these crystals are characterized by their sponginess and irregular shape in contrast to the **alpha-hemihydrate** *(stone)* **crystals,** which are more dense and have a prismatic shape. When the alpha-hemihydrate is mixed with water, the product obtained *(dental stone or die stone)* is **much stronger and harder** than that resulting from beta-hemihydrate *(plaster).* The chief reason for this difference is that the alpha-hemihydrate powder *(stone)* requires **much less** gauging water when it is mixed than does the beta-hemihydrate. The beta-hemihydrate *(plaster)* requires more water to float its powder particles so that they can be stirred, because the crystals are more irregular in shape and are porous in character.

Note: All gypsum products that are reacted with water form **calcium sulfate dihydrate** as a reaction product.

Dental plaster and stone are **vibrated after mixing** to:

- Minimize distortion
- Reduce setting time
- Eliminate air bubbles
- Increase the setting time

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- **Eliminate air bubbles**

Using a **vibrator** when pouring models helps to eliminate air bubbles *(trapped air)*. This produces a more accurate, usable model. Another way of preventing entrapment of air is to place the proper amount of water in the mixing bowl first and then sift the model plaster or stone into the bowl. When mixing dental plaster or stone, any of the following will cause the gypsum product to **set faster** → **increased** spatulation, a **lower** water-powder ratio, and using a **mixture** of water and ground-up set gypsum particles to mix with the plaster or stone.

Once the impression is **poured,** it should be allowed to harden for **45 minutes to 1 hour** *(or until cool to the touch)* before removing the cast from the impression.

If **nodules of stone** appear in the occlusal pits of a stone cast, it is most likely due to the entrapment of air during the insertion and seating of the tray.

Note: All types of gypsum products **are weaker** in tensile strength than compressive strength.

Dental plaster is produced by:

- Heating gypsum in an open vessel at 150 –160°C
- Heating gypsum under steam pressure in an autoclave at 120 –150°C
- By boiling gypsum in a 30% aqueous solution of calcium chloride and magnesium chloride

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• **Heating gypsum in an open vessel at 150 – 160°C**

***This process produces particles that are **porous and irregularly** shaped. **Note:** It is the **weakest** gypsum product.

Heating gypsum under steam pressure in an autoclave at 120 -150°C produces **dental stone.** This process produces particles that are **uniformly shaped and less porous.**

Boiling gypsum in a 30% aqueous solution of calcium chloride and magnesium chloride produces **high strength** *(improved)* **die stone**. This process produces the **least porous and strongest** particles.

All gypsum products come from the mineral gypsum, which is the **dihydrate form of calcium sulfate.** During heating, *(the manufacturing process)*, water is lost and gypsum is converted to the **hemihydrate** form of calcium sulfate *(powder)*. When water is added to the powder, a chemical reaction takes place and the **hemihydrate** is converted back to the **dihydrate** form of calcium sulfate.

Notes:

1. When mixing gypsum products **always sprinkle** the powder into the water. This results in better powder mixing and reduces the chance for air bubbles.
2. When gypsum products are mixed with water, heat is given off. This is called an **exothermic reaction.**
3. Exposure of a stone cast to tap water should be **minimized** because eroding of the cast will result.

An edentulous patient has **slight undercuts on both tuberosities** and also on the facial of the anterior maxilla. To construct a satisfactory maxillary complete denture, you should **reduce** which of the following?

- All undercuts
- The anterior undercut only
- Both tuberosity undercuts
- None of them

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• **Both tuberosity undercuts**

Undercut tuberosities will interfere with the seating of the denture.

Explanation of answer: Maxillary anterior undercuts are very common and present no special problems **unless accompanied** by large bilateral posterior undercuts. Even this situation can usually be managed by reducing the inner surface of the denture lateral to the tuberosities.

The **maxillary sinus** appears to enlarge throughout life if it is not restricted by natural teeth or dentures. As the sinus enlarges, the tuberosity moves downward. If there is no contact with the retromolar pad at the vertical dimension of occlusion, the tuberosity must be reduced.

If a **low tuberosity** is not removed before constructing new dentures *(C/C)*, an accidentally underextended mandibular denture will probably be made and limited space to position posterior teeth will occur.

A **submucosal vestibuloplasty** is usually performed on the maxillary arch to improve the available denture base area. This procedure is favored because no raw tissue surface remains to granulate and re-epithelialize.

Rotational movements take place in which compartment of the **TMJ?**

- Upper *(mandibular fossa - articular disc)* compartment
- Lower *(condyle - articular disc)*
- Both the upper and lower compartments

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• **Lower *(condyle - articular disc)* compartment**

The temporomandibular joints are considered to be the most complex joints in the human body because they must provide for rotational movements, sliding movements *(translatory motion)* and an infinite range of combined movements and functions, unlike any other joint in the body.

When the mouth opens, two distinct motions occur at the joint. The first motion is **rotation** around a horizontal axis through the condylar heads. The second motion is **translation.** The condyle and meniscus move together anteriorly beneath the articular eminence.

In the **lower** *(condyle - articular disc)* **compartment,** only a **hinge-type or rotary motion can occur.** This rotational or terminal hinge-axis opening of the mandible is possible only when the mandible is retruded in **centric relation** with a conscious effort by the patient or by the dentist's control. **Note:** A pure hinging movement is possible **only in** the terminal hinge position.

In the **upper** *(mandibular fossa - articular disc)* **compartment,** only **sliding movements or translatory motion can occur.** When the lateral pterygoid muscles contract simultaneously, the discs and condyles slide forward down over the articular eminence ***(protrusion),*** or can move backwards together ***(retrusion)*** during opening and closing of the mouth, respectively.

Remember: The TMJ is a **ginglymoarthrodial joint** *(meaning that it glides and rotates),* permitting both **hinge-like rotation and sliding** *(gliding)* movements. Ginglymus means rotation, and arthrodial means freely movable.

An articulator which has the **condylar elements** on the lower member of the articulator and the **condylar path elements** on the upper member is called the:

- Arcon articulator
- Non-arcon articulator

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• **Arcon articulator**

***The **non-arcon** articulator has the **reverse** sequence with the condylar elements on the upper member of the articulator.

***The **angle** between the condylar inclination and the occlusal plane is **fixed** on the arcon articulator. This is **not so** with the non-arcon articulator.

- **Arcon** (**ar**ticulated **con**dyle) articulator → this type of articulator is commonly used for:
  - **Diagnostic mounting of study casts** to allow examination of occlusal contacts in the retruded contact position and analysis of tooth contacts during excursive movements of the mounted models.
    Note: Occlusal records in right and left lateral excursions **are necessary** for setting both the medial and superior condylar guides.
  - The fabrication of **cast and porcelain restorations,** thus ensuring correct tooth contacts in centric as well as lateral and excursive movements.

- **Non-arcon** ***(non-articulated condyle)*** articulator → this design is more popular for the **fabrication of dentures.** The following clinical records are required to mount the casts and set the articulator elements.
  - An occlusal record in the **retruded contact** position is necessary so that the lower cast can be correctly related to the upper for mounting on the lower component of the articulator.
  - Occlusal records in **protrusion and right and left lateral excursions** are required in order to set the sagittal condylar guidance angles.

You are ready to place **packing cord** around a tooth that was prepared for a crown on a patient with **hypertension.** It is recommended to use a cord impregnated with:

- Epinephrine
- Alum *(aluminum potassium sulfate)*
- Zinc chloride
- Any of the above

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• **Alum** ***(aluminum potassium sulfate)***

Epinephrine causes **local vasoconstriction,** which in turn results in transitory gingival shrinkage. Epinephrine impregnated cord has been shown to produce minimal physiologic changes when placed in an intact gingival sulcus. **However,** there is evidence of increased heart rate and elevated blood pressure when the cord is applied to the severely lacerated gingival sulcus. For those patients with medical conditions such as certain types of cardiovascular disease or hyperthyroidism, or a known hypersensitivity to epinephrine, a cord impregnated with **alum** should be substituted.

Note: Zinc chloride is caustic and causes delayed healing *(causes necrosis of the sulcular epithelium and the adjacent layer of connective tissue*). Therefore, it **should not** be used in impregnated cord.

Tissue retraction **is necessary** to:

- Control bleeding
- Retract the gingival tissues slightly away from the margins
- Allow impression material to flow into the sulcus
- Expose all gingival margins

A small hook-like projection of bone that extends from the medial pterygoid plate of the sphenoid bone is called the:

- Hamulus
- Hamular notch
- Maxillary tuberosity
- Fovea palatini

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• **Hamulus** → also known as the hamular process

***It is a thin, curved process that serves as the superior attachment of the **pterygomandibular raphe.** This raphe is a tendon between the buccinator and superior constrictor muscles.

The **hamular notch** is a thin cleft between the maxillary tuberosity and the hamulus. The vibrating line is an imaginary line drawn across the palate that marks the beginning of motion in the soft palate when an individual says "ah". It extends from one hamular notch to the other. At the midline, it usually passes about 2 mm in front of the fovea palatinae.

Remember: The distal end of the maxillary denture **must** cover the tuberosities and extend into the hamular notches. Overextension at the hamular notches will not be tolerated because of pressure on the pterygoid hamulus and interferences with the pterygomandibular raphe. When the mouth is opened wide, the pterygomandibular raphe is pulled forward. If the denture extends too far into the hamular notch, the mucous membrane covering the raphe will be traumatized.

The **fovea palatinae** are indentations near the midline of the palate formed by a coalescence of several mucous gland ducts. They are always in soft tissue, which makes them an ideal guide for the location of the posterior border of the denture.

The **torus palatinus** is a hard bony enlargement that occurs in the midline of the roof of the mouth and is found in about:

- 2% of the population
- 20% of the population
- 50% of the population
- 75% of the population

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• **20% of the population**

Palatal tori are bony enlargements located at the midline of the hard palate. They occur in approximately 20% of the population and are more prevalent in women than men. They usually reach maximum size in the third or fourth decade. Because the torus is usually covered by thinner and less resilient mucosa than the residual ridge, it may act as a fulcrum and cause rocking of the maxillary denture.

Because the soft tissues over the torus are generally thin and have a poor blood supply, post-operative healing is slow. It is best to cover the operated site with a surgical stent lined with a sedative dressing. If a patient is having all of their maxillary teeth out at one time, it is best to also remove the tori at the same time.

Note: Palatal tori **are usually not removed** for denture fabrication whereas mandibular tori are usually removed prior to denture fabrication. The following conditions warrant removal of palatal tori, if it → **(1)** impinges on the soft palate **(2)** is so large that it fills the vault and prevents the formation of an adequate denture base **(3)** is undercut **(4)** extends so far posteriorly interfering with the posterior palatal seal **(5)** is psychologically disturbing to the patient *(cancerphobia).*

Which of the following is the **most important reason** for treatment of hyperplastic tissue **before** construction of a complete or removable partial denture?

- It will make the patient feel better
- It will make the face-bow transfer easier to perform
- To provide a firm, stable base for the denture
- The final impression material will flow better

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- **To provide a firm, stable base for the denture**

Treatment may include:
- Tissue rest
- Soft reline of existing dentures
- Change in denture habits *(not wearing them 24 hours a day)*
- Surgical removal of tissue *(if tissues changes are extensive)*

Note: Mandibular tori, sharp prominent mylohyoid ridges, and epulis fissuratum should also be evaluated for surgical removal before the fabrication of new dentures is begun.

Current concepts of **impression making** for complete dentures recommend using a technique that:
- Affords placement and control of the impression material in recording border tissues *(border molding).*
- Results in minimal displacement of tissues under the denture *(registers the tissue in its passive position).*
- Is dependent on the oral conditions present.

Note: The **best impression technique** for a patient with loose hyperplastic tissue is to register the tissue in its **passive position.** There must be intimate contact of the impression material with the tissue.

When **inflammatory papillary hyperplasia** is seen on the palate of a patient wearing a maxillary complete denture, the condition is **most likely** going to be associated with:

- A vitamin B deficiency
- A sudden increase in body weight
- A hypersensitivity of the patient to the acrylic denture base
- Ill-fitting dentures and a poor state of oral hygiene

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- **Ill-fitting dentures and a poor state of oral hygiene**

The **hyperplasia** is produced in response to irritation from movement of the denture and from accumulating food debris. The masses present as **painless, firm, pink, or red nodular proliferations** of the mucosa. Candida albicans may contribute to the inflammation.

Most patients are **unaware of its presence.** It usually involves only the **hard palate** but can occasionally involve the residual ridges. Treatment of IPH depends on the size of the lesions. Although the nodules are not completely reversible, smaller papilla will usually regress somewhat with treatment *(removal of the denture, soft relines, good oral hygiene, and nystatin therapy).*

Remember: Denture-induced fibrous hyperplasia *(also called epulis fissuratum because of the clefts found in the hyperplastic tissue)* is also related to the chronic trauma produced by an ill-fitting denture. It occurs in the vestibular mucosa where the denture flange contacts tissue. It appears as **painless folds** of fibrous tissue surrounding the overextended denture flange. If the amount of hyperplasia is minimal, **tissue conditioning,** the fabrication of new dentures, and a change in denture habits may be sufficient to arrest tissue changes. However, surgical excision is usually required.

The **most common** cause of dry mouth *(xerostomia)* is:

- Aging
- Alcoholism
- Vitamin A or Vitamin B deficiency
- The use of drugs to manage chronic diseases
- Diabetes

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• **The use of drugs to manage chronic diseases**

Xerostomia is a possible side effect associated with more than 400 drugs including antihypertensives, antidepressants, antihistamines, bronchodilators, anticholinergics, and sedatives. Mouthwashes, alcohol, tobacco, and caffeine may alter salivary flow or cause dryness of the oral mucosa.

Even though **xerostomia** is not a disease, it can be a symptom of certain diseases. It can cause health problems by affecting nutrition as well as psychological health. It can contribute to and increase the chances of having tooth decay and mouth infections.

Temporary relief may be found from several sources:
• Saliva substitutes
• Sugarless hard candies
• Glycerine-based cough drops and lemon flavored glycerine mouthwash
• Medications may be added, changed, or dosages altered to provide increased salivary flow

Remember → saliva has several important functions:
• Washes away food debris and plaque from the teeth to help prevent decay.
• Limits the growth of bacteria that cause tooth decay and other mouth infections.
• Bathes the teeth and supplies minerals that allow remineralization of early cavities.
• Lubricates foods so they may be swallowed more easily.
• Provides enzymes that aid in digestion.
• Helps us enjoy foods by aiding in the "tasting" process.
• Moistens the skin inside the mouth to make chewing and speaking easier.

A chronically ill-fitting denture may cause an inflammatory fibrous hyperplasia adjacent to its border. This condition is known as:

- Verrucous vulgaris
- Inflammatory papillary hyperplasia
- Stomatitis nicotina
- Epulis fissuratum

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• **Epulis fissuratum**

The cleft-like lesions of **epulis fissuratum** result primarily from overextension of **denture flanges.** The overextension may result from long-term neglect or settling subsequent to residual ridge resorption. Traumatic occlusion of natural teeth opposing an artificial denture may also cause this condition.

Denture stomatitis is a localized or generalized chronic inflammation of the denture bearing mucosa. Clinically, there is redness and a burning sensation. There may be or may not be discomfort. **Trauma and secondary fungal infection** appear to be the most likely causes of denture stomatitis.

The treatment generally consists of:

1. Improved oral hygiene
2. Tissue rest
3. Antifungal therapy *(nystatin)*
4. Resilient tissue conditioners
5. New, well-fitting dentures

Most cases of which disease below are detected because patients complain that their dentures are not fitting since the bone has become too large for them?

- Addison's disease
- Paget's disease
- Hashimoto's disease
- Multiple sclerosis

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• **Paget's disease**

Paget's disease *(also called osteitis deformans)* of the bone is a chronic bone disorder in which bones become enlarged and deformed. The exact cause is not known. It is characterized by excessive breakdown of bone tissue, followed by abnormal bone formation. The new bone is structurally enlarged, but weakened with heavy calcifications.

Important: Involvement of the skull may enlarge head size and cause hearing loss and blindness if the cranial nerves are damaged by the bone growth.

Dental Considerations → relieving the tissue surface of the dentures and relining with resilient materials can extend the life of the dentures. However, remaking the dentures frequently is **unavoidable.**

Notes:

1. Children who wear dentures and patient's with acromegaly who wear dentures also often need to have their dentures relined or remade to allow for bone growth.
2. Diseases of bone growth or expansion are **much rarer** than those of bone loss.
3. **Osteoporosis** is the most common change associated with systemic disease. This condition is a generalized defect in which the quantity and quality of bone in the skeleton is reduced.

Which of the following is **not** associated with **diabetes?**

- Delayed healing
- Rapidly progressing periodontal disease with marked alveolar bone loss
- Mucosal bleeding
- Increased calculus formation
- A predilection for periapical abscesses

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• **Mucosal bleeding** → bleeding disorders **are not** associated with diabetes.

Diabetes is a disease that can affect the whole body — your eyes, nerves, kidneys, heart, and other important systems in the body. It can also affect your mouth. People with diabetes face a higher than normal risk of oral health problems. The link between diabetes and the development of oral health problems is high blood sugar. If the blood sugar is poorly controlled, it is more likely that oral health problems will arise. This is because uncontrolled diabetes impairs white blood cells, which are the body's main defense against bacterial infections that can occur in the mouth. Just as studies have shown that controlling blood sugar levels lowers the risk of major organ complications of diabetes — such as eye, heart, and nerve damage — so too can good diabetes control protect against the development of oral health problems.

Even controlled diabetics present problems for the prosthodontist. The oral mucosa is prone to the development of sore spots which heal poorly and often become secondarily infected.

Principles to keep in mind when constructing **dentures** for patients with any debilitating disease:
• **Maximum** extension
• **Narrow** occlusal table
• **Non-pressure** impression technique
• **Do not** use porcelain teeth
• **Establish** a good occlusion
• **Reinforce** oral hygiene
• Place on **6-month recall** *(sooner if necessary to reinforce oral hygiene)*

All of the following muscles are involved in elevating the mandible *(closing the mouth)* **except:**

- Masseter muscles
- Medial pterygoid muscles
- Lateral pterygoid muscles
- Temporalis muscles

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- **Lateral pterygoid muscles** → contract to cause protrusion

Muscles Acting on Joint				
Open *(Depress)*	**Close** *(Elevate)*	**Protrude**	**Retract**	**Lateral Displacement**
Lateral pterygoid Digastric *(anterior belly)* Omohyoid	Masseter Medial pterygoid Temporalis *(anterior fibers)*	Lateral pterygoids *(together)*	Posterior fibers of temporalis	Lateral pterygoids *(individually)*

Important:

- The **lateral pterygoid muscles** are mostly responsible for positioning and translating the condyles.
- If the mandible fractures, **upward displacement** of the fractured segment would be caused by the closing muscles *(masseter, medial pterygoid, and temporalis).*

You have prepared tooth # 8 for a dowel core. Which of the following will best serve the purpose of provisionally restoring the tooth.

- Place a piece of paper clip or stiff wire into the canal and put a resin filled polycarbonate crown over it
- Take an alginate impression, fabricate an acrylic crown and cement it in with zinc phosphate
- Use the overimpression technique to construct the provisional crown and cement it in with any temporary luting cement
- Use a pre-formed metal crown and cement it in with a temporary luting cement
- None of the above

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- **Place a piece of paper clip or stiff wire into the canal and put a resin filled polycarbonate crown over it**

***It is difficult to fabricate a provisional restoration for a tooth that has been prepared for a dowel crown because there is so little intact supragingival tooth structure. The above mentioned option is a satisfactory solution for this problem.

A temporary should satisfy the following requirements:

- **Provide pulpal protection** → the restoration **must be** fabricated of a material that will prevent the conduction of temperature extremes.
- **Positional stability** → the tooth **should not** extrude or drift in any way.
- **Occlusal function** → being able to function occlusally on the temporary will aid patient comfort and will also **aid** in preventing tooth migration.
- **Easily cleaned** → the temporary **must be** made of a material and contour that the patient will be able to keep clean.
- **Nonimpinging margins** → it is of the **utmost importance** that the gingival margins of temporary **not impinge** upon the gingival tissue. The resulting inflammation could result in hypertrophy, gingival recession, etc. the margins **must be** well polished.
- **Strength and retention** → the temporary **must** stand up to the forces to which it is subjected without breaking or coming off.
- **Esthetics** → if the temporary is on an anterior tooth, it **must** provide a good cosmetic result.

One **disadvantage** of dental porcelain restorations is:

- Poor esthetics
- Expansion
- Brittleness
- Radioactivity

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• **Brittleness**

The compressive strength of ceramic bodies is greater than either their tensile or their shear strength. The tensile strength is low because of the unavoidable surface defects. The shear strength is low because of the lack of ductility or ability to shear, caused by the complex structure of the glass ceramic materials. The shear and tensile strengths of the fired porcelain are so low that the slightest imperfection in the preparation of the cavity in the tooth may cause the jacket crown to fracture in service.

Notes:

1. Many porcelains rust at a temperature **over 2000°F.**
2. The **glaze** firing is the last firing and it produces a smooth, translucent surface.
3. Dental porcelain has **good biocompatibility,** but is very brittle.
4. All-porcelain crowns are superior to ceramo-metal crowns in **esthetics** only *(as compared with strength, hardness and toughness).*
5. In a ceramo-metal unit, the porcelain surface should be under **slight compressive stress** *(it should not be under tensile or shear stress).*
6. The core material in an all ceramic crown is usually a **high strength sintered ceramic.**
7. Porcelain substrate alloys, when compared to traditional alloys, **melt at a higher temperature.**

Which of the following is true concerning the **first application** of dental porcelain to a restoration?

- The first layer is called incisal and provides a stable substrate for additional layers
- The first layer is called body and is very translucent
- The first layer is called opaque and effectively covers the metallic sheen of the underlying casting
- The first layer is called glaze and provides reduction of surface oxides on the metal casting

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- **The first layer is called opaque and effectively covers the metallic sheen of the underlying casting**

The elements in the opaque layer **create the chemical bond** of the porcelain to the metal alloy. This layer **masks the color** of the metal.

The **body porcelain** makes up the bulk of the restoration, providing most of the color or shade.

The **incisal porcelain** is a translucent layer of porcelain in the incisal or cuspal portion of the tooth.

The opaque is **applied first** to mask the metal and to give the restoration its basic shade. **Body** porcelain is then added over the opaque. **Incisal** porcelain is added to the incisal one-third to give translucency. The restoration is bulked out *(overcontoured)* to compensate for the **20% shrinkage,** which occurs during firing.

Opaque porcelain showing through on the facial surface of a metal-ceramic crown may be caused by the following:
- Inadequate tooth reduction → fault of the dentist
- The metal is too thick → fault of the lab
- The opaque porcelain is too thick → fault of the lab
- Inadequate thickness of the body porcelain → fault of the lab

Color can be described in terms of three standard components. Which of the following **is not** a standard component used to describe a color?

- Hue
- Chroma
- Value
- Intensity

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• **Intensity**

Three standard descriptions of color:

1. **Chroma** → is the saturation or strength of a color *(degree of saturation of the hue)*
2. **Value** *(or brightness)* → is the relative amount of lightness or darkness in a color
3. **Hue** → refers to color tone *(e.g., red, blue, yellow, etc.)* and is synonymous with the term color.

Notes:

1. Value is the single **most important** factor in shade selection.
2. **Intensity** is included in the term value.

Important points about staining porcelain:

- Stains are **metallic oxides** that fuse to the porcelain during a predetermined firing cycle.
- Drastic changes of the hue *(color or shade)* are often impossible. Orange stain is the **most often** used to change the hue.
- **Staining** a porcelain restoration will **reduce the value** *(as will using a complementary color)*. It is **almost impossible** to increase the value.
- **Chroma** can be successfully **increased** by the use of stains, particularly in the gingival area.

Note: In **esthetics,** the value of a denture tooth depends upon the relative whiteness or blackness of its color.

The **surface characteristics** of porcelain can affect the perceived form of the final restoration in the following ways:

- A **smooth surface** will give the impression of a **larger size.**
- Changes in **contour** can be used to **alter the apparent** long axis inclination of a tooth.

Teeth that appear to be color matched under one type of light may appear very different under another light source. This **phenomenon** is called:

- Fluorescence
- Metamerism
- Opaqueness
- Translucency

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• **Metamerism**

This property is important in **matching** the shade of a metal-ceramic crown to a natural tooth. Thus, if possible, color matching should be done under **two** or **more** different light sources, one of which should be sunlight. **Note:** Staining of the porcelain **will increase** metameric responses.

Fluorescence is the optical property by which a material *(for example, teeth)* **reflects** ultraviolet radiation. The energy that the tooth absorbs is converted into light with longer wavelengths, in which the tooth actually becomes a light source. Human teeth fluoresce mainly blue-white hues *(400–450 nm range)*. Fluorescence makes a **definite contribution** to the brightness and vital appearance of natural teeth.

The production of color sensation with a **pigment** is a physically different phenomenon from that obtained by optical reflection, refraction and dispersion. The color of a pigment is **determined** by selective absorption and selective radiation *(scattering)*.

Remember: The **light source** affects the perception of color, because the **light source** must contain the wavelength of the color to be matched in order to see that color.

Glazed porcelain is:

- Obtained by heating the previously fired body very slowly for 60 minutes at its fusing temperature
- Nonporous, resists abrasion, possesses esthetic ability and is well tolerated by the gingiva
- Not as durable *(in its surface characteristics)* as an over-glazed porcelain
- All of the above

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- **Nonporous, resists abrasion, possesses esthetic ability and is well tolerated by the gingiva**

At least three stages are generally recognized in the firing of dental porcelain: **1)** low bisque firing **2)** medium bisque firing and **3)** high bisque firing. The temperature at which each occurs depends upon the type of porcelain used.

A **natural glaze** occurs when the porcelain restoration itself is glazed by a separate firing *(this process is referred to as* **"the glaze firing"***).* If the body, previously fired as a high bisque, is heated rapidly *(10-15 minutes)* to its fusion temperature and maintained at that temperature for approximately 5 minutes before it is cooled, the glass grains flow over the surface to form a vitreous layer, which is called a **glaze.**
Note: This type of glaze is much **more permanent** than the overglazes.

Overglazes *(or applied glazes)* are ceramic powders that may be added to a porcelain restoration after it has been fired. A transparent, glossy layer forms over the surface of the porcelain restoration at a maturing temperature lower than that of the body porcelain. The result is a glossy or semi-glossy surface that is non-porous. **Erosion of this overglaze may occur in the mouth and this leaves a rough and sometimes porous surface.**

Note: Glazed porcelain *(either type)* is the **least irritating** to the gingival tissues compared with polished cast gold, polished direct filling gold and polished acrylic resin.

Low–fusing **porcelain** is usually used for the manufacture of:

- Denture teeth
- All ceramic crowns
- Metal - ceramic crowns
- All of the above

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• **Metal - ceramic crowns**

Classification of dental porcelain:
• High-fusing porcelains → used for denture teeth
• Medium-fusing porcelains → used for all – ceramic and porcelain jacket crowns
• Low-fusing porcelains → used for metal – ceramic crowns

Remember: The **compressive strength** *(350 – 550 MPa)* of a porcelain restoration **is greater** than its tensile *(20 – 60 MPa)* or shear strengths, which is typical of a brittle solid.

Dental Porcelain is a mixture of **feldspar** *(main constituent)* and **quartz.** Metallic oxides *(i.e., silica, alumina and potassium oxide)* are used to impart the proper shade to the porcelain. When feldspar undergoes fusion, it forms a glassy material, which gives porcelain its translucency. It acts as a matrix for the high fusing quartz, which in turn forms a refractory skeleton around which the other materials fuse. **Quartz** is a strengthener. **Kaolin,** a clay, is a sticky material that binds the particles together when the porcelain is "green" or unfired.

Note: Aluminous porcelain uses **alumina** instead of quartz as a **strengthener.** This type of porcelain is **considerably stronger** than conventional porcelains.

***Dental porcelain restorations are **brittle** and **are not** capable of much plastic deformation.

The **process** by which a casting is heated in a porcelain furnace to a temperature of 980°C **to burn off** any remaining impurities prior to adding porcelain is called:

- Quenching
- Pickling
- Degassing
- Investing

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• **Degassing** → sometimes called heat treatment

It is necessary for **all gold-porcelain systems.** Degassing of the metal at too low a temperature will effect the formation of the oxide layer, which is important in bonding of the porcelain. The number of bubbles formed at the interface **decreases** as the time and temperature of degassing are **increased.**

After degassing the casting is ready for porcelain addition. The metal framework must **not be contaminated** by handling prior to porcelain addition. If it is, the bond of the opaque will be weakened.

Causes of porcelain fracture at **porcelain - metal interface:**
- Poor metal framework **design** → **main cause** of fracture
- Degassing of the metal at **too low a temperature,** which effects the formation of the oxide layer, **thus** decreasing the bond
- Contamination of metal **prior** to opaque application
- **Fusing** the opaque coat of porcelain at **too low** a temperature or for **too short** a time

Note: Both the metal *(alloy)* and ceramic *(porcelain)* **must** have coefficients of thermal expansion that are **closely matched** *(alloy is usually slightly harder)* if undesirable tensile stresses at the interface are to be avoided *(fracture of the porcelain)*. Alloys should have a **high proportional limit,** and particularly, a **high modulus of elasticity.** Alloys with a high modulus will reduce stress on the porcelain.

Pickling is the process of removing surface oxides from a casting prior to polishing. The casting is placed in an acidic solution which **reduces the surface oxides.** To prevent injury, safety goggles should always be worn when pickling.

Comparing household porcelain to dental porcelain, household porcelain:

- Contains larger amounts of feldspar
- Contains larger amounts of kaolin
- Is fabricated at temperatures above the melting point
- All of the above

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- **Contains larger amounts of kaolin**

***Kaolin,** a clay, is a sticky material that binds the particles together when the porcelain is "green" or unfired.

The earliest successful porcelain systems used conventional feldspathic porcelain, derived from the natural mineral feldspar. This material was used for producing all-ceramic jacket crowns, which were very esthetic. Dental feldspathic porcelain is predominantly a glass material with an amorphous *(non-crystalline)* structure. Glass mainly consists of a three-dimensional network structure of silica *(silicon-oxygen)*. The newer systems make use of alumina, leucite, etc. as the reinforcing phase.

All-ceramic crowns may be classified by the following two ways:

1. **Composition**
   - **Feldspathic porcelain** → sometimes still used used for a conventional porcelain jacket crown
   - **Aluminous porcelain** → found in Vitadur, Hi-Ceram, Cerestore and In-Ceram systems
   - **Mica glass** → found in Dicor and Cerapearl systems
   - **Crystalline-reinforced glass** → used with Cerestore and IPS Empress systems
2. **Method of fabrication**
   - **Refractory die technique** → used with Optec, Hi-Ceram and In-Ceram systems
   - **Casting** → used with Dicor systems
   - **Press Technique** → used with Cerestore and IPS Empress systems

Notes:

1. **Crystalline-reinforced glass** *(employed in the Optec and IPS Empress systems)* is a glass in which a crystalline substance such as leucite is dispersed. **It imparts strength to the ceramic.**
2. **Alumina** *(employed in the Vitadur, Hi-ceram, Cerestore and In-Ceram systems)* is used to **reinforce glass.** The strength is determined by the amount of alumina reinforcement.

How would you classify a **removable partial denture** in which a portion of the functional load is **carried by the residual ridge** at one end of the denture base segment while the other end of the base segment is **supported by natural teeth?**

- A tooth-borne removable partial denture
- A bilateral distal extension removable partial denture
- A unilateral distal extension removable partial denture

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• **A unilateral distal extension removable partial denture**

A **distal extension** removable partial denture *(either bilateral or unilateral)* receives its support from the residual ridge, tissue-bearing areas, selected abutment teeth and the fibrous connective tissues overlying the alveolar process.

The **most important factor** in determining the success of distal extension removable partial dentures *(bilateral and unilateral)* is **proper coverage over the residual ridge.** Coverage of the free-end should extend **over the retromolar pad** to create stability of the RPD and to minimize the torquing forces on the abutment teeth.

Notes:

1. The **first step in relining** a distal extension removable partial denture is to **verify the fit of the framework. Important:** This is done first even if you are also relining a maxillary complete denture which is opposing this distal extension.
2. When relining a distal extension partial denture, apply finger pressure to the rests and indirect retainer.
3. If the indirect retainers are not seated as the extension bases are depressed, **the bases need relining.**

Important: If a patient complains of sensitivity to percussion on an abutment tooth of a **distal extension partial denture,** the most likely cause is the **occlusion** on this abutment. **Deflective occlusal contacts** can also cause a feeling of **"looseness"** to the denture.

The **major connecter** is:

- The connecting tang between the denture and other units of the prosthesis
- The part of the denture base which extends from the necks of the teeth to the border of the denture
- The unit of a partial denture that connects the parts of the prosthesis located on one side of the arch with those on the opposite side
- None of the above

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- **The unit of a partial denture that connects the parts of the prosthesis located on one side of the arch with those on the opposite side**

The major connector **must be rigid** so that stresses applied to any one portion of the denture may be effectively distributed over the entire supporting area. It connects other components of the prosthesis and provides cross-arch stabilization.

They should be **designed** and **located** with the following guidelines:

- They should be free of movable tissues
- Relief should be provided
- They should not impinge on gingival tissues
- Bony and soft tissue prominences should be avoided during placement and removal

Common Major Connectors	
Maxillary	**Mandibular**
• Palatal plate • Palatal strap • Anterior or posterior palatal bars • Anterior-posterior palatal bar • Horseshoe design	• Lingual plate • Lingual bar • Sublingual bar • Labial bar

Note: A **minor connector** is the connecting link *(or tang)* between the major connector or base of the partial denture and other units of the prosthesis, such as **clasps, indirect retainers and occlusal rests.**

When designing an **anterior-posterior palatal bar maxillary major connector,** the anterior, posterior, and lateral straps should be about:

- 2 – 4 mm wide
- 6 – 8 mm wide
- 8 – 10 mm wide
- At least 12 mm wide

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- **6 – 8 mm** → this allows for adequate strength while allowing for maximum tissue exposure both over the palate and in the area of the marginal gingiva

The **most rigid** palatal major connector is the anterior-posterior palatal bar connector. It may be used in almost any maxillary partial denture. To attain symmetry, both anterior and posterior connectors should cross the midline at right angles rather than on a diagonal.

Note: If the palatal bar is too thick, the patient may have difficulty in pressing food backward for swallowing.

The **palatal plate connector** is a thin, broad connector which may be used in the following ways:
- Connector for simple edentulous areas
- Full palatal coverage

*** This type of connector covers more tissue, and in turn, produces added retention from atmospheric pressure and interfacial surface tension.

The **palatal horseshoe-shaped** connector should only be used when a large, inoperable torus prevents the use of other designs.

Single palatal bars are objectionable because they lack rigidity. Their use is limited to tooth-borne restorations for bilateral short span edentulous areas. The wide, thin bar *(strap)* **is more rigid** with less bulk compared to a narrow bar.

Remember: The major and minor connectors **must be rigid** in order for the functional stresses that are applied to the partial dentures to be distributed evenly throughout the mouth.

A **mandibular lingual bar** major connector requires a minimum of:

- 3 mm of vertical height between the gingival margin and the floor of the mouth
- 5 mm of vertical height between the gingival margin and the floor of the mouth
- 7 mm of vertical height between the gingival margin and the floor of the mouth
- 9 mm of vertical height between the gingival margin and the floor of the mouth

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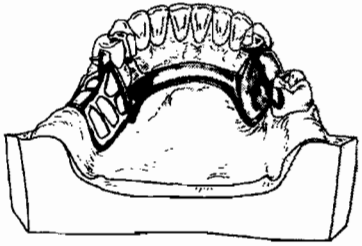
- **7 mm of vertical height between the gingival margin and the floor of the mouth**

Lingual bars should be placed so that the upper border is a **minimum of three millimeters** below the gingival margins and at least 4 mm is required for the vertical height of the lingual bar *(3 + 4 = 7 mm minimum)*. When severely tipped premolars and molars are present, an alternate framework design or crowns are recommended.

The mandibular labial bar is rarely indicated as a major connector for a removable partial denture. It can be used satisfactorily when large mandibular tori interfere with conventional lingual bar placement or when the lower teeth are severely lingually tipped and placement of a lingual bar is not possible.

Note: Lingually inclined mandibular premolars interfere **most frequently** with major connectors.



Lingual Bar

All of the following are **advantages** of using a cast chromium-cobalt alloy for removable partial dentures **except:**

- Corrosion resistance
- High strength
- High flexibility
- Low specific gravity

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- **High flexibility**

***Chromium-cobalt alloys are quite **inflexible.** They have essentially **no ductility** or **malleability** after they are cast.

The popularity of chromium-cobalt alloys for fabrication of cast frameworks for removable partial dentures has been attributed to their low density *(weight),* high modulus of elasticity *(stiffness)*, low material cost and resistance to corrosion.

Composition of chromium alloys for partial dentures:
- **Chromium** → ensures that the alloy will resist tarnish and corrosion *(due to formation of a complex chromium oxide film)*
- **Cobalt** → contributes strength, rigidness and hardness
- **Nickel** → increases ductility
- **Minor constituents** → carbon has a pronounced effect on the strength, hardness and ductility. Tin, indium and other readily oxidized minor components of the alloy function to improve bonding.

Remember: The form of **chrome-cobalt alloy** connectors is flat, broad and reinforced along the borders by the bead on the tissue surface. The process of beading **not only** helps maintain tissue contact, but also provides additional strength *(for maxillary major connectors).*

Possible causes of fracture of chromium-cobalt partials include work hardening, shrinkage porosity, low percent elongation and excessive carbon in the alloy.

The type or form of **clasp** is generally selected after:

- Looking at the x-rays
- Talking to the patient
- Surveying the cast
- The initial try-in of the metal framework

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• **Surveying the cast**

Surveying is generally performed at right-angles to the occlusal plane in the first instance, as this is the likely path of displacement. Surveying will identify three principal factors: **(1)** The presence of undercuts **(2)** The contour of the undercuts relative to the gingival margin and **(3)** The depth of the undercuts

Extracoronal retainers are the **most common** type of direct retainer that is used for removable partial dentures. They are called **clasps.** Their ability to provide retention is based on the resistance of metal to deformation. There are two basic categories of extracoronal retainers: **suprabulge** and **infrabulge retainers.**

Intracoronal retainers are the other type of direct retainer that is sometimes used for removable partial dentures. These are attachments which are built into the contour of a crown *(casting)* to produce mechanical and frictional retention. By eliminating the need for a visible retentive clasp, these retainers give **optimal esthetics.** They provide vertical support through the rest seat located more favorably in relation to the horizontal axis of the abutment teeth.

Important: Intracoronal retainers **are not used** when a partial denture depends upon an edentulous area for support *(distal extension)*. These retainers may provide a rigid connection between the denture and the abutment *(fine for tooth-borne partials)*. However, in distal extensions, functional motion **must be permitted** without torquing the abutment teeth.

Suprabulge retainers *(clasps)* originate:

- Below the height of contour
- Above the height of contour
- Above the 0.08" undercut
- Above the occlusal surface of most molars

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• **Above the height of contour**

Clasps that originate from **above the survey line,** usually from an occlusal rest, and angle downward across the clinical crown until the tip is located in a prescribed amount of undercut are suprabulge retainers *(clasps)*.

Examples of suprabulge retainers:

- **Circumferential clasp** → is composed of a buccal and lingual arm originating from a common body. Usually one arm is retentive while the other functions in bracing. This clasp is used to engage undercuts located on the side *(mesial-distal)* of the tooth opposite to the site of the rest.

- **Ring clasp** → encircles nearly all of a tooth in order to engage an undercut located on the same side of the tooth as the rest. It should **not be used** where the caries rate is high or where esthetic considerations are dominant.

- **Embrasure clasp** → is used when **no edentulous space exists** at the clasp assembly site.

- **Reverse-action clasp or hairpinclasp** → may be **used to engage** an undercut located on the same side of the abutment as the rest. It may be used on any posterior tooth, but it covers a lot of tooth surface and may be esthetically objectionable.

- **Extended arm clasp** → is a circumferential clasp which **extends to** neighboring teeth in order to provide increased splinting and to engage a more favorably located undercut.

- **Half and half clasp** → consists of **one circumferential clasp** emanating from the rest area and **another arm** from the minor connector on the opposite side.

All of the following are **advantages** of infrabulge retainers as compared to suprabulge retainers **except:**

- More efficient retention
- Less distortion of coronal contours
- Less tooth contact
- Cleaner
- Less bothersome to vestibular tissues
- Less prone to caries
- Esthetically superior in most cases
- Greater adjustability

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- **Less bothersome to vestibular tissues**

***This is **false;** infrabulge retainers are **more bothersome** to vestibular tissues.

Other **disadvantages** of infrabulge retainers:

- **Too flexible** for effective bracing
- Can be **esthetically objectionable** in patients with a high lip line
- Where there are not enough guideplanes to positively establish the path of insertion, opposing cross-arch undercuts can be used. In these cases, the effectiveness of infrabulge clasps are diminished because the unseating motion *(without guideplanes)* is rotary, vertical and horizontal.

Infrabulge retainers are clasps that originate from below the survey line. They are metal projections emanating from the denture base struts in the framework. They course through the denture base and project parallel to the mean plane of the gingiva until they make a gentle right angle turn. Then they cross the gingiva and come to rest upon the abutment tooth in a specified undercut area.

Infrabulge retainers **must not** be placed into tissue undercuts, nor should they contact the abutment at any place **except** at the specified undercut.

The **most common** forms are I, J, U, L and T bar clasps. These bar clasp arms and circumferential clasp arms *(a suprabulge retainer)*, **both provide** retention by the resistance of metal to deformation, rather than frictional resistance created by the contact of the clasp arm to the tooth.

Which of the following **indirect retainers** will provide the best leverage against lifting of the denture base?

- The one located the **closest** to the clasp tips which is **located furthest** from the edentulous area
- The one located the **furthest** from the clasp tips which is **located nearest** to the edentulous area
- The one located the **furthest** from the clasp tips which is **located furthest** from the edentulous area

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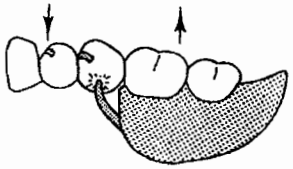
- **The one located the furthest from the clasp tips which is located nearest to the edentulous area** *(see picture below)*

Explanation of answer: As unseating occurs in the edentulous segments, a line through the rests located furthest from the retentive clasp tips acts as the fulcrum in a Class III lever system. Moving the fulcrum line still further from the clasp tips improves the mechanical advantages of the lever arm system. By **maintaining this position,** the most distant rests augment the retentive action of the clasp and indirectly contribute to retention. Thus, the term **indirect retainers** pertains to rests, which augment mechanical retention.



As the denture base moves upward, the most **anterior rest** *(which is the indirect retainer)* resists downward movement and this increases the effectiveness of the direct retainer.

Which of the following Kennedy classes of removable partial dentures are **not** tooth-borne?

- Class I
- Class II
- Class III
- Class IV

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• **Class I and Class II**

Tooth-borne removable partial dentures *(Class III and Class IV)* **depend entirely** on abutment teeth for support.

Kennedy classifications are based on the most posterior edentulous area to be restored. Although Class III and IV partial dentures are supported entirely by the abutment teeth, Class I and II partial dentures are **supported also** by the residual ridge, the subjacent tissues and the fibrous connective tissue overlying the alveolar process.

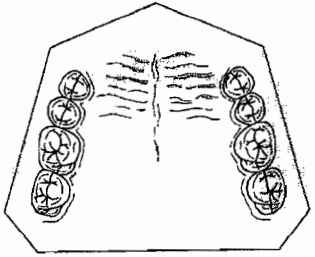
Note: The alveolar ridge resorption under the distal extension partial denture is of particular concern and can be reduced by **maximizing** the coverage of these supporting areas.

Very Important: Likewise, the periodontal damage to abutment teeth **is avoided** with firm tissue support → maintaining a stable base-tissue relationship.

Remember: Rests should be placed on abutment teeth next to the edentulous areas for maximum support when designing a tooth-borne partial. These rests **limit movement** of the denture in a gingival direction.

The following **partially edentulous** arch would be classified as:

- Kennedy Class I
- Kennedy Class II
- Kennedy Class III
- Kennedy Class IV



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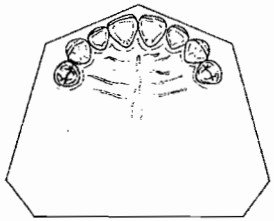
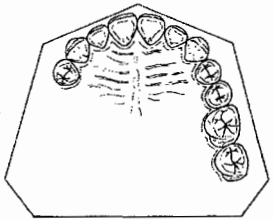
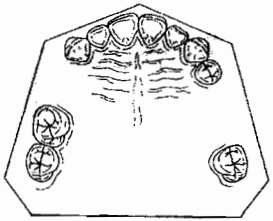
• **Kennedy Class IV** → anterior extensions crossing the midline



Kennedy Class I
Bilateral distal extension



Kennedy Class II
Unilateral distal extension



Kennedy Class III
Unilateral edentulous spaces bound by teeth

Any other additional edentulous area is referred to as modification *(except in Class IV)* e.g., If Kennedy Class I *(bilateral distal extension)* above, also has another edentulous area anteriorly, then it would be referred to as Class I modification I.

Remember: Craddock classification is based upon the denture type.

- Type I → Mucosa borne
- Type II → Tooth borne
- Type III → Mucosa and tooth borne

What is the **recommended treatment** for a patient who has lost her four maxillary incisors some time ago and has suffered **excessive ridge resorption?**

- A conventional six - unit fixed bridge
- No treatment
- A removable partial denture
- A Maryland bridge

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• **A removable partial denture**

***If **excessive ridge resorption** has occurred after tooth loss in the anterior region, the pontics required to replace these teeth may be quite unesthetic. A **removable partial denture** with its tissue colored acrylic base can provide this esthetic consideration.

Other situations in which a **removable partial denture** is specifically intended rather than a fixed bridge:

- **Distal extension** → obviously, if several teeth must be replaced and no posterior abutment is present, then a removable partial denture must be used *(other option would be implants)*. It is possible, however, to cantilever one tooth in a fixed bridge if **at least** two very sound teeth exist anterior to the space.

- **Long span edentulous area** → sufficient abutment teeth are not present to support the occlusal forces, which would be placed on the fixed bridge.

- **Periodontally involved abutment teeth** → the bracing and cross-arch stabilization of a removable partial denture in this case makes it the ideal treatment.

- **Following recent extractions** → a temporary removable partial denture may be provided until tissues have had time to heal properly and fixed bridgework can be done.

- **Economics** → may force the use of a partial denture as an interim solution to a problem that must eventually be solved with fixed prosthodontics.

All of the following are **indications** for using a **linguoplate** as a mandibular major connector **except:**

- To avoid a high lingual frenum or when there is no space in the floor of the mouth
- Mandibular tori that cannot be removed
- To support periodontically weakened teeth
- Lingually tilted mandibular anterior teeth
- Severe anterior crowding
- To serve as retention for lower anterior denture teeth when the prognosis for the natural teeth is guarded

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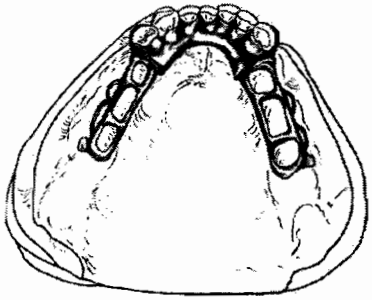
• **Severe anterior crowding**

***This is a **contraindication** to the use of linguoplate.

The **linguoplate is a lingual bar** that has been extended upward to cover the cingula and interproximal spaces between mandibular anterior teeth. It should be thin and follow the contours of the teeth and embrasures. The upper border should be located at the middle third of the lingual surface of the teeth and extend upward to cover interproximal spaces to the contact point.



Lingual Plate

It is important that a **wrought wire** clasp have an **elongation percentage** of:

- More than 25%, allowing the clasp to bend without microstructure changes that could compromise its physical properties
- More than 6%, allowing the clasp to bend without microstructure changes that could compromise its physical properties
- Less than 6%, allowing the clasp to bend without microstructure changes that could compromise its physical properties
- Less than 25%, allowing the clasp to bend without microstructure changes that could compromise its physical properties

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- **More than 6%, allowing the clasp to bend without microstructure changes that could compromise its physical properties**

A **wrought wire** clasp is fabricated by drawing the metal from which it is made into a wire. The success of wrought wire clasps depends on their physical properties and the changes that may occur during fabrication. Laboratory procedures can compromise desirable physical properties due to improper heating and cooling. Manufacturer directions should be followed for each particular alloy. It is important that a wrought wire clasp have an **elongation percentage of more than 6%,** allowing the clasp to bend without microstructure changes that could compromise its physical properties. Tapering a wrought wire clasp to 0.8 mm at the tip before contouring allows for more uniform stress distribution throughout the clasp, making it more serviceable and efficient. **Remember:** The **most important** mechanical property involved when a clasp is adjusted is **elongation.**

Advantages of **wrought-wire clasps** *(gold or cobalt-chromium)* over clasps of the same composition:
• Higher yield strength • Greater flexibility • More ductile • More resilient

Designing a **chrome cobalt clasp** to engage less undercut is the **most reliable way** to decrease its retentiveness; switching to a gold clasp while maintaining the same amount of undercut will have a similar result. Gold clasps offer **half the retention** of chrome cobalt clasps while engaging identical undercuts. Because the grain size of chrome cobalt is large by comparison, it possesses a lower proportional limit; as a result, the risk of fracture increases as its bulk decreases. Since cobalt work hardens more rapidly than gold, bending chrome cobalt clasps is associated with an increased likelihood of fracture.

Note: A **cast metal** is any metal that is melted and cast into a mold *(e.g., an inlay, crown or clasp)*. When the casting is **cold-worked** in some manner to provide the required article or appliance *(e.g., wire)*, it is called a **wrought metal** in contrast to a cast metal. As stated above, many mechanical properties of the wrought structure are **superior** to those of the cast structure *(e.g., tensile strength, hardness and strength)*. This means that a wrought structure having a smaller cross-section than a cast structure may be used as a retainer arm *(retentive)* to perform the same function.

All of the following are true regarding indirect retention **except:**

- It is a rest seat
- It is located as far anterior as possible
- The function is to prevent vertical dislodgement of the distal extension base of a removable partial denture
- It is usually an MO rest seat on a first molar

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- **It is usually an MO rest seat on a first molar**

***This is **false;** it is usually found on a canine or premolar.

It is an **anti-rotational device** fabricated by a rest / connector combination and placed as far forward from the embrasure clasp as possible. It counteracts the upward rotation of the edentulous base. This also serves as a 3rd reference for seating of the framework and making altered cast impressions. **Note:** The indirect retainer **may be omitted** in some designs → tooth-borne partial dentures.

Indirect retainers also serve another related function. In their absence, upward dislodgement of distal expansion bases would be accompanied by downward motion of the anterior part of the major connector. **By preventing** such downward movement, the indirect retainer **protects the soft tissues** from impingement by the major connector.

Remember:
- An **indirect retainer** should be placed **as far** from the distal extension base as possible in a prepared **rest seat** on a tooth capable of supporting its function.
- The term indirect retainer pertains to **rests,** which augment mechanical retention.

The strength, hardness, and tensile strength of wrought wire is approximately:

- 5% greater than the cast alloy from which it was fabricated
- 25% greater than the cast alloy from which it was fabricated
- 50% less than the cast alloy from which it was fabricated
- 75% greater than the cast alloy from which it was fabricated

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- **25% greater than the cast alloy from which it was fabricated**

Wrought wire clasps:

- Have a **greater flexibility** and **adjustability** than the cast clasps
- **Are tougher** than cast clasps
- Have a **greater tensile strength** than cast clasps and therefore can be used in smaller diameters to provide greater flexibility without fatigue and ultimate fracture.

Important: Having been formed by being drawn into a wire, the wrought-wire clasp has **toughness** and **ductility** exceeding that of a cast clasp arm. The clinical effect of this is that there is an increased capacity for deformation of the wrought-wire without breaking.

However, the yield strength of both gold and chromium-cobalt alloy wrought wires can be **drastically reduced** simply by subjecting the wire to too much heat. If the heat is high enough, the fibrous microstructure of the wrought wire disappears and is replaced by a grain or crystalline microstructure. This process is known as **recrystallization** or gain growth and is a most undesirable occurrence in wrought-wire retainer arms.

Note: The **terminal end** of the retentive arm is optimally placed in the middle of the gingival third of the clinical crown. **However,** it is acceptable to place it at the junction of the gingival and middle one-third of the clinical crown. When the partial is completely seated, the retentive arm should be **passive** and applying **no pressure** on the teeth.

In the mesial rest, guide plane and I-bar design, **reciprocation** is achieved by all of the following **except:**

- Rigid plating
- Minor connectors
- Guide planes extended around the vertical line of abutments
- Achieving balanced occlusion
- Contact areas of proximal teeth
- Reciprocal clasp arms

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• **Achieving balanced occlusion**

Reciprocation as applied to partial dentures refers to the function of the lingual clasp arm *(which is the reciprocal clasp arm)* to **counteract forces** exerted by the buccal clasp arm *(which is the retentive clasp arm).*

Reciprocation is the means by which one part of the framework **opposes the action** of the retainer in function. Reciprocation may be achieved by opposing flexible retainers with guide planes, minor connectors, rigid clasp arms or plating. If true reciprocation is to occur, the reciprocating element **must be placed opposite** the direct retainer and must contact the abutment as the retentive tip passes over the height of the contour of the tooth.

The **cingulum rest** is usually confined to preparation on:

- Maxillary lateral incisors
- Maxillary canines
- Mandibular lateral incisors
- Mandibular canines

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• **Maxillary canines**

***These teeth have a **gradual lingual incline** and a **prominent cingulum.** In some instances, cingulum rests may be placed on maxillary central incisors. The lingual slope of the mandibular canine is usually too steep for an adequate cingulum rest to be placed in the enamel.

The **cingulum rest** is a vertical stop on an anterior tooth whose lingual anatomy lends itself to ready preparation for a positive seat. Not all teeth have sufficient cingulum contour to receive a seat.

The **incisal rest** is employed when other preferred support is not available. The high placement of this style of rest may be esthetically objectionable. The distal incisal rest is usually less esthetically visible than the mesial incisal rest. Never place an incisal rest so deep that it interferes with the proximal contact.

Note: Two advantages of a cingulum rest **over an** incisal rest are: **1)** It is more esthetic; **2)** The resulting stress relayed to the abutment has a less torquing influence.

Important: The **primary purpose** of the rest *(any type → occlusal, cingulum or incisal)* is to provide **vertical support** for the removable partial denture.

When designing a clasp assembly for the abutment tooth of a distal extension RPD, on which part of the crown surface should the retentive and reciprocal clasp arms be located?

- Occlusal third
- Middle third
- Gingival third

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• **Gingival third**

Retentive components should be located as close as possible to the tooth's horizontal axis of rotation. It is easier to overstress the tooth support by clasping it near the occlusal surface. The distal extension moves up and down during function, indicating that a clasp design in the gingival third of the tooth that disengages the undercut during function would be less stressful to the periodontal support of the tooth.

The **clasp assembly** consists of a **retentive clasp arm** and a **reciprocal or stabilizing clasp arm,** plus any **minor connectors and rests** from which they originate or with which they are associated.

The functions of the **reciprocal clasp arm** of a removable partial denture include reciprocation, stabilization and auxiliary indirect retention *(bracing).* **Points to remember concerning reciprocal clasp arms:**

- In positioning cast clasps on abutment teeth, the horizontal undercut is considered a significant measurement and height of contour is considered a controlling factor in clasp positioning. *(Reciprocal clasp should contact tooth on or above contour).* **See note below.**
- In general, you should not use retentive areas on the buccal and lingual of the same tooth. **Reciprocal bracing on the lingual and retentive portion of the clasp on the buccal is more desirable.**
- As with all clasps, they should be designed to permit insertion and removal without applying excessive force.

Notes:

1. Altering the natural tooth form to allow effective clasping may involve **producing guiding planes or changing the location of the height of contour.** Facial and proximal contours of premolars and molars most often need to be altered. Crown fabrication may be necessary to provide the appropriate contour.
2. **Guiding planes** serve to ensure predictable clasp retention. Failure of partials due to poor clasp design can be avoided by altering tooth contours.

Which of the following is defined as **"The quality of a restoration to be firm, steady, constant and not subject to change of position when forces are applied"?**

- Retention
- Stability
- Adhesion
- Reciprocation

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- **Stability**

In dentures, stability is the relationship of the denture base to bone that resists dislodgement of the denture in a horizontal direction. In **removable partial dentures** stability is best insured by incorporating a harmonious occlusion.

Retention is that quality in a restoration, which resists the force of gravity, sticky foods and forces associated with mandibular movement. **Note:** For RPD's the distal parts of the retentive clasps produce the active retention.

Reciprocation is the means by which one part of a restoration is made to counter the effects created by another part. **Note:** For RPD's true reciprocation can only be achieved if the reciprocating element touches the tooth before the retentive clasp.

For partial dentures, **support** is given by occlusal rests and the edentulous ridge areas. This design characteristic *(support)* is **most important** to oral health. Other design characteristics of a partial denture include:

- **Retention** → by clasps placed in undercut areas of abutment teeth
- **Bracing** → or horizontal force transmission through placement of rigid portions of clasps or other parts of the partial denture in non-undercut areas of abutment teeth.
- **Guidance** → during insertion and removal obtained by contact of rigid parts of the framework with areas on axial tooth surfaces parallel to the path of insertion.

A patient of yours walks into your office with the following complaint. "When I smile, my upper denture doesn't hold". Which area of the denture base needs to be adjusted?

- Labial notch and labial flange
- Buccal notch and buccal flange
- Posterior border
- Distobuccal flange

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• **Buccal notch and buccal flange**

Excessive thickness of this area can lead to this problem. As the buccal frenum moves posteriorly during smiling *(or other facial expressions)* it encroaches on the denture border that is too thick and the denture becomes loosened.

You can test the borders for **overextension** by slowly seating the denture. If you observe premature contact with frenae or vestibular tissues as the denture continues toward its final position, then the border is probably overextended. Adapt a thin roll of disclosing wax to the denture border. Seat the denture and instruct the patient to exert vigorous muscle function. In about one minute, the wax will soften and be displaced by muscular action across the overextended denture border.

Notes:

1. The complaint that the denture becomes loose when the mouth is wide opened as in yawning, could be due to the **distobuccal flange** of the denture being too thick. This may interfere with the movement of the coronoid process.
2. If patient complains of sore gums and aching muscles at the bottom of the face after wearing dentures for several hours, opposing teeth of the denture have insufficient space. **Reduce the vertical dimension of the occlusion.**
3. Tingling or a numbing sensation at the corner of the mouth or in the lower lip after a few days of denture wearing is caused by **excessive pressure from the lower buccal flange in the region of the mental foramen.**

Free end saddles are liable to be displaced under occlusal pressure *(anteroposterior rocking around the abutment tooth, which acts as a pivot)*. This is as a result of the displaceability of the mucosa. Which technique is employed to try and prevent this by taking an impression of the mucosa under controlled pressure?

- The functional load technique
- The altered cast technique
- The residual ridge technique
- The total occlusal load technique

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• **The altered cast technique**

Impression materials **cannot** record anatomic form of the teeth and physiologic form of the soft tissue in a functional relationship all at the same time. To achieve these objectives, the altered cast technique can be used. This technique is a secondary impression system which utilizes the metal framework to hold customized impression trays for the edentulous area. The **advantage** of the altered cast procedure is that an accurate relationship between the denture base and the metal framework is established prior to tooth arrangement which should result in less occlusal adjustment at the time of insertion. The **objectives** of the altered cast technique are to obtain the maximum possible support from the distal extension base of the RPD and to accurately relate the soft tissue surface of the denture base to the metal framework

Distal extension removable partial dentures *(RPD's)* derive their support from the abutment teeth and the mucosal tissues overlaying the residual alveolar process. There are differing philosophies in the scientific literature regarding how much support should be provided by the abutment teeth and how much support should be provided by the soft tissues. However, there is consensus that: **(1)** occlusal stress should be shared by both in such a manner that neither the abutment teeth nor the residual ridge is abused; **(2)** accurate fit of the denture base is an important factor in minimizing stress on the abutment teeth; **(3)** stability of the prosthesis is the most important requirement for proper function and patient comfort.

Important points to **remember** concerning removable partial dentures:

- In order to determine whether the **alveolar bone** is capable of withstanding occlusal forces of a removable partial denture, an x-ray should be taken of the abutment teeth and the bone level surrounding these teeth should be evaluated.
- Periodontal health of the **abutment teeth** and maintaining the health of the supporting tissues is best achieved by **maintaining tissue support** *(preserving denture bone support)* of the edentulous areas.
- The **total occlusal load** applied to an RPD is influenced by the occlusal surface area, occlusal efficiency and the number of existing teeth.

Most designs of stressbreakers will rather effectively dissipate vertical forces to terminal abutments. However, this occurs at the expense of what supporting entity?

- The periodontal ligament of the abutment teeth
- The alveolar support of the abutment teeth
- The residual ridge
- All of the above

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• **The residual ridge**

When these devices are incorporated next to a **free-end distal extension RPD,** the thrust of the functional **stress is directed onto the residual ridge.** Only minimal transfer of functional stress to the abutment teeth occurs. Since vertical and horizontal forces are concentrated on the residual ridge, **increased ridge resorption** frequently occurs. Relining of the free-end saddle area **must be done** when needed to prevent excessive ridge resorption.

Types of Stressbreakers

1. Have a **flexible connection** between the direct retainer and the denture base:
   - **Simplest form** of stress relief is the **wrought-wire** retentive clasp
   - **Split bar major connectors;** example is the Ticonium **"Hidden-Lock"** design
2. Have a **movable joint** between the direct retainer and the denture base:
   - The **"DE"** hinge
   - The **Dalbo** attachment
   - The **Crismani** attachment
   - The **ASC-52** attachment

Note: When a stressbreaker **is placed** on the distal surface of a pontic, occlusal forces will tend to **unseat** the key from the key.

All of the following are **advantages** of precision attachment restorations **except:**

- They provide retention without an unsightly display of metal
- They are easy to repair
- The functional load is dispersed down the long axis of the abutments by virtue of the low central loading at the base of the attachments
- The restorations permit the patient access to all areas of the tissues when the denture is not in place
- If both sides of the dental arch have this type of restoration and are joined by a rigid major connector, excellent bilateral stabilization is provided to the abutments

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• **They are easy to repair**

***This is **false;** precision attachment restorations are **difficult to repair.**

A precision attachment is preconstructed with **male** and **female** portions *(both constructed of a metal)* that fit together in a precise fashion with little tolerance. It may be rigid in function or it may incorporate a movable stress control unit to reduce the torque on the abutment.

Disadvantages of precision attachments:

- They must **never be used** in a distal extension removable partial denture **without** using a stressbreaker→ the primary indication for precision attachments is when teeth are present on both ends of the edentulous area
- Full cast crowns **must be** prepared for all abutments
- They **cannot** be used on short clinical crowns
- They **cannot** be used when the pulp is large *(requires extensive tooth reduction)*
- Both clinical and laboratory procedures require **special skill**
- They are **difficult** to repair
- The metal parts **wear** and **lose** retention
- The **cost** is much **greater**

A **semiprecision attachment** is cast into the crown and the RPD. The female portion is normally made of preformed plastic that is positioned into the wax form and then cast. The male portion is cast with the RPD framework. The female and male parts fit together with much more tolerance than in the precision attachment, resulting in less retention.

When **surveying casts,** the clinician / technician must perform an important step in order to correctly record the path of insertion, the position of the survey line and the location of undercut and non undercut areas. Which of the following is considered to be that step?

- The recontouring of proximal walls of abutments parallel to the path of insertion
- The use of indelible lead marker
- Placing tripod marks on the cast to record the orientation of the cast to the surveyor
- Fixing the casts with screws on the surveying table
- All of the above steps are equally important

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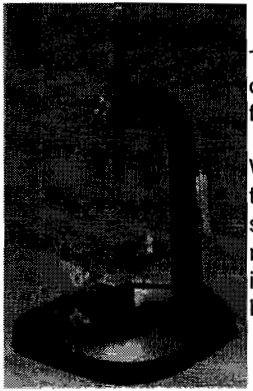
• **Placing tripod marks on the cast to record the orientation of the cast to the surveyor**

The **tripod marks** *(which are three spots placed at three different locations around abutment teeth from a single point of view)* ensure **reproducible orientation** of the cast to the surveyor.



The **dental surveyor** *(picture on left)* is an instrument used to determine the relative parallelism of oral anatomy. **Note:** Areas used for support **cannot** be determined by surveying.

When surveying casts, the correct procedure is to **first** adjust the tilt to permit the establishment of guiding planes. The anterior edentulous space will frequently dictate the angulation selected. Normally, some recontouring of the **proximal walls** of abutments is necessary to improve guideplane alignment. These alterations are accomplished by disking the proximal surface parallel to the path of insertion.

Tilting of the cast during surveying changes the:

- Path of insertion
- Position of the survey line
- Location of the undercut and non-undercut areas of each tooth

Which primary design-quality of the occlusal rest would categorize it as a **"positive"** rest?

- Allow no tilting of the appliance
- Prevent the movement of the appliance
- Transmit stress down the long axis of the tooth
- Form acute angles with the minor connectors that connect them to the major connectors
- Have a thickness of 1.5 mm
- All of the above

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- **Form acute angles with the minor connectors that connect them to the major connectors** → this defines the positive rest and also permits maximum bracing

The **primary purpose** of the rest is to provide vertical support for the RPD. *(***Note:** *Occlusal rests are prepared primarily to resist the* **vertical forces** *of occlusion).* In doing so the rest also does the following:

- It maintains established occlusal relationships by preventing settling of the denture
- Prevents impingement of soft tissues
- Directs and distributes occlusal loads to abutment teeth

Form of the occlusal rest and rest seats:

1. The outline form should be a **"rounded"** triangular shape with the apex toward the center of the occlusal surface.
2. It should be as long as it is wide and the base of the triangular shape should be at least **2.5mm** for both molars and premolars.
3. The marginal ridge of the abutment tooth at the site of the rest seat must be lowered to permit a sufficient bulk of metal for strength and rigidity. This means that a reduction of the marginal ridge of about **1.5mm** is usually necessary.
4. The floor of the occlusal rest should be apical to the marginal ridge and be concave or spoon shaped *(no sharp edges or line-angles in the prep).*
5. The angle formed by the occlusal rest and the vertical minor connector from which it originates should be **less than 90°.**

Remember: The rest must be **rounded** *(spoon shaped)* to permit functional movement.

The **size of posterior teeth** for a removable partial denture is determined primarily by which **two** of the following?

- The useful posterior tooth space
- The lip line of the patient
- The age of the patient
- The characteristics of the denture-supporting tissues
- The face-bow transfer

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- **The useful posterior tooth space**
- **The characteristics of the denture-supporting tissues**

Factors which are relevant to the selection of **posterior teeth** for a removable partial denture:

- **Occlusogingival length** → the most important factor in determination of posterior tooth length is the **available interarch space.**
- **Mesiodistal width** → the total mesiodistal space available for the posterior teeth is determined by **measuring from the distal of the lower canine to the point where the mandibular residual ridge begins to slope upward.**
- **Buccolingual width** → the buccolingual width **is narrowed in relation to the missing natural tooth.** It is thought that reducing the area of the occlusal table decreases stressed transferred to the denture support area during food bolus penetration. Additionally, reducing the buccolingual width increases tongue space.
- **Shade** → the shade for posterior teeth is usually selected to **harmonize** with that of the anteriors.
- **Occlusal surface form** → at this time, it appears that no superior tooth form or arrangement is identified. It is therefore logical to use the **least complicated approach** that fulfills the needs of the patient.
- **Material** → **plastic bonds well** to the acrylic resin and therefore plastic teeth are retained better than porcelain teeth.

Dr. Blandi requested that you mix alginate and take an impression. While measuring the water, you got involved in a conversation with your patient and did not notice how cold it was. This oversight will:

- Shorten the gelation time
- Make the mix unusable
- Lengthen the gelation time
- Not affect the gelation time

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• **Lengthen the gelation time**

***The **best method** to control the gelation time of alginate impression materials is to alter the temperature of the water used in the mix. The **higher** the temperature, the **shorter** the gelation time, the **lower** the temperature, the **longer** the gelation time. The mix is usable regardless of water temperature as long as there is adequate working time.

Changing the **water / powder ratio** and the mixing time **will alter** the gelation time, but these methods also **impair** certain properties of the material. Too little or too much water will **weaken** the gel. Undermixing may prevent the chemical action from occurring evenly; overmixing may break up the gel.

Calcium sulfate *(the reactor in alginate),* is not so soluble in water that is entirely consumed before gelation is completed. Therefore, the set mass becomes an entanglement of calcium alginate fibrils around residual sodium alginate sol, filler and water. The residual sodium alginate has the nasty habit of readily giving up water *(syneresis)* or gaining water *(imbibition).* For accurate results, the cast should be poured immediately.

Notes:

1. When taking an alginate impression for a **partial denture,** it is best to apply some alginate directly on the teeth to eliminate bubbles and saliva from the rest seat preparations.
2. Inaccuracies in impressions can be caused by **fracture** of the fibrils during gelation.

All set impressions are **rinsed with water** thoroughly to remove residual saliva from the surface of the impression *(or rinse with a watery mix of dental stone before pouring up the impression).*

Which of the following is **not** a characteristic of **polyether** impression materials?

- The working and setting times are shorter than those for polysulfide materials; they are more like those of addition silicone materials
- Slow setting
- Dimensionally stable if more than one cast is poured
- Excellent dimensional stability when dry
- They are very stiff compared to other materials
- They are clean materials to use but have an unpleasant taste
- They are very accurate and easy to pour with gypsum products

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• **Slow setting** → this is **false;** they set very quickly

***Polyethers** are two-component materials. The base includes a polyether, silica filler and a plasticizer. The accelerator contains a cross-linking agent. When mixed, a rubber is formed by a **cationic polymerization** process. Cationic polymerization is very similar to addition polymerization, except that instead of a free radical, a cation *(positive ion)* is the reactive molecule. No reaction by-product is produced. Polyethers have excellent dimensional stability. They are also truly hydrophilic, resulting in superior wettability.

Polyether Impression Materials	
Advantages	**Disadvantages**
• Excellent dimensional stability **when dry** • **Fast setting** • Dimensionally stable if more than one cast is poured • Stable even if poured 24 hours after making impression • Very accurate & easy to pour with gypsum products	• Difficult to remove *(most rigid or stiff)* • Tears easily • May adhere to teeth • High water absorption • Fine margins may break • Unpleasant taste

Examples → Impregum *(Premier)* and Polygel *(Caulk).*

Polyether materials are **dimensionally unstable** in the presence of moisture. These materials are the **most rigid** *(stiffest)* and **most difficult** to remove from the mouth.

Which of the following impression materials has a "putty / reline" form, which affords it ***delayed* pouring** of up to six hours?

- Polysulfides
- Silicones
- Polyvinyl siloxanes
- Polyethers

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• **Silicones** *(also called conventional silicones or condensation silicones)*

These materials record surface **detail well** and have excellent elastic properties **but** a low tear strength. They are less expensive than polyvinyl siloxanes *(addition silicones)* and polyethers.

Indications: Complete dentures, crown & bridge
Examples → Speedex *(Coltene / Whaledent)* and Primasil *(TISS Dental)*
Composition:

• **Base** - Poly *(dimethylsiloxane)*
- tetraethylorthosilicate
- filler (silica)

• **Catalyst** - metal organic ester

• **By-product** - ethyl alcohol

Silicones	
Advantages	**Disadvantages**
• Better elastic properties	• Poor dimensional - high shrinkage * polymerization * evaporation of ethanol - poor immediately *(within 30 minutes)*
• Clean, pleasant taste	• Hydrophobic → poor wettability
• Stock tray	
• Good working and setting time	

Why poor dimensional stability? The principal reaction, which takes place during setting of this material, is a **condensation reaction** and hence called condensation silicone. It occurs by elimination *(evaporation)* of ethyl or methyl alcohol. This is also responsible for shrinkage of the material and resultant poor dimensional stability.

Notes:

1. Reaction is sensitive to **heat** and **moisture** *(will reduce working and setting times).*
2. **Do not** mix initially by hand *(allergic reaction to catalyst may occur).*

Which of the following is an example of an **inelastic** impression material?

- Polysulfide
- Alginate
- Addition silicone
- ZOE impression paste

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• **ZOE impression paste** → sets into a hard, brittle mass *(this makes it inelastic)*

Remember: ZOE pastes **differ** from elastic impression materials in that they set hard in the mouth *(use only when there are no deep undercuts of soft tissues)*. This limits their use to impressions of edentulous ridges from removable dentures.

Zinc Oxide-Eugenol Impression Pastes	
Advantages	**Disadvantages**
• Can record soft tissue at rest • Sets hard • Setting time in 5 minutes • Not as expensive as polysulfides • Stable	• Messy to mix, very sticky • Tissue irritant • Not elastic • More difficult to manipulate • Not recommended for the gagging patient

Notes:

1. The dimensional stability of a zinc oxide-eugenol impression is **most likely** to be affected by failure to use a custom-made impression tray.
2. The setting time of a zinc oxide-eugenol impression paste may be **accelerated** by adding a drop of water to the mix.
3. The setting time of a zinc oxide-eugenol impression paste may be **retarded** by adding inert oils *(olive or mineral oils)* during mixing.

The popularity of agar impression material *(reversible hydrocolloid)* is limited by the:

- Difficulty in pouring the impression
- Poor reproduction of detail
- Need for special equipment
- High cost

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• **Need for special equipment**

The use of agar impression material does require special equipment. The reproduction is excellent, and the impression is easy to pour compared to elastomeric impression material.

A **reversible hydrocolloid** is an impression material whose **physical state** can be changed from a **gel to a sol** by the application of heat and back again by withdrawing heat.

Indications: crown & bridge → high accuracy
Example → Slate Hydrocolloid *(Van R)*

Composition of reversible hydrocolloids:

- 12-15% agar *(complex polysaccharide → seaweed)*
- Traces of borax *(for strength)*, potassium sulfate *(improves gypsum surface)* and sodium tetraborate
- 85% water

Advantages:

- No custom tray or adhesives
- Hydrophilic → displaces moisture, blood, fluids
- Clean and pleasant with acceptable odor
- Excellent shelf life
- Inexpensive *(after initial equipment)*
- Does not require mixing

Disadvantages:

- Initial expense of special equipment
- Material prepared in advance
- Tears easily
- Dimensionally unstable
  - immediate pour, single cast
- Difficult to disinfect

The filler in alginate gives the mixed material "body" that allows acceptable handling. Without filler, the mixed material would be too runny for use. Which of the following is a main component of the alginate powder and functions as the filler?

- Zinc oxide
- Calcium sulfate
- Potassium titanium fluoride
- Diatomaceous earth *(silica)*
- Potassium alginate
- Tri-sodium phosphate

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• **Diatomaceous earth** *(silica)*

Alginate materials are the most widely used impression materials. They are termed irreversible impression materials because they will not reverse to a sol once they react and become a gel.

Indications: study models, RPD's → framework
Examples → Jeltrate *(Dentsply / Caulk)*, COE Alginate *(GC America)*

Chemical Constituents of an Alginate Powder	
Constituents	**Percentage *(Action)***
• Diatomaceous *(Silica)*	• 50% *(filler)*
• Potassium alginate	• 20% *(dissolves in water forming the sol)*
• Calcium sulfate	• 16% *(reactor)*
• Zinc oxide	• 7% *(plasticizer)*
• Potassium fluoride	• 6% *(improves gypsum surface)*
• Sodium phosphate	• 1% *(retarder)*

Irreversible Hydrocolloid	
Advantages	**Disadvantages**
• Inexpensive	• Unstable → immediate pour, single cast
• Can use stock tray	• Tears easily
• Hydrophilic, displaces moisture, blood, fluids	• Low detail reproduction
• Easy to use	• High permanent deformation

ZOE impression material:

- Will spring back from undercuts
- Has its set retarded by water
- Should be mixed to a uniform color
- Sets by cooling the material

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• **Should be mixed to a uniform color**

Typically the two pastes of ZOE are different colors. The pastes are swirled, stropped, and scraped together during mixing until one homogeneous color is obtained.

One of the dentally useful chemical reactions is that between **zinc oxide** and **eugenol.** Under the proper conditions, a relatively hard mass is formed, which possesses certain medicinal advantages as well as a mechanical usefulness in certain dental operations.

The basic composition of all these materials *(whether it be a cementing medium, surgical dressing, temporary filling material or impression paste)* is the same: mainly **zinc oxide, eugenol and resin.** Plasticizers, fillers, accelerators and other additives are incorporated as necessary to provide the desired properties for the particular use of the product.
Note: The fixed vegetable or mineral oil present in a tube of zinc oxide acts as a plasticizer when it is mixed with eugenol. ***It also aids in masking the action of eugenol as an irritant.

The setting reaction that occurs is a typical acid-base reaction to form a **chelate.** This reaction can take place either in solution or at the surface of the zinc oxide particles. The chelate is thought to form as an amorphous gel that tends to crystallize, imparting increased strength to the set mass.

Remember: Moisture *(H_20)* will **accelerate** the set of the paste.

You are taking an impression with alginate and unbeknownst to you there is debris on the tissue. How will this effect the final impression?

- It will be grainy
- It will tear easily
- There will be irregularly shaped voids
- It will be distorted

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- **There will be irregularly shaped voids**

Problem	Cause
Grainy material	• Improper mixing • Prolonged mixing • Undue gelation • Water/powder ratio too low
Tearing	• Inadequate bulk • Moisture contamination • Premature removal from mouth • Prolonged mixing
Irregularly shaped voids	• Moisture or debris on tissue
Rough or chalky stone cast	• Inadequate cleaning of impression • Excess water left in impression • Premature removal of cast • Leaving cast in impression too long • Improper manipulation of stone
Distortion	• Impression not poured immediately • Movement of tray during gelation • Premature removal from mouth • Improper removal from mouth • Tray held in mouth too long *(only with certain brands)*

Which component of **zinc oxide-eugenol impression paste** facilitates the speed of the reaction which results in a smoother, more homogenous product?

- Oil of cloves
- Resinous balsam
- Rosin
- Calcium chloride

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- **Rosin**

Other **components** of ZOE impression paste:

- **Calcium chloride** *($CaCl_2$)* → functions as an accelerator of the setting time

- **Oil of cloves** → contains 70-85% eugenol. It is sometimes used in preference to eugenol because it reduces the burning sensation in the soft tissues of the mouth.

- **Mineral or fixed vegetable oil** → plasticizer, aids in masking the action of eugenol as an irritant.

- **Resinous balsam** → often used to increase flow and improve mixing properties.

Note: If the paste is **too thin** or **lacks body** before it sets, a filler → such as a wax or an inert powder *(lanolin, kaolin, etc.)* may be added to one or both of the original pastes.

ZOE impression materials were once very popular. Today, however, ZOE materials have been replaced by newer materials, such as addition silicones.

Polyvinyl siloxane impression materials:

- Set by opening of the Si=O bond
- Are also known as condensation silicones
- Should be teased from around the teeth to prevent tearing
- Are also known as addition silicones

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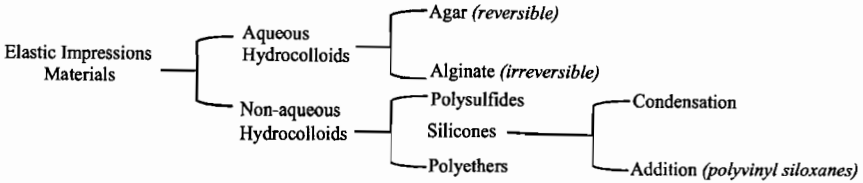
- **Are also known as addition silicones**



Polysulfide rubber base, silicones *(condensation silicones)*, polyethers and polyvinyl siloxanes *(addition silicones)* are **non-aqueous** polymer-based rubber impression materials that have good elasticity. Reversible hydrocolloid *(agar)* and irreversible hydrocolloid *(alginate)* are **aqueous** hydrocolloids.

Polyvinyl siloxanes *(addition silicones)* are the **most widely used** and are the **most accurate** of the elastic impression materials. They have **less** polymerization shrinkage, **low** distortion, **fast** recovery from deformation and a **moderately high** tear strength. Most of the polyvinyl siloxanes can be poured up to one week after impression making and are stable in most sterilizing solutions. **Important:** The **sulfur** in latex gloves and in ferric and aluminum sulfate retraction solution **will retard** the setting of addition silicone materials. Also, addition silicones are **temperature sensitive** → increases in temperature will shorten the working and setting times.

Examples → Extrude *(Kerr)*, Express *(3M/ESPE)*, Aquasil *(Dentsply Caulk)*, Genie *(Sultan Chemists)*, Virtual *(Ivoclar Vivadent)*
Note: Reversible and irreversible hydrocolloids have the **advantage of wetting** intraoral surfaces well, but they have **very limited dimensional stability** because they include as much as 85% water in their composition.

Place the following **non-aqueous** elastic impression materials in order from best to worst in regards to **dimensional stability.**

- Polyether
- Polysulfide
- Condensation silicone
- Addition silicone

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- **Addition silicone > polyether > polysulfide > condensation silicone**

Comparison of Properties	
Cost	**- lowest to highest** • alginate < agar = polysulfide < condensation < addition silicone < polyether
Dimensional stability	**- best to worst** • addition silicone > polyether > polysulfide > condensation silicone > hydrocolloid
Wettability	**- best to worst** • hydrocolloids > polyether > hydrophilic addition silicone > polysulfide > hydrophobic addition silicone = condensation silicone
Castability	**- best to worst** • hydrocolloids > hydrophilic addition silicone> polyether > polysulfide > hydrophobic addition silicone = condensation silicone
Stiffness	**- most to least** • polyether > addition silicone > condensation silicone > polysulfide = hydrocolloids
Tear strength	**- greatest to least** • Polysulfide > addition silicone > polyether > condensation silicone > hydrocolloids

Place the following **elastic** impression materials in order from longest to shortest in regards to **working time.**

- Polyether
- Polysulfide
- Agar *(reversible hydrocolloid)*
- Alginate *(irreversible hydrocolloid)*
- Silicones

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• **Agar > polysulfide > silicones > alginate = polyether**

Handling Properties						
	Agar	**Alginate**	**Polysulfide**	**Condensation Silicone**	**Addition Silicone**	**Polyether**
Preparation	Boil, temper., store	Powder, water	2 pastes	2 pastes or paste/liquid	2 pastes	2 pastes
Ease of Use	Technique sensitive	Good	Fair	Fair	Excellent	Good
Patient Reaction	Thermal shock	Pleasant, clean	Unpleasant, stains	Pleasant, clean	Pleasant	Unpleasant, clean
Ease of Removal	Very easy	Very easy	Easy	Moderate	Moderate	Moderate to difficult
Disinfection	Poor	Poor	Fair	Fair	Excellent	Fair
Working Time *(min.)*	7-15	2.5	5-7	3	2-4.5	2.5
Setting Time *(min.)*	5	3.5	8-12	6-8	3-7	4.5
Stability	1 hour 100% RH	Immediate pour	1 hour	Immediate poor	1 week	1 week kept dry
Wettability & Castability	Excellent	Excellent	Fair	Fair	Fair to good	Good
Cost	Low	Very low	Low	Moderate	High to very high	Very high

All **elastomeric** impression materials:

- Expand initially and then contract during curing
- Expand slightly during curing
- Contract slightly during curing
- Contract initially and then expand during curing

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• **Contract slightly during curing**

– **Elastomers** are impression materials that have elastic or rubber-like qualities.
– **Alginate** is an irreversible hydrocolloid and **agar** is a reversible hydrocolloid.

Impression Materials	
Non-elastic	- Plaster - Compound—Waxes—ZnO - Eugenol
Elastic	**- Aqueous hydrocolloids** • Agar • Alginate
	- Non-aqueous elastomers • Polysulfide • Silicones - Condensation - Addition *(polyvinyl siloxanes)* • Polyether

There are **two basic types** of polymerization, which is the process of changing elastomeric materials from a paste into a rubber-like material. **Addition polymerization** involves the adding of the units on each side of the carbon-carbon double bond. No ionic forms are involved, as a rule, with the entire reaction sequence being carried out by free-radical chemical species. **Condensation polymerization** often involves ionic species and produces a small molecule *(usually water of an alcohol)* as a **by-product** of each step of the reaction.

Some uses of elastomers include crown and bridge, secondary impressions for dentures and inlays / onlays.

When **removing** elastomeric impressions, use a steady force, however, a snap is not required. → **minimizes permanent deformation.** The following impression materials set by way of a **chemical reaction,** as do gypsum products: alginate, elastomeric materials, and ZOE. The setting of agar impression materials and dental compound **does not** involve a chemical reaction.

Some impression materials are most accurate when at least 3 mm of space is present between the impression tray and the oral tissue. Which of the following impression material types shows this characteristic?

- Polyethers
- Polysulfides
- Silicones
- Irreversible hydrocolloids

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• **Irreversible hydrocolloids** → alginate

Irreversible hydrocolloids *(alginate)* is most accurate when at least 3 mm of space exists between the impression tray and the tissue. The other impression types are most accurate when a small but definite space exists between the impression tray and the tissue.

Remember: The setting time of alginate is controlled by the **amount** of sodium phosphate that is present. **Sodium phosphate** serves a **retarder** in this reaction, which means it slows down the process. As long as sodium phosphate is present, it will react with soluble calcium ions. Once all the sodium phosphate has reacted, then the sodium alginate reacts with the remaining calcium ions and calcium alginate is formed.

$$2\,Na_3PO_4 + 3\,CaSO_4 \longrightarrow Ca_3(PO_4) + 3\,Na_2SO_4$$

$$\text{Na alginate} + CaSO_4 \xrightarrow{H_2O} \text{Ca alginate} + Na_2SO_4$$

Notes:

1. **Fast** removal of impression from the mouth **increases** both the compressive and tear strength of the impression.
2. All impressions must be rinsed and disinfected **prior** to pouring or sending to the laboratory. **Soak** or **spray** for a minimum of **10 minutes. Important:** Always follow the manufacturer's recommendation for the specific product!!!

Today was a very busy day for Ashley, the dental hygienist in our office. Ashley took alginate impressions on her first patient in the morning, who needed a nightguard. Since she was so busy, Ashley left the alginate impressions in the lab most of the morning. Ashley decided to place the impressions in a bowl of water so that they would not dry up before she had a chance to pour them up in dental stone. Which of the following was the result of Ashley leaving these impressions immersed in water for a few hours?

- Gelation
- Hysteresis
- Syneresis
- Imbibition

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• **Imbibition**

Imbibition occurs when the impression absorbs water, which expands the dimensions of the impression. When this occurs, the impression is no longer accurate. Shrinkage will occur in alginate impressions, even when they are placed under 100% relative humidity. The shrinkage and exudation of water is called **syneresis.**

Since shrinkage is **undesirable** *(causes distortion of impressions),* alginate impressions **should not** be left either in water *(will expand)* or exposed to air *(will shrink)*. They should be poured immediately after they are taken to ensure accuracy. When immediate pouring is not possible, they may be stored **briefly** in a moist paper towel.

Important: While taking an impression with alginate, it is advisable that the tray be placed in the mouth after all critical areas are wiped with alginate. Critical areas are buccal to the maxillary tuberosities and retromylohyoid space. Rest seats and guide planes should be covered with alginate as well as any other soft tissue undercuts.

Notes:

1. **Gelation** is the term given to the setting process *(changing a from sol to a gel)* of hydrocolloid material.
2. **Hysteresis** refers to a material's characteristic of having a melting temperature different from its gelling temperature.

If your patient indicates a tendency to gag while taking alginate impressions, all of the following maneuvers can help **except:**

- Lessening the time to take an impression
- Using cold water to mix the alginate
- Having the patient breathe through his / her nose
- Seating the patient in an upright position
- Seating the posterior portion of the tray first

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• **Using cold water to mix the alginate**

Cold water will make the alginate take **longer** to set.

Mixing the alginate material **rapidly** will cause setting to occur **more rapidly.**

Decreasing the water to powder ratio will cause alginate to set up **more rapidly** *(affects consistency of the mix → mix is much thicker when less water is used).*

Note: The **mandibular** alginate impression is taken **first** since gagging is more likely to occur when taking the maxillary impression. For the maxillary impression, the posterior portion of the tray is seated first, then the anterior portion. This helps to prevent the alginate material from being squeezed out of the tray, back toward the patient's throat *(which may cause gagging).*

Always remove alginate impressions in **one quick movement,** with a snap. This helps to decrease permanent deformation.

Notes:

1. The setting reaction of alginate is a **"double decomposition"** reaction between potassium alginate and calcium sulfate. Also remember that casts must be poured within 24 hours and the impression must be kept damp.
2. Both under and over-mixing can reduce the strength of the impression.
3. Do not over-seat the tray → 0.25 inch *(minimum)* of alginate should remain over all critical structures *(especially occlusal surfaces).*

Characteristics of **polysulfide** rubber base impression materials include all of the following **except:**

- Moisture accelerates set
- Sets by S–S crosslinking
- Short setting time
- Strong odor
- Deform readily when removed from undercut areas
- Good tear resistance

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• **Short setting time**

***The setting time of polysulfide rubber base impression materials is long *(8 – 12 minutes)* after placement in the mouth.

Polysulfide impression material was the first non-aqueous elastomeric "rubber" impression material developed for dentistry. These materials set via a condensation reaction.

Mercaptan + lead dioxide → → → polysulfide rubber + lead dioxide + water

Examples → Permlastic *(Kerr)*, Omni-Flex *(GC America)*

Properties						
	Agar	**Alginate**	**Polysulfide**	**Condensation Silicone**	**Addition Silicone**	**Polyether**
Elastic Recovery *(%)*	98.8	97.3	95.5-96.9	98.2-99.6	99-99.9	98.3-99.0
Flexibility *(%)*	11	12	8.5-20.0	3.5-7.8	1.3-5.6	1.9-3.3
Flow	—	—	0.4-1.9	<0.10	<0.05	<0.05
Shrinkage, 24 hours *(%)*	Extreme	Extreme	0.4-0.5	0.2-1.0	0.01-0.2	0.2-0.3
Tear Strength *(g/cm)*	700	380-700	2240-7410	2280-4370	1640-5260	1700-4800

The **brown paste** used in polysulfide rubber base is called the:

- Filler
- Base
- Polymer
- Accelerator

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- **Accelerator**

Categories of Elastomeric Impression Materials

- **Polysulfides** *(also called rubber base, mercaptan or Thiokol)* → the white **"base"** paste contains a low-molecular-weight polysulfide polymer *(mercaptan)* mixed with an inorganic filler, such as titanium oxide. The brown **"accelerator"** paste contains **lead dioxide** and an "oily" organic chemical that does not react. Sulfur is also included in the brown paste because it promotes the polymerization reaction. Water is the reaction by-product.
- **Silicones** *(also called conventional silicones or condensation silicones)* → the base is a liquid silicone polymer *(dimethyl siloxane)* mixed with inert fillers. The reactor consists of a cross-linking agent, **ethyl ortho-silicate,** with an activator, **tin octoate.** The materials are cross-linked by a reaction between terminal hydroxyl groups on the polymer and ethyl ortho silicate. Ethyl alcohol is a **by-product** of the setting reaction. The evaporation of this alcohol is believed to be responsible for shrinkage of the material and resultant poor dimensional stability.
- **Polyvinyl siloxanes** *(also called vinyl polysiloxanes or addition silicones)* → one tube contains **silicone** with terminal silane hydrogen groups and an inert filler. The other tube is a **vinyl silicone** with terminal vinyl groups, a chloroplatinic acid catalyst and a filler. Upon mixing, there is an addition of silane hydrogen groups across vinyl double bonds with the formation of **no by-products.** The result is a **very dimensionally stable material.**
- **Polyethers** → the base consists of the **polyether polymer** with ethylene imine groups and the accelerator contains the **aromatic sulfonic acid ester.** The aromatic sulfinate produces cross-linking by cationic polymerization.

Custom trays are an important part of rubber base impression techniques, since **elastomers** are:

- More accurate in uniform, thin layers 0.5 to 1.0 mm thick
- More accurate in uniform, thin layers 1.0 to 1.5 mm thick
- More accurate in uniform, thin layers 2.0 to 4.0 mm thick
- More accurate in uniform, thin layers 5.0 to 6.0 mm thick

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• **More accurate in uniform, thin layers 2.0 to 4.0 mm thick**

With all elastomers, a **custom tray** should be fabricated with a plastic material. This tray should be **rigid,** have **occlusal stops** to avoid permanent distortion during polymerization and be **coated with an adhesive.** With hydrocolloid impressions *(alginate)*, a greater bulk of material produces greater accuracy, however, the thickness of rubber-like materials should not only be less, but should be evenly distributed. **Important:** Let adhesive that is applied to the tray dry completely. If it is wet, impression material may pull away.

Custom trays are recommended for the following reasons:

- They require **less** impression materials
- They facilitate **uniform concentration** of the impression materials
- Stock trays usually are short in the flange area
- With stock trays, the uneven bulk of the impression material is conducive to distortion

The accuracy and reliability of an elastic impression is **controlled by the tray** in which it is taken. The **best tray** is one that is custom-made for each patient. In most cases, it is best to take a complete arch impression, which will provide maximum reliability.

The shade of the ceramic crown should be matched by what order of color characteristics?

- Hue, chroma, value
- Chroma, hue, value
- Chroma, value, hue
- Value, hue, chroma
- Value, chroma, hue

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• **Value, chroma, hue**

Value *(brightness of color)* being the most critical characteristic is matched first followed by; **chroma** *(the strength / saturation of the color);* and finally **hue** *(the basic color).*

Shade selection sequence:

- Use the same **shade** guide as given by the manufacturer
- **Match** the shade before you do any preparation of the tooth
- **Remove** all distractions *(e.g., lipstick, dark glasses, heavy make-up, etc.)*
- Quick rubber cup and paste prophylaxis can make shade selection more accurate
- **Position yourself** between the patient and the light source
- When observing, **do not gaze** for greater than 5 seconds at a time. Prolonged gazing decreases the ability to discriminate colors and shades
- Proceed by process of elimination. Exclude first, shades which are too light or dark
- Half-closed eyes can increase the sensitivity of retinal rods to better choose the "value" of the color

Remember: "Blue" fatigue accentuates "yellow" sensitivity. This means that if you look at blue color objects *(drapes, charts, wall-color or any other object around)* while selecting the shade, it will help to accentuate the ability to discriminate yellow shades.

Sintering of a ceramic:

- Involves heating the raw materials above the melting point
- Results in an increase in porosity
- Decreases its mechanical strength
- Increases its density

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• **Increases its density**

Firing porcelain causes the powders to become "sintered". Sintering changes the porcelain from a powder to a solid. The powder is not melted, so the general shape is maintained. Sintering porcelain is the same process that is used to fire clay pots, china, and ceramic tiles. Reducing the porosity of the resulting product is very important. The less porous *(more dense)* the product is, the greater the strength of the final product will be. After sintering, the final shape of the restoration is refined by grinding.

The dental porcelain used in the fabrication of restorations **must have** the following properties:

- Low fusing temperature
- High viscosity
- Resistance to devitrification *(crystallization)*

Notes:

1. When porcelain is fired too many times, it may devitrify. This appears as a "milky state" and makes glazing very difficult.
2. Aluminum oxide is added to low-fusing dental porcelains *(during its manufacture)* in order to increase its resistance to "slumping down" during firing.
3. Glass, which is a prevalent phase in dental porcelain is amorphous and fragile.
4. The strength of a ceramic decreases with flaw size.
5. Ceramic restorations are severely damaged by acidulated fluoride.

All of the following are morphological changes associated with the edentulous state **except:**

- Deepening of nasolabial groove
- Loss of labiodental angle
- Retrognathic appearance
- Decrease in horizontal labial angle
- Narrowing of lips
- Increase in columella-phitral angle

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• **Retrognathic appearance** → a **prognathic appearance** is associated with the edentulous state.

It must be emphasized that one or more of these items are also frequently encountered in persons with intact dentitions because the compromised facial support of the edentulous state is not the exclusive cause of the morphological changes. Patient's weight loss, age, and heavy tooth attrition manifest oro-facial changes suggestive of compromised, or absent, dental support for the overlying tissues.

Pre-extraction guides for selecting artificial teeth from edentulous patients include:

- **Photographs** → provide general information about width and possibly outline form.
- **Diagnostic casts** → the form of the teeth can be very well judged from previous diagnostic casts of natural teeth, if available *(check with the patient's previous dentist)*.
- **Intra-oral radiographs** → the size and form can be determined but beware because radiographs can be distorted and usually are larger images of the teeth.
- **The teeth of close relatives** → when no other means are available to get an idea about the form, size and shade of teeth to be used for the denture of an edentulous patient, records of son's or daughter's teeth can give a clue. It may also help in the arrangement of teeth as well.
- **Extracted teeth** → sometimes patients keep their extracted teeth, which could be an excellent source and aid to delineate the form of the teeth, thus helping in the selection process.

Notes:

1. Degenerative joint disease is frequently seen in denture wearers but this may be age related rather than the state of the dentition.
2. The recording of centric relation is considered as an **essential starting point** in the design of the artificial denture.
3. In complete denture prosthodontics the position of the maximum planned intercuspation of teeth or centric occlusion, is established to coincide with the patient's centric relation.

There are several disadvantages associated with the use of base metal dental casting alloys. Which of the following is an **advantage** that base metal alloys have over the noble metal alloys?

- Base metal is much heavier *(denser)* than noble metal alloys
- Base metal is easier to cast and finish
- Base metal has a stronger porcelain bond
- Base metal is stronger and has a lower density

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- **Base metal is stronger and has a lower density**

***Base metal alloy advantages are principally found only in their **strength and low density.**

<u>As compared to Type IV gold alloys, base metal alloys have:</u>

- A higher resistance to deflection in thin segments
- A lower yield strength
- A higher modulus of elasticity
- A lower specific gravity
- A much higher melting temperature *(2300° F to 2600°F)*

Remember: The **nickel** in the composition of base metal alloys is responsible for ductility of the alloy. It is also measured as a percentage of elongation and determines how much margins can be closed by burnishing. **Chromium** produces a passivating film for corrosion resistance and **cobalt** increases the rigidity of the alloy.

Metals are classified as noble elements based on their lack of chemical reactivity. The **noble metals** include gold, platinum, palladium, and other inert metals. Alloys with less than 25% noble elements are called **base metals.**

Four types of casting alloys are described in the ADA specifications: Type, I, II, III, and IV. The differences between them are predominantly the strength and elongation of the casting alloy. Type I is the weakest, has the greatest elongation, and is used for inlays. Type IV is the strongest, has the least elongation, and is used for high-stress bridges and partial denture frameworks.

If a mold that is created for investing and casting does not _____ to compensate for the action of the metal alloy, the casting will not fit.

- Equate
- Contract
- Expand
- None of the above

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• **Expand**

Four mechanisms play a role in producing an expanded mold and thus compensating for the solidification shrinkage of the alloy.

1. **Setting expansion** → results from normal crystal growth. In air, it is about 0.4% but it is partially restricted by the metal investment ring.

2. **Hygroscopic expansion** → employed to augment normal expansion by allowing the investment to set in the presence of water. It is said that this water will replace the water used by the hydration process and thus maintain the space between the growing crystals. Allows continued expansion outward rather than restricting them. This expansion ranges from 1.2% to 2.2%.

3. **Wax pattern expansion** → the wax pattern is warmed while the investment is still fluid. The heat may come from the chemical reaction of the investment itself or the water bath in which the casting ring is immersed.

4. **Thermal expansion** → occurs when the investment is heated in the burn out oven. It also serves to eliminate the wax pattern and to prevent the alloy from solidifying before it completely fills the mold.

A **seven-eighths** crown is a three-quarter crown whose vertical:

- Mesiobuccal margin is positioned slightly distal to the middle of the buccal surface
- Distobuccal margin is positioned slightly mesial to the middle of the buccal surface
- Mesiolingual margin is positioned slightly distal to the middle of the lingual surface
- Distolingual margin is positioned slightly mesial to the middle of the lingual surface

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- **Distobuccal margin is positioned slightly mesial to the middle of the buccal surface**

A **partial crown** is a cast restoration made entirely from metal and covers more than half but not all of the tooth's clinical crown. A partial crown is named according to the fractional amount of the clinical crown it covers. Examples are the half, three-quarters, four-fifths, and seven-eighths crowns. In most instances, the facial surface of the tooth is not disturbed for esthetic reasons.

The **seven-eighths** crown design is especially effective either as a single tooth or an abutment restoration on maxillary molar teeth where both proximal surfaces are involved as well as the distal buccal surface of the tooth. In many instances, the mesio-buccal cusps of maxillary first and second molars can be preserved for esthetics and still provide adequate extension to include extensive areas of destruction.

Seven-eighths crown:

- It can be used on any posterior tooth.
- Esthetics is good since the veneered distobuccal cusp is obscured by the mesiobuccal cusp.
- Distobuccal finish line is easy to access, which makes preparation easier to do. It also makes cleaning of the margins easier for the patient.
- More coverage than the standard 3/4 crown which improves its resistance.
- Especially useful when the distal surface has caries or decalcification.
- Serves as an excellent abutment for a bridge.

Regarding the occlusion of a complete denture:

(A) A decreased vertical dimension of occlusion refers to excessive interocclusal distance *(increased freeway space)*
(B) A decreased vertical dimension of occlusion refers to the loss of interocclusal distance in the rest position
(C) An excessive vertical dimension frequently results in cheek biting
(D) An excessive vertical dimension is the usual cause of clicking of teeth
(E) Phonetics helps in verifying the vertical dimension of occlusion
(F) Esthetics helps in verifying the vertical dimension of occlusion

- (A), (C), and (E) are true
- (B), (D), and (F) are true
- (A), (C), (E) and (F) are true
- (B), (C), (E) and (F) are true
- (A), (D), (E) and (F) are true
- All of the above statements are true

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- **(A), (D), (E) and (F) are true**

The following **must be** considered while verifying the vertical dimensions of the occlusion:

- Pre-extraction records
- The amount of interocclusal distance *(freeway space)* to which the patient was previously accustomed to
- Esthetics → facial harmony should be noted along with facial expression
- Phonetics → speech sounds
- Length of the lip in relation to the teeth
- The condition and amount of shrinkage of the ridges

Clicking of Dentures	
Cause	**Treatment**
Excessive VDO *(usual cause of clicking)*	• Patient remount • Fabricate new CD/CD
Lack of retention of mandibular or maxillary denture	• If due to underextension: border mold and reline • If due to overextension: reduce as indicated by PIP and disclosing wax
Porcelain teeth	• Use acrylic resin teeth

Polysulfide rubber base impression materials:

- Set in 5 to 6 minutes
- Have a very poor moisture tolerance
- Have a good odor and taste
- Have a shelf-life that is relatively long, up to 48 months
- Require custom trays for impression making

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• **Require a custom tray for impression making**

The polymerization of polysulfides is **exothermic** and is accelerated by an increase in the temperature or humidity.

Polysulfides have **good flow properties** and **high flexibility** and **tear strength.** These materials show the **strongest resistance to tearing,** but as a result, impressions can distort when removed from areas where deep undercuts are present. They have a long working time and a relatively long polymerization time, which may add to patient discomfort. Their resistance to deformation is low. **Note:** The **lead dioxide** *(the accelerator)* is responsible for the brown color and difficulty in cleaning clothes that come in contact with this impression material.

Generally, the use of this material demands the construction of a special tray **(custom tray)** in order to control polymerization shrinkage by the use of a uniform thickness of impression material.

Advantages:

- Lower cost *(as compared to silicones and polyethers)*
- Long working time *(5 - 7 minutes)*
- High tear strength
- High flexibility
- Good detail reproduction

Disadvantages:

- Poor dimensional stability
  - water by-product
  - pour within one hour
  - single pour
- Custom trays
- Messy *(paste-paste mix, bad odor, may stain clothing)*
- Long setting time *(8 - 12 minutes)*

A wire-gauge is selected on the basis of its active length. A short arm clasp (< *7mm)* should be made in what gauge of wire for optimum function below its proportional limit?

- 20 – gauge
- 19 – gauge
- 18 – gauge
- 16 – gauge

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- **20 – gauge** → or finer, as < 7mm, is a short arm clasp and shorter length clasps must have a finer gauge of wire for optimum flexibility

The **flexibility** of a retentive clasp arm depends on:

- **Length of the clasp** → the flexibility of a clasp varies directly with the cube of its length. Thus, **increased length** results in a marked **increase in flexibility.**
- **Thickness of the clasp** → as a clasp becomes **thicker;** its flexibility is **reduced** by a cube ratio.
- **Width of the clasp** → as the width of a clasp **increases;** its flexibility **decreases** by a ratio of 1:1.
- **Cross-sectional form** → a round form is equally flexible in all directions. In contrast, a half-round form flexes readily only when the stress is applied perpendicular to the flat surface.
- **Taper of the clasp** → a uniform taper permits increasing flexibility toward the tip of the clasp.
- **Clasp material** → different metals flex more than others. **Remember:** Wrought wire retentive arms have increased flexibility.

All-ceramic crowns are generally known for their low flexural strength. Which type of porcelain is considered better than other types for this property?

- Feldspathic
- In-Ceram
- IPS-Empress
- In-Ceram Zirconia
- Dicor

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• **In-Ceram Zirconia**

Note: All-ceramic crowns also have a relative tendency to fracture at a minimum deformation. Microscopic surface defects, under load lead to crack propagation and eventually to failure.

Examples of Flexural Strengths	
In-Ceram Zirconia	⟹ 800 Mpa
Procera	⟹ 687 Mpa
In-Ceram	⟹ 450 Mpa
In-Ceram Spinell	⟹ 350 Mpa
OPC	⟹ 150 Mpa
IPS Empress	⟹ 140-180 Mpa
Vitablocs	⟹ 120 Mpa
Dicor	⟹ 120 Mpa
Aluminous	⟹ 100 Mpa
Feldspathic	⟹ 60-90 Mpa

PERIODONTICS

Legend

Major Topic	Abbreviation
Flaps / Grafts / Surgery	F/G/S
Gingivitis / Periodontal Disease	Ging / Pdl Dis
Inflammation	Infl
Miscellaneous	Misc.
Oral Hygiene	OH
Periodontal Ligament / Gingiva	Pdl / G
Plaque / Calculus	P / C
Pockets / Furcations	Poc / Fur
Probing	Prob
Root Sensitivity / Recession	Rt Sens / Rec
Scaling / Root Planing / Gingival Curettage	Scal / Rp / Gc

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Which statement regarding an autogenous free gingival graft is **not** true?

- It can be placed to prevent further recession
- It can be used to effectively widen the attached gingiva
- It retains its own blood supply and is not dependent on the bed of recipient blood vessels
- The greatest amount of shrinkage occurs within the first 6 weeks
- It is also useful for covering non-pathologic dehiscences and fenestrations

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- **It retains its own blood supply and is not dependent on the bed of recipient blood vessels**

***This is **false;** It retains none of its own blood supply and is totally dependent on the bed of recipient blood vessels.

In some instances, it can be used to cover a root surface with a narrow denudation. The procedure yields a high degree of successful results when used for increasing the width of the attached gingiva. The free gingival graft may be used **therapeutically** to widen the gingiva after recession has occurred. It may be used **prophylactically** to prevent recession where the band of gingiva is narrow and of a thin, delicate consistency.

The free gingival graft is an **autogenous graft** of gingiva that is placed on a viable connective tissue bed where initially buccal or labial mucosa was present. In most cases, the donor site from which the graft is taken is an **edentulous region** or the **palatal area.** The graft epithelium undergoes degeneration after it is placed. Then it sloughs, the epithelium is reconstructed in about a week by the adjacent epithelium and proliferation of surviving donor basal cells. In two weeks' time, the tissue appears to have reformed, but maturation is not completed until 10 to 16 weeks. The time required is proportional to the thickness of the graft. **Note:** The free gingival graft **receives its nutrients from the viable connective tissue bed.**

The procedure **may or may not yield** a successful result when used to obtain root coverage; the result is not highly predictable in such cases. The graft may be used to correct localized narrow recessions or clefts **but not deep, wide recessions.** In these instances, **the laterally repositioned flap** *(a pedicle graft)* **has a greater predictability.** The free gingival graft is rarely used on the facial or lingual surfaces of mandibular third molars *(especially facial).*

In a hemisection the tooth is cut in half. The technique is used almost exclusively on:

- Mandibular first and second premolars
- Maxillary first and second molars
- Maxillary canines
- Mandibular molars to treat Class II or III furcation invasions

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- **To eliminate periodontal pockets** → the existing bony topography is changed to **eliminate periodontal pockets**

It **does not cure** periodontal disease. It provides the patient with the opportunity and the access to maintain his / her own periodontium and dentition with routine oral hygiene procedures.

Before employing **osseous resection** or **recontouring** to treat an infrabony defect, the therapist should consider the following alternative treatments:

- **Maintenance** with periodic root planing
- **Bone grafts**
- **Reattachment-fill** procedures
- **Hemisection** or **root amputation**

Osseous resection surgery should **not be done** until the etiologic factors that resulted in the formation of the osseous defects are arrested. Clinically detectable inflammation must be eliminated by scaling and root planing and by the patient's exercise of optimal plaque control.

Important: The **most critical factor** in determining whether a tooth should be extracted or have surgery performed on it is the **amount of attachment loss** *(which is the apical migration of the epithelial attachment)*.

Note: In some surgical procedures, it is necessary to leave **interradicular bone exposed.** This usually results in bone loss of no clinical consequence.

PERIODONTICS F / G / S

All of the following statements regarding periodontal flaps are true **except:**

- Full thickness periodontal flaps involve reflecting all of the soft tissue, **including the periosteum** to expose the underlying bone
- The partial thickness periodontal flap includes **only** the epithelium and a layer of the underlying connective tissue
- Both full thickness and partial thickness periodontal flaps can be **displaced**
- Flaps from the palate are considered easier to be displaced than any other region
- Flaps should be uniformly thin and pliable

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• **Flaps from the palate are considered easier to be displaced than any other region**

***This is **false;** palatal flaps **cannot be** displaced *(owing to the absence of unattached gingiva).*

A periodontal flap is a segment of marginal periodontal tissue that has been surgically separated coronally from its underlying support and blood supply and attached apically by a pedicle of supporting vascular connective tissue. **Flap procedures are the most commonly used of all periodontal surgical techniques.** The most commonly used flaps are full thickness mucoperiosteal flaps. These flaps include the surface mucosa *(defined as epithelium, basement membrane, and connective tissue lamina propria)* and the contiguous periosteum of the underlying alveolar bone. A **partial thickness flap** includes only the mucosa, which is separated from the periosteum by sharp dissection. Alveolar bone is not exposed. These flaps are used in the preparation of recipient sites for free gingival grafts or **when a dehiscence or fenestration** is present on a prominent root.

The **base of the flap** must be uniformly thin, **usually about 2 mm thick.** One determinant of how the flap will be raised, that is either as full thickness or as a partial thickness flap, is the thickness *(amount)* **of the attached gingiva prior to surgery.** Generally, a full thickness flap will be used where the attached gingiva is thin *(2 mm or less in width),* and a partial thickness flap is used when the attached gingiva is thick *(2 mm or more).*

The internal bevel incision is basic to most periodontal flap procedures. It is the incision from which the flap is reflected to expose the underlying bone and root. The internal bevel incision accomplishes **three important objectives: 1)** it removes the pocket lining; **2)** it conserves the relatively uninvolved outer surface of the gingiva, which, if apically positioned, becomes attached gingiva; and **3)** it produces a sharp, thin flap margin for adaption to the bone-tooth junction.

There are various distal flap approaches used for retromolar reduction. The **simplest** is the:

- Gingivectomy
- Apically positioned flap
- Distal wedge
- Laterally positioned flap

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- **Distal Wedge**

Distal wedge procedures *(sometimes called proximal wedge procedures)* are frequently performed after wisdom teeth are extracted because the bone fill is usually poor, leaving a periodontal defect. This region is occupied by glandular and adipose tissue covered by unattached non-keratinized mucosa. **Only if sufficient space exists distal to the last molar,** a band of attached gingiva may be present. In such a case a distal wedge operation can be performed.

These procedures are also performed in the following areas of the mouth:

- The **maxillary tuberosity** region
- The **mandibular retromolar triangle** area
- **Distal to the last tooth in an arch,** or mesial to a tooth which approximates an edentulous area

Many designs have been presented for this flap procedures. However, the basic principle is one of making at least two incisions distal or mesial to the tooth and carrying these incisions parallel to the outer gingival wall, **thus forming a wedge;** the base of which is the periosteum overlying the bone and the apex of which is the coronal gingival surface. Detachment of the wedge from the periosteal base and elimination of the tissues involved in the distal pocket region also reduces tissue bulk and allows for access to the underlying bone.

Which situation would be a **contraindication** for a gingivectomy?

- A fibrotic area of free gingiva that covers part of the occlusal surface of tooth number 17
- An infrabony pocket of 8 mm on the distal aspect of tooth number 19
- Correction of severe gingival overgrowth
- Gingival pockets composed of enlarged fibrotic tissue

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- **An infrabony pocket of 8 mm on the distal aspect of tooth number 19**

Gingivectomy is a surgical procedure where **pocket depth is eliminated** by resecting the tissue coronal to the pocket base. The final aspect of the gingivectomy is to bevel or contour the coronal margin of the wound so as to provide the most physiological shape and marginal thickness so adequate oral hygiene techniques may be carried out. This procedure works well when treating pseudopockets, hereditary gingival enlargement, suprabony pockets, and hyperplasia caused by dilantin therapy. **Important:** An adequate amount of attached gingiva **must be present** before a gingivectomy is done; otherwise the result will be an area with minimal or no attached gingiva.

Contraindications to gingivectomy include **infrabony** pockets *(defects)* and a **lack of attached tissue.** Limitations include compromised esthetics with longer teeth, lack of access to bony defects, and having a broad, open wound post-surgically.

A **gingivoplasty** is directed towards **reshaping the gingiva and papilla** of a tooth for correction of deformities and to provide the gingiva with normal and functional form.The overall objective **is not** to eliminate periodontal pockets, but rather to provide a **more physiological tissue contour.** While it is true that portions of the gingiva are excised during the gingivoplasty procedure, it is the reshaping, not the excision, of gingiva that defines gingivoplasty. **Note:** This procedure is commonly used to correct the tissue contours that result from **ANUG.**

The **following factors** should be considered when electing to perform a gingivectomy **rather than** a periodontal flap:
- Pocket depth *(if base of pocket is located at the mucogingival junction or apical to the alveolar crest, **do not** perform gingivectomy)*
- Need for access to bone
- Amount of existing attached gingiva

The **primary objective** and advantage of surgical flap procedures in the treatment of periodontal disease is:

- Reduce or eliminate periodontal pockets
- To provide access to root surfaces for debridement
- Regrowth of alveolar bone
- Maintenance of biological width
- Establishment of adequate soft tissue contours

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- **To provide access to root surfaces for debridement** → all the other choices are goals but not a primary objective

The techniques vary with the goal that is sought. However, the common goal of all flap procedures is to **provide access for instrumentation.** It gives the clinician the opportunity to visualize the roots so that calculus may be removed more completely.

Without direct visualization provided by a flap, it is rare that a clinician can effectively root plane beyond **5mm** of probing depth or into furcations of lesser depth. It also makes removal of **granulomatous tissue** from the region of the periodontal defect difficult. It is important to remove this, due to the fact it contains **epithelium and the potential presence of bacterial infiltration.**

Important: If a patient fails to demonstrate adequate oral hygiene during initial therapy *(scaling and root planing)*, **surgery is contraindicated** because after surgery the incidence of disease recurrence will be greater if oral hygiene remains poor. The best course of action is to continue to stress oral hygiene and maintain areas with scaling and root planing.

A **modified Widman flap** is:

- A partial-thickness flap
- A full-thickness flap

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- **A full-thickness flap**

The **modified Widman flap** *(MWF)* is a modification of the replaced flap. It is a **full-thickness** flap that may be used in open flap debridement and regenerative periodontal procedures. This procedure is a mainstay of periodontal surgery on single-rooted teeth and on flap surfaces of molars affected by moderate pockets and infrabony defects.

Note: Repositioned flaps, as a category, include replaced flaps, modified Widman flaps, and excisional new attachment procedures. They all heal by repair *(i.e., a new attachment consisting of a long junctional epithelium and connective tissue adhesion or attachment)* and are considered pocket reduction procedures. The pocket reduction is achieved mainly by gains in clinical attachment mediated by repair.

The **specific objectives** for using the MWF are:

- To **gain access** to the underlying bone and root surfaces
- To **reduce pocket depth** by establishing a new attachment at a more coronal level
- To **preserve** an adequate zone of attached gingiva
- To **provide** an environment for healing by primary closure

Indications for the MWF include:

- Pockets where the bases are located coronal to the mucogingival junction
- Where there is little or no thickening of the marginal bone
- When shallow to moderate pocket depths can be reduced
- When esthetics is important *(anterior region)*

PERIODONTICS

F / G / S

A soft tissue graft that is rotated or otherwise repositioned to correct an adjacent defect is called a:

- Free gingival graft
- Pedical graft
- Connective tissue graft
- Frenectomy

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• **Pedicle graft**

The pedicle graft was the first periodontal plastic surgery procedure to be used for root coverage. It provides a superior result from an esthetic standpoint, but is less versatile than the connective tissue graft. **Important:** The base of the graft remains attached to the donor site to maintain the blood supply.

With pedicle grafts, there is less concern about nutrient flow from graft bed to graft. The properly performed pedicle graft never loses its blood supply during the surgical procedure.

The **major advantages** of pedicle grafting include:
- Predictable correction of gingival recession is possible, because the graft has an uninterrupted blood supply
- Postoperative discomfort is usually minor
- Since the color of the graft matches the adjacent gingiva, the procedure provides good esthetics

Indications include:
- To widen an inadequate zone of attached gingiva
- To repair an isolated area of gingival recession

Contraindications include:
- The prospective donor site lacks sufficient attached gingiva
- The donor site has a fenestration or dehiscence of its supporting bone

Note: Pedicle grafts are not well-suited for repairing generalized recession defects. They were designed for repair of isolated recession. Many recession defects don't have a suitable adjacent donor site.

What is **guided tissue regeneration?**

- A **soft tissue graft** used to correct mucogingival junction involvement
- Placement of nonresorbable barriers or resorbable membranes and barriers over a bony defect
- A **free gingival graft** used to increase the amount of attached gingiva
- Placement of an **autograft** to treat a bony defect

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- **Placement of nonresorbable barriers or resorbable membranes and barriers over a bony defect**

Guided tissue regeneration is a procedure that blocks the re-population of the root surface by long junctional epithelium and gingival connective tissue to allow cells from the periodontal ligament and bone to re-populate the periodontal defect. This physical blockade may be accomplished using nonresorbable barriers such as expanded polytetrafluoroethylene *(Teflon)* or resorbable membranes and barriers such as Type I bovine collagen, calcium sulfate (Plaster of Paris), or polyactic acid. If a nonresorbable barrier is used, 6-10 weeks after placement a second surgical procedure is done to retrieve the barrier.

Currently, regenerative procedures are applicable and predictable under a certain set of circumstances: **(1)** The patient exhibits exemplary plaque control both before and after regenerative therapy, **(2)** The patient does not smoke, **(3)** There is occlusal stability of the teeth at the regenerative site, **(4)** Osseous defects are vertical in nature, with the more walls of bone remaining increasing the likelihood of regenerative success.

Note: This technique is based on the assumption that only the periodontal ligament cells have the potential for regeneration of the attachment apparatus of the tooth.

The primary reason for the **failure** of a free gingival graft is:

- Infection
- Edema
- Disruption of the vascular supply before engraftment
- The formation of scar tissue

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• **Disruption of the vascular supply before engraftments**

The **second** most common reason is infection.

A **free gingival graft** involves taking a section of attached gingiva from another area of the mouth *(usually the hard palate or an edentulous region)* and suturing it to the recipient site. The success depends upon the graft being **immobilized at the recipient site.** This procedure is used to increase the zone of attached gingiva with the possibility of gaining root coverage as well. The difficulty in getting complete root coverage lies in the fact that an avascular graft is placed over a root surface also devoid of a blood supply.

Remember: Positioned flaps (*i.e., laterally repositioned flap, coronally positioned flap, and apically positioned flap)* are procedures in which the coronal margins of the flap are lifted from an area adjacent to the recipient site, but the flap is not freed-up. In these procedures, the **vascular supply to the flap is maintained** → as opposed to the free gingival graft.

Important: There is **no necrotic slough** of positioned flaps because these flaps take their vascular supply with them. In a **free gingival graft,** the healing involves revascularization of the graft. The top layers of the graft are the last to be revascularized; therefore the epithelium dies off *(degeneration),* producing the necrotic slough.

A **variation** of the laterally positioned flap is called:

- A coronally positioned flap
- A modified Widman flap
- A double papilla flap
- A free gingival flap

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• **A double papilla flap**

A **double papilla flap** is a variation of the laterally positioned flap. This procedure uses the same principles as the laterally positioned flap; except the gingiva between the teeth *(papilla)* on either side are moved over the exposed root.

Indications include:
- Trauma from incorrect toothbrushing
- Covering the exposed root surface with gingiva also helps to reduce or eliminate the problem of hypersensitivity

The procedure is indicated where there has been recession of the labial or lingual gingiva but when destruction of the interdental papillae on either side of the denuded area either has not occurred or is minimal. The dental alveolar process is much less susceptible to permanent damage after surgical exposure than either the labial or the lingual plates of bone. This procedure uses interdental tissue rather than the buccal and lingual tissue.

Notes:

1. **Deep periodontal pockets** are often treated by flap surgery. These cases will often result in reduced pocket depth by **formation of a long junctional epithelium** *(soft tissue reattachment)*, even if there is no change in the position of the gingival margins.
2. The **best indicator of success** of a **periodontal flap** procedure is postoperative maintenance and plaque control by the patient.

All **corners** of a periodontal flap should be:

- Sharp
- Rounded
- It doesn't matter whether the corners of a periodontal flap are sharp or rounded

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- **Rounded**

The Four Basic Rules For Flap Design:

1. The **base of the flap** should be wider than the free margin in order to afford sufficient blood circulation to the free margin of the flap.

2. The **lines of the incision** must not be placed over any defect in the bone to prevent delayed healing.

3. Incisions that **traverse a bony eminence** *(canine)* should be avoided. The mucosa covering bony eminences is thin and healing is slow and may result in an ugly scar formation.

4. All corners of the flap should be **rounded.** Sharp points will delay healing.

Important:

- Healing should take place without complication **if basic surgical principles are followed.**
- **Incisions made in tissues that harbor uncontrolled infection** may cause rapid spread of the infection. **Do not do this. Most** periodontal surgical procedures are performed **only after** anti-infective therapy **has been** completed.

Which of the following is a full-thickness, mucoperiosteal flap that has a relatively high degree of predictability and is a **"work-horse"** of periodontal therapy?

- Coronally positioned flap
- Laterally positioned flap
- Double papilla flap
- Apically positioned flap

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• **Apically positioned flap**

An apically positioned flap *(APF)* is a full-thickness, mucoperiosteal flap. It has a relatively high degree of predictability and is a **"work-horse"** of periodontal therapy. The objective of this type of flap is to surgically eliminate deep pockets by positioning the flap apically while retaining the attached gingiva. Additionally, surgical access is obtained for osseous surgery, treatment of infrabony *(intrabony)* pockets, and root planing.

In the course of flap surgery, after gaining access to the underlying osseous tissue and performing the required therapy, the flap is sutured to place at a more apical level, exposing the alveolar margin. When this is done, additional attached gingiva granulates from the periodontal ligament and covers the barely exposed bone. This additional tissue joins the apically positioned attached gingiva to form a broader zone of gingiva. **Note: On the palatal surface of maxillary molars** you need to trim the flap margin to the proper length **during the procedure.**

Indications for APF include:
• Moderate or deep pockets
• Furcation-involved teeth
• Crown lengthening

A **contraindication** to an APF is a patient at risk for root caries. Excessive root surfaces are often exposed after performing an apically positioned flap. APF's are also contraindicated where tooth exposure would be unesthetic.

A free mucosal autograft **differs** from a free gingival graft in that the transplant in a free mucosal graft is:

- Connective tissue with an epithelial covering
- Connective tissue without an epithelial covering
- Epithelial tissue with its own blood supply
- Epithelial tissue without its own blood supply

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- **Connective tissue without an epithelial covering**

A free mucosal autograft **differs** from a free gingival graft in that the transplant in a free mucosal graft is connective tissue without an epithelial covering.

Epithelial differentiation is induced by the underlying connective tissue, so that free grafts of dense connective tissue taken from keratinized areas result in the formation of keratinized tissue even when transplanted to non-keratinized zones. This procedure is somewhat more difficult than free gingival grafting. **This procedure is often used on canines where there is little keratinized gingiva to create a band of gingiva-like tissue.**

Remember: During healing, the epithelium of **free gingival grafts** degenerates *(necrotic slough),* and re-epithelialization occurs by **proliferation of epithelial cells from adjacent tissue and surviving basal cells of the graft tissue.**

Note: Free gingival grafts are often used in conjunction with a **frenectomy** to prevent reformation of high frenal attachments.

Which of the following mucogingival surgical techniques is indicated in areas where there is gingival recession that is narrow, adjacent to which a wide band of attached gingiva exists, which can be used as a donor site?

- Free gingival autograft
- Double papilla flap
- Modified Widman flap
- Laterally positioned flap

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• **Laterally positioned flap**

The **laterally positioned flap,** also known as the pedicle flap, is usually a full-thickness flap that is attached at its base with its free end adjacent to the defect *(recipient site)*. The defect is covered by stretching the flap laterally until the free end comes over it.

The laterally positioned flap is used to **correct or prevent recession** by providing root coverage and creating a broader band of gingiva. The procedure may be used in the absence of recession to widen the zone of gingiva.

Positioned flaps are flap procedures where the coronal margins of the flap are advanced apically, coronally, or laterally to a new location relative to the site they occupied before the procedure. Like repositioned flaps, most positioned flaps heal by repair.
The category includes:

1. The **laterally positioned flap** is usually a full-thickness flap but may be a partial-thickness gingival flap. An objective of this procedure is to halt recession and to restore denuded areas cosmetically with attached gingiva.
2. The **apically positioned flap** is a full-thickness, mucoperiosteal flap. It is a "work horse" of periodontal surgery. It is most commonly used in conjunction with osseous surgery.
3. The **coronally positioned flap** is a full-thickness mucoperiosteal flap that has almost exclusively been used to restore gingival height and the zone of attached gingiva over isolated areas of gingival recession.

Note: Positioned and repositioned flaps (*i.e., replaced flaps, modified Widman flaps, and excisional new attachment procedures)* are, in reality, **pedicle flaps.** They are all physically attached at their apical base by a pedicle of lining mucosa and an **intact blood supply.**

Non-supporting bone is alveolar bone not directly related to tooth support. It includes all of the following **except:**

- Bony exostoses
- Edentulous ridges
- Tori
- The alveolar bone proper
- Flattened interdental contours and ledges

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• **The alveolar bone proper** → this is supporting bone that is directly involved in tooth support

Osteoplasty is the **reshaping or recontouring of bone** that does not provide attachment for the periodontal fibers *(non-supportive bone)*. This is similar to the gingivoplasty in that it is not directed toward eliminating the walls of the pocket, but is performed to recontour and reshape the underlying osseous structures. It is important to note that in this technique the bone removed is not supporting bone. This means that the osseous reduction **does not reduce** bone to which the periodontal ligament is attached.

Osteoplasty indications include:
• Deep interproximal pockets on posterior teeth involving the buccal interdental bone
• Pockets on the buccal, lingual, and palatal surfaces where resorption of bone results in thick ledges.
• Tilted lower second molar adjacent to non-replaced extracted first molar.

Ostectomy is directed toward removal of osseous defects or infrabony pockets by elimination of the bony pocket walls. This bone is classified as **being supporting in nature**, but is sacrificed in the belief that eliminating the pocket is worth the price paid for by the loss in attachment apparatus. After the removal of the osseous pocket walls, some recontouring is usually done to provide the most optimal osseous architecture to which the overlying gingival tissues can conform and be maintained. A **major contraindication** to the removal of crestal bone is when you may be weakening the support of an adjacent tooth.

Ostectomy indications include:
• Interproximal craters in bone: shallow and **wide craters** are not favorable for reattachment while deep and narrow are.
• Extremely deep interproximal pockets **where the neighbor** areas are intact or minimally affected.
• Shallow infrabony **defects** *(interproximal)*, **and where** reattachment **has failed.**

Clinically, a bone grafting procedure is most likely to be successful in:

- One-walled defects
- Two-walled defects
- Three-walled defects
- Through-and-through furcation defects

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• **Three walled defects**

***Defects that conceivably **"will hold water"** offer excellent opportunities for bone graft containment and periodontal regeneration procedures.

The relative degree of success of periodontal bone grafting is reported to **vary directly** with the number of bony walls of the defect *(vascularized, osseous surface area)* and inversely with the surface area of the root against which the graft is implanted. Thus a narrow, three-walled infrabony defect usually yields the greatest success, a two-walled defect the next best, and a one-walled defect the least *(of infrabony defects)*. Sometimes the graft may take even when the bone graft is piled on the crest of the interdental septum. Clinically, the chances of the success are best in a three-walled infrabony pocket and **least** in a through-and-through furcation defect on a maxillary molar.

Root resorption is the most likely side effect of an autogenous bone graft in managing an infrabony pocket. This resorption often extends into the dentin and the pulp chamber. Other postoperative problems, occurring occasionally after osseous or marrow transplants are infection, exfoliation of the graft, various and sometimes prolonged rates of healing, and rapid recurrence of the defect.

Notes:

1. A **dehiscence** is a loss of the buccal or lingual bone overlaying the root portion of a tooth, leaving the area covered by soft tissue only.
2. An **allograft** is a graft taken from one human *(donor)* and placed in another human *(recipient)*.
3. **Osseous grafting techniques** include: Osseous coagulum, autogenous intraoral bone, iliac crest bone, freeze-dried bone allograft *(FDBA)* with autologous bone, porous, coral-derived hydroxyapatite and Undecalcified freeze-dried bone **allograft** *(UDFA)*.

Which of the following is the **LEAST** important diagnostic aid in recognizing the early stage of gingivitis?

- Bleeding upon probing
- Gingival color
- Pocket depths
- Stippling of the gingival tissue

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• **Stippling of the gingival tissue**

Inflammation, bleeding upon probing, and pocket depths are the **most important diagnostic aids** or signs of gingival or periodontal disease. Gingiva may or may not be stippled whether healthy or inflamed. The presence or absence of stippling is not diagnostic.

Clinical criteria used for diagnosing gingivitis:

Color → normal gingival color ranges from coral pink to various stages of pigmentation. ***Erythema** refers to an intense red color. Probably the **most common** color change noted with periodontal disease is **cyanosis** *(bluish-purple hue)*.

Contour → has a range of normal. This is influenced by missing teeth, position of teeth, etc. Papillae should fill the interproximal spaces. Gingival margins should be **scalloped** in form.

Tone → the normal consistency of the gingival tissue should be **resilient** and **fibrotic** in nature from the free gingival groove, apical to the mucogingival junction. **Texture,** stippling of the attached gingiva *(the so-called orange peel appearance)* may or may not be present.

Size → the healthy gingival tissues should be **well-contoured** to the underlying osseous architecture with the free gingival margin being of such thinness to allow for a **"knife edge"** thickness at the dentogingival margin.

Plaque, calculus → the best way to evaluate the amount and distribution of plaque is by the utilization of disclosing solution. **Remember: Without** bacterial plaque there **would be no** gingivitis.

Important: The predominant periodontal disease is **gingivitis.**

Generally speaking, bacteria associated with **periodontal health** are characterized as:

- Gram-negative, motile, aerobes
- Gram-negative, nonmotile, anaerobes
- Gram-positive, nonmotile, facultative anaerobes
- Gram-positive, motile, aerobes

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- **Gram-positive, nonmotile, facultative anaerobes**

In the **healthy** mouth, more than 350 species of microorganisms have been found. Periodontal infections are linked to fewer than 5% of these species. Healthy and disease-causing bacteria can generally be grouped into two categories:

1. The harmless or **helpful** bacteria are usually known as **gram-positive, nonmotile, facultative anaerobic** bacteria *(coccal and rod forms)*. **Note:** Streptococcus species *(specifically Streptococcus gordinii)* and Actinomyces species are associated with periodontal health.
2. In **periodontal disease,** the bacterial balance shifts over to **gram-negative, motile, strictly anaerobic** bacteria. Inflammatory disease and injury cannot develop without these bacteria.

Among the bacteria **most implicated** in periodontal disease and bone loss are the following:

- **Actinobacillus actinomycetemcomitans** *(Aa)* → associated with **aggressive** periodontal disease *(formerly called early onset periodontitis)* and **localized aggressive** periodontitis *(former called Localized juvenile periodontitis).*
- **Porphyromonas gingivalis** → associated with **chronic** periodontitis *(formerly called adult periodontitis*
- **Bacteroides forsythus** → strongly linked to periodontal disease
- **Treponema denticola, sokranskii** → associated with deep periodontal pockets and **ANUG**
- **Prevotella intermedia** → associated with deep periodontal pockets and **ANUG**

Note: Eikenella corrodens, Campylobacter **rectus**, Fusobacterium nucleatum, Peptostreptococcus, Prevotella **nigresens**, Enteric **rods** / Pseudomonas **species** and Eubacterium species have also **been implicated** as **periodontal** pathogens.

Endotoxins are the lipopolysaccharide component of the cell wall of:

- Gram-positive bacteria
- Gram-negative bacteria
- Both gram-positive and gram-negative bacteria
- Neither gram-positive and gram-negative bacteria

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- **Gram-negative bacteria**

The cell wall of Gram-negative bacteria consists of a lipopolysaccharide base, also known as **endotoxin,** that has significant pathogenic potential. Typically, LPS containing Gram-negative cell wall extracts are capable of promoting bone resorption, inhibiting osteogenesis, chemotaxis of neutrophils, and other events **associated with active periodontitis.**

Important facts:

- **Free endotoxin** is present in dental plaque and inflamed gingiva
- Plaque accumulation has a **direct effect** on the severity of gingivitis
- Plaque bacteria **produce enzymes** *(hyaluronidase, collagenase, chondroitin sulfatase, elastase and proteases)* that may initiate periodontal disease.
  1. **Collagenase** *(which is produced by Bacteroides species)* catalyzes the degradation *(hydrolysis)* of collagen.
  2. **Hyaluronidase** *(which is produced by Streptococcus mitans and salivarius)* and **Chondroitin sulfatase** *(which is produced by Diptheroids)* may lead to the destruction of the amorphous ground substance.
- **Antibodies or immunoglobulins** are produced by plasma cells in response to oral bacteria or their byproducts. The most numerous are **IgG** which act to neutralize bacterial toxins by enhancing phagocytosis.
- The most likely **source of bacteria** found in diseased periodontal tissue is **subgingival plaque.**
- The likelihood that **bacterial endotoxins** play a major role in gingival inflammation is evidenced by the following:
  1. A reduction in inflammation by the **removal of** plaque.
  2. A reduction **of the** inflammatory state **with** antibiotic treatment.

Which of the following clinical signs and symptoms is characteristic of acute necrotizing ulcerative gingivitis *(ANUG)*?

- Minimal bleeding
- Ulceration of papillae *(punched out)*
- Painless
- Periodontal pocket formation

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• **Ulceration of papillae** ***(punched out)***

ANUG is an acute recurring gingival infection of complex etiology, characterized by necrosis of papillae, spontaneous bleeding and pain. The **two principal bacteria** associated with ANUG are **Prevotella intermedia** and **spirochetes** *(Treponema denticola is the intermediate-sized spirochete associated with ANUG)*. Fusobacterium species as well as Selenomonas species can also be seen.

ANUG *(which is also called "Vincent's infection" and "trench mouth")* is a condition which presents rather **pathognomonic** signs and symptoms. The two most important clinical signs are:

1. **Interproximal necrosis** *(ulceration of papillae)* and pseudomembrane formation on marginal tissues → only affects the gingiva. **Important:** There is **no attachment loss** associated with ANUG.
2. **History of soreness** *(pain)* and **bleeding gums** caused by eating and brushing. Other signs and symptoms include a fetor oris *(offensive odor)*, a low-grade fever, lymphadenopathy, and malaise. **Note:** The **dominant WBC** noted in the inflammatory infiltrate of ANUG is the **neutrophil.**

ANUG **occurs most often** in adults between the ages of 18-30. Factors which seem to predispose someone to ANUG include a history of gingivitis, tobacco smoking, gross neglect *(poor oral hygiene)*, fatigue, stress, poor nutrition, and immuno-compromised patients.

The **treatment** of ANUG includes debridement, hydrogen peroxide *(or warm saline)* rinses, and **antibiotic therapy** *(Pen. V)* if there is systemic involvement *(i.e., fever, malaise, lymphadenopathy)*. **Important:** Patients with **HIV-associated ANUG** require gentle debridement and antimicrobial rinses.

Note: Histologically, **the deeper areas** of the **lesion** contain a zone of **spirochetal infection.**

Important: According to the American Association **of Periodontics** *(AAP)*, ANUG is now correctly referenced simply as **"Necrotizing Ulcerative Gingivitis"** without the "acute" **qualifier.** The National Boards may or may not reflect this change.

In a healthy sulcus, which bacteria below are **most** abundant?

- Actinobacillus actinomycetemcomitans and Bacteroides forsythus
- Streptococcus species and Actinomyces species
- Treponema species and Capnocytophaga species
- Prevotella intermedia and Porphyromonas gingivalis

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• **Streptococcus species and Actinomyces species**

***Gram-positive cocci** *(Streptococci)* and **filamentous bacteria** *(Actinomyces)* are most abundant in a healthy sulcus. Viridans streptococci consist of a variety of alpha-hemolytic streptococci, including S. salivarius, mutans, sanguis, and mitis, **all common oral flora.**

Normal Inhabitants of the Oral Cavity:

Gram-positive

- Streptococcus
- Peptostreptococcus
- Actinomyces
- Lactobacillus

Gram-negative

- Veillonella
- Fusobacterium
- Corynebacterium
- Campylobacter
- Eikenella

Important information:

1. The oral cavity is usually **sterile at birth.** Microorganisms appear about 10-12 hours after birth.
2. After one year, the **following bacteria** are present:
   - Streptococci
     - S. salivarius is **most abundant**
     - S. mutans and sanguis **do not** appear until teeth are present
   - Staphylococci
   - Neisseria
   - Actinomyces
   - Fusobacterium

***By the age of 4-5, the oral flora resembles that of an adult.**

Which **two organisms** are most commonly associated with the etiology of localized aggressive periodontitis *(formerly called juvenile periodontitis)*?

- Actinobacillus actinomycetemcomitans *(Aa)*
- Wolinella recta
- Capnocytophaga ochraceus
- Porphyromonas gingivalis
- Actinomyces israeli

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- **Actinobacillus actinomycetemcomitans** *(Aa)*
- **Capnocytophaga ochraceus**

Important: The **new classification system** for periodontitis is more descriptive and not as temporal as was the previous system. The terms adult, juvenile, early onset, and prepubertal **have been replaced** with various forms of chronic and aggressive disease. The term refractory periodontitis **has been removed** as a distinct disease entity, as the current thinking is that any type of periodontitis may be refractory.

Aggressive periodontitis *(formerly called Juvenile periodontitis)* occurs in **two** forms:

1. **Generalized form** *(formerly known as rapidly progressive periodontitis)* → prevotella intermedia and Eikenella corrodens predominate. It occurs between the ages of 12-25 and is characterized by **rapid, severe periodontal destruction around most teeth.** It is characterized by episodic, rapid, and severe attachment loss.

2. **Localized form** → gram-negative anaerobes **Actinobacillus Actinomycetemcomitans** *(Aa)* and **Capnocytophaga species** *(ochraceus)* predominate. Prevotella intermedia and Eikenella corrodens may also be present to a lesser extent. It occurs in an otherwise healthy adolescent *(12-19).* It is characterized by **rapid and severe attachment loss** confined to the **incisors and first molars.** The **one outstanding negative feature** is the relative absence of local factors *(plaque)* to explain the severe periodontal destruction which is present. Possible etiologic factors include a genetic predisposition or a **dysfunction of neutrophils** *(a chemotactic defect).*

Note: Aa and Capnocytophaga species *(specifically C . Ochraceus)* are also associated with periodontitis in juvenile diabetes.

A patient who presents with fiery red marginal and attached gingiva and demonstrates ulcerated and necrotic epithelium that sloughs *(or peels off)* with air blasts probably has:

- Periodontitis
- Acute necrotizing ulcerative gingivitis
- Desquamative gingivitis
- Hyperplastic gingivitis

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• **Desquamative gingivitis**

Desquamative gingivitis *(DG)* is a clinical term to describe red, painful, glazed and friable gingivae which maybe a manifestation of some mucocutaneous conditions such as lichen planus or the vesiculobullous disorders. It is important to be aware of this rare clinical entity so as to distinguish desquamative gingivitis from plaque induced gingivitis which is an extremely common condition, easily recognized and treated daily by the dental practitioner.

It is characterized by fiery red, glazed, atrophic or eroded looking gingiva. There is loss of stippling and the gingiva may desquamate easily with minimal trauma. As opposed to plaque induced gingivitis, DG is more common in middle-aged to elderly females, is painful, affects the buccal / labial gingiva predominantly, frequently spares the marginal gingiva but can involve the whole thickness of the attached gingiva and its clinical appearance is not significantly altered by traditional oral hygiene measures or conventional periodontal therapy alone. **Note:** The role of plaque is vague in desquamative gingivitis.

The majority of cases of DG are now known to be due to mucocutaneous conditions, in particular lichen planus, pemphigoid and pemphigus. Other causes include allergic reactions to toothpastes / mouth rinses, Crohn's disease, psoriasis, and chronic ulcerative stomatitis. DG can be mistaken for plaque induced gingivitis and this can lead to delayed diagnosis and inappropriate treatment of serious dermatological diseases such as pemphigoid or pemphigus.

Remember: Histologically, where non-ulcerated areas are found, the stratified squamous epithelium is significantly atrophic. Rete pegs are short or absent. Inflammatory cells, **mainly plasma cells,** may be found on the basal layer.

Which of the following statements regarding periodontitis is **incorrect?**

- Periodontitis **does not** always begin with gingivitis
- Gingivitis and periodontitis **cannot be induced** without bacteria
- There are **no** radiographic features of gingivitis
- The presence of pockets **cannot** be determined from radiographs
- Chronic gingivitis **does not** always lead to periodontitis

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• **Periodontitis does not always begin with gingivitis**

Periodontitis **always begins** as a gingivitis which is usually due to local irritation, primarily plaque, and the inflammation then spreads from the gingiva and soft tissues into the underlying structures. Gingivitis and periodontitis **cannot be induced** without bacteria *(plaque)*.

Periodontitis is inflammation that affects and **destroys the attachment apparatus.** The histology is marked by **apical migration** of the junctional epithelium from the CEJ, loss of connective tissue attachment, loss of periodontal ligament, and destruction of bone. This generally progresses slowly and painlessly. The progress of periodontitis may be arrested with proper therapy.

Remember: There are **no radiographic** features of gingivitis. **In periodontitis, radiographic changes are noted,** which may include the following: **1)** loss of lamina dura, **2)** horizontal or vertical bone resorption, and **3)** thickening *(widening)* of the periodontal ligament space.

Important: The presence of pockets **cannot be determined** from the evaluation of radiographs.

Note: Gingivitis **does not always** lead to periodontitis. Chronic gingivitis may exist for long periods without advancing to periodontitis.

The **junctional epithelium** in health is:

- A collar-like band of **stratified columnar epithelium** 2-5 cells thick near the sulcus and 1-2 cells thick at the apical end
- A collar-like band of **stratified squamous epithelium** 10-20 cells thick near the sulcus and 2-3 cells thick at the apical end
- A collar-like band of **simple columnar epithelium** 5-10 cells thick near the sulcus and 20-25 cells thick at the apical end
- A collar-like band of **pseudostratified columnar epithelium** 2-5 cells thick near the sulcus and 10-15 cells thick at the apical end

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- **A collar-like band of stratified squamous epithelium 10-20 cells thick near the sulcus and 2-3 cells thick at the apical end**

The **junctional epithelium in health** is usually .25 to 1.35 mm long. It is the stratified squamous, non-keratinized epithelium that surrounds the tooth like a collar. It is attached by one broad surface to the tooth and by the other to the gingival connective tissue. The junctional epithelium has 2 basal laminas, one that faces the tooth *(internal basal lamina)* and one that faces the connective tissue *(external basal lamina)*. The proliferative cell layer responsible for most cell divisions is located in contact with the connective tissue *(i.e., next to the external basal lamina)*. The desquamative *(shedding)* surface of the junctional epithelium is located at its coronal end, which also forms the bottom of the gingival sulcus. The junctional epithelium is more permeable than the oral or sulcular epithelium. It serves as the preferential route for the passage of bacterial products from the sulcus into the connective tissue and for fluid and cells from the connective tissue into the sulcus.

The term **epithelial attachment** refers to the attachment apparatus *(i.e., the internal basal lamina and hemidesmosomes)* that connects the junctional epithelium to the tooth surface. This term is **not synonymous** with junctional epithelium which refers to the entire epithelium.

In order for a new attachment to form **after periodontal treatment** the following must occur:
1. **Complete removal** of calculus, altered cementum, diseased junctional epithelium, and pocket epithelium
2. Need **undifferentiated mesenchymal cells**

Important: The junctional epithelium **in disease** ***(which is referred to as a long junctional epithelium)*** is different from the junctional epithelium in **health. In disease,** migration of the junctional epithelium occurs, along with degeneration in the connective tissue **under the** attachment; as the junctional epithelium proliferates along the root **surface** *(gets longer)* **the coronal** portion detaches. **Barrier membranes**, which are often used to treat **bony defects, help** to prevent **this long** junctional **epithelium from forming**.

A cuplike resorptive area at the crest of the alveolar bone is a radiographic finding of:

- Gingivitis
- Occlusal trauma
- Early periodontitis
- Acute necrotizing ulcerative gingivitis

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• **Early periodontitis**

Radiographic Changes in Periodontal Disease

Early periodontitis → areas of localized erosion of the **alveolar bone crest** *(blunting of the crest in anterior regions and a rounding of the junction between the crest and lamina dura in the posterior regions).*

Moderate periodontitis → the destruction of alveolar bone **extends beyond early changes in the alveolar crest** and may include buccal or lingual plate resorption, generalized horizontal erosion or localized vertical defects and possible clinical evidence of tooth mobility.

Advanced periodontitis → the bone loss is **so extensive** that the remaining teeth show excessive mobility and drifting and are in jeopardy of being lost. There is usually **extensive horizontal bone loss or extensive bony defects.**

Notes:

1. In **gingivitis,** the radiographic appearance of the bone will be normal.
2. The crest of the alveolar bone is affected in periodontal disease. **In health,** it lies 1-2 mm below the level of the CEJ's of adjacent teeth.
3. A reduction of only 0.5 or 1.0 mm in the thickness of the cortical plate is sufficient to permit radiographic visualization of destruction of the inner cancellous trabeculae.

Which of the following is most **significant** in regard to the **prognosis** of a periodontally involved tooth?

- Pocket depth
- Attachment loss
- Anatomical crown length
- Bleeding upon probing

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• **Attachment loss**

Attachment loss is **much more significant** than periodontal pocketing *(actually it is the most significant factor)* because with attachment loss **supportive structures are being destroyed.** Pocketing can increase or decrease, depending on the amount of inflammation without attachment loss. On the other hand, extensive attachment loss and gingival recession may be accompanied by shallow pockets *(poor prognosis of tooth).*

Important: The two most critical parameters for the prognosis of a periodontally involved tooth are **attachment loss** *(most critical)* and **mobility.**

Attachment level refers to the position of the junctional epithelium at the base of a sulcus or pocket. **In health,** the junctional epithelium is on enamel or at the CEJ. **In disease,** the junctional epithelium migrates apically along the root surface. It is measured from an established reference point *(CEJ or restoration margin)* to the attachment with a **periodontal probe.** The periodontal pocket is measured from a changeable point *(margin of the free gingiva)* to the attachment.

Note: Shallow pockets attached at the level of the apical third of the root suggests more severe destruction than deep pockets attached at the coronal third of the roots. When the gingival margin coincides with the cementoenamel junction, the loss of attachment equals the pocket depth.

Gingivitis is **most often** caused by:

- A hormonal imbalance
- Inadequate oral hygiene
- Occlusal trauma
- A vitamin deficiency
- Aging

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- **Inadequate oral hygiene** → this leads to the formation of bacterial plaque and its products which are the primary etiologic factors in gingivitis

In **acute gingivitis,** gram-positive organisms predominate. Specifically, **Actinomyces species** *(filaments)* and **Streptococci species** *(cocci)*.

In **chronic gingivitis,** gram-negative anaerobic organisms increase. Fusobacterium species, Prevotella intermedia , and Capnocytophaga species comprise 3/4 of the flora. Spirochetes become evident and Actinomyces species *(filaments)* are still present.

A freshly cleaned tooth surface is rapidly covered with a glycoprotein deposit referred to as a **"pellicle"**. The pellicle is derived from salivary constituents which are selectively adsorbed onto the tooth surface. Components of the dental pellicle include albumin, lysozyme, amylase, immunoglobulin A, proline-rich proteins and mucins. The formation of pellicle is the first step in plaque formation.

The pellicle-coated tooth surface is colonized by **gram-positive bacteria** such as Streptococcus sanguis, Streptococcus mutans, and Actinomyces viscosus. These organisms are examples of the **"primary colonizers"** of dental plaque. The **secondary colonizers** include gram-negative species such as Fusobacterium nucleatum, Prevotella intermedia, and Capnocytophaga species. The **"tertiary colonizers"** include Porphyromonas gingivalis, Campylobacter rectus, Eikenella corrodens, Actinobacillus actinomycetemcomitans, and the oral spirochetes *(Treponema species)*. The overall pattern observed in dental plaque development is a very characteristic shift from the early predominance of gram-positive facultative microorganisms to the later predominance of gram-negative anaerobic microorganisms.

Note: The major factor in determining the **different** bacteria is **oxygen. The redox** potential of the gingival sulcus **greatly influences** the bacterial **composition**.

Which of the following **needs to be evident** in order to make a **diagnosis of periodontitis?**

- Bleeding
- Pocket depths of 5 mm or more
- Radiographic evidence of bone loss
- A change in tissue color and tone

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- **Radiographic evidence of bone loss**

Note: Bitewing x-rays tend to be most accurate in assessing alveolar bone resorption. If extensive bone loss has occurred, **vertical bitewings** should be taken. They will reveal more of the periodontium. Also remember that more than **30%** of the bone mass at the alveolar crest must be lost for a change in bone height to be recognized on radiographs.

Other clinical criteria that should be **evaluated during a periodontal exam** *(besides color, tone, contour and size of gingiva):*

- **Level of the free gingival margin in relation to the CEJ** → the normal level of epithelial attachment should be on enamel or at the CEJ. This would place the free gingival margin 2 to 3 mm coronal to this sulcular base.
- **Periodontal pocket depth** → all measurements in excess of 3 mm are recorded for sulcular depth, as well as any reading which locates the free gingival crest less than 2 mm at or below the CEJ. ***The loss of attachment** is determined by measuring the distance between the CEJ and the base of the attachment.
- **Bleeding** → physiologically, bleeding from the gingival sulcus should not be caused by gentle provocation. Bleeding in the **absence of local irritants** may indicate a systemic disease.
- **Exudate** → the presence of exudate, **specifically suppuration,** is evaluated by digital pressure on the buccal and lingual of each **tooth.**
- **Mucogingival complications** → this term **indicates** the involvement of not only the gingival component in the disease state, but also **the presence** of imminent involvement of the alveolar mucosa.

Pseudopockets are most likely to be seen in a patient with:

- Acute gingivitis
- Advanced periodontitis
- Inflammatory gingival enlargement
- Desquamative gingivitis

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• **Inflammatory gingival enlargement**

Clinically, this form of gingivitis is easily differentiated from simple gingivitis. Notable clinical findings include an increase in gingival size, distortion of normal form and change in tissue tone. There is a **significant increase** in sulcular depth. Pocket formation occurs. This pocket is classified as a **pseudo** or **relative pocket** since it is caused by expansion of the marginal tissue coronally **rather than** apical movement of the epithelial attachment beyond its physiological level. Histologically, there is a greater degree of sulcular proliferation through rete pegs than is noted in simple gingivitis.

Hereditary gingivofibromatosis it is a rare genetic disease. Clinically, there is generalized diffuse gingival enlargement, often extensive enough to cover the teeth. The tissue is dense and firm with considerable distortion of normal contour. Gingival color is normal, but erythematous changes are a result of secondary bacterial involvement. **There is a striking lack of inflammatory cells, proliferating capillaries and vascular engorgement** commonly seen with most types of gingivitis.

Note: Gingivectomy may be used to correct the gingival contours for both of the above.

Remember: Medications *(specifically dilantin, cyclosporine A and nifedipine)* cause the highest incidence of fibrous gingival hyperplasia.

Important: A pseudopocket is a pocket **formed by gingival** enlargement **without** apical migration of the **junctional epithelium**. It does not **involve the loss** of bone. Pseudopockets are also referred to as **gingival, false, or** relative pockets. **All pseudo**pockets are **suprabony** ***(the base of the pocket is coronal to the crest of the alveolar bone).***

The most important **plaque retentive factor** is:

- Subgingival and / or overhanging margins of restorations
- Carious lesions that extend subgingivally
- Calculus
- Furcations exposed by loss of attachment and bone
- Crowded and mal-aligned teeth
- Root grooves and concavities

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- **Calculus**

The microbiologic etiologic factor in periodontal diseases is dental plaque with dental calculus as probably the most significant local contributing factor.

Other contributing or complicating factors in **periodontal disease** include:

- **Food impaction or retention** → overlapping, malposed, tilted or drifted teeth are frequently associated with food impaction or retention. If not removed, this will lead to inflammatory periodontal disease.
- **Open and loose contacts** → leads to food impaction and possible retention.
- **Overhanging margins of restorations and improperly designed prostheses** → can provoke or initiate periodontal disease. There is a direct correlation between **surface roughness or marginal irregularities of a tooth and the retention of plaque.**
- **Soft or sticky consistency of diet** → food debris tends to collect between the teeth and along the gingiva and can be a prominent cause of inflammation.
- **Violation of the "biologic width"** → if restorative materials render the invasion of the biologic width permanent, periodontitis will produce apical migration of the junctional epithelium.
- **Occlusal traumatism**

Gingival changes evident **during pregnancy** probably result from the effect of:

- Estrogen
- Progesterone
- Histamine
- A vitamin deficiency

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• **Progesterone** → probably, but not definitely

***As well as **an increase** in the number of mast cells which are found throughout the gingival tissues.

It should be **stressed** that no gingival pathology will be found during pregnancy, providing no etiological factors accumulate *(plaque)* or are not present prior to the pregnancy.

Pregnancy gingivitis is a nonspecific gingivitis and definitive diagnosis is made on the physical state of pregnancy. **There is an exaggerated and modified response of the gingiva to local factors** *(plaque)*. There is considerable loss in tissue tone as well as a change in color *(bright red)* with marginal rolling and papillary enlargement which obliterates the embrasure spaces. If the patient is in her first or second trimester, scaling and polishing along with OHI can be performed. If she is well along into her third trimester, the prudent treatment may be just giving OHI and reappointing after childbirth for scaling and polishing.

Note: Gingival hemorrhage to **gentle pressure** is the prominent clinical finding.

Pregnancy gingivitis has been shown **to be** associated with increased levels of **Prevotella intermedia** in the inflamed **sites. These** bacteria crave steroid hormones *(for example, Progesterone)* for their own **metabolism**.

In the **chronic** stage of gingivitis:

- Mast cells predominate
- Plasma cells predominate
- Lymphocytes predominate
- Macrophages predominate

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• **Plasma cells predominate**

Three stages of disease in developing gingivitis:

1. **Transient** *(incipient)* **stage** → within 2 to 4 days after cessation of oral hygiene, the earliest changes are visible microscopically. These consist of a margination of **leukocytes** *(PMN's)* in vessels close to the junctional epithelium. Sloughed epithelial cells and bacteria are found in the gingival sulcus.
2. **Developing stage** → the area of collagen destruction becomes larger and is occupied by fluid that contains serum proteins: fibrin; **immunoglobulins, especially IgG**; complement; inflammatory cells, **principally lymphocytes** *(B or T cells);* and **macrophages.**
3. **Chronic stage** → the cytologic characteristics of the inflammatory infiltrate in the gingival lamina propria are changed. In the developing stage, lymphocytes were the predominant cell; now, **plasma cells predominate.** IgG is produced by most of the plasma cells, with a few cells present containing IgA *(mostly in saliva)*; IgM-containing cells are rarely seen.

Note: The four stages of the **periodontal lesion** are: **initial, early, established and advanced.**

Significant levels of immunoglobulins are found in both epithelial and connective tissue compartments. **Although** severe periodontal **disease** has been reported in individuals suffering **abnormalities** in neutrophil function **or neutropenia**, increased or more severe periodontal **disease has not** been reported **in young** persons in **which other** immune functions are compromised, **such as** T or B cell **deficient** states.

People suffering from diabetes type 1 and 2:

- Have no chance of developing periodontal disease
- Have just as much a chance of developing periodontal disease as the nondiabetic population
- Have 15 times the risk of developing periodontal disease as compared to the nondiabetic population
- Have only a slight risk of developing periodontal disease

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- **Have 15 times the risk of developing periodontal disease as compared to the nondiabetic population**

Much evidence exists on the link between diabetes type 1 and 2 and periodontal disease. People with these diseases have 15 times the risk of the nondiabetic population. Diabetes causes abnormalities in blood vessels and high levels of specific inflammatory chemicals, such as interleukins, that significantly increase the chances of periodontal disease. High levels of triglycerides *(which are common in type 2 diabetes)* appear to impair periodontal health.

Although the bacterial and immune factors play a major role, genetic, systemic and functional factors are also involved in the development of inflammatory periodontal disease or exacerbation of an existing disease. Genetic factors may predispose to or even trigger periodontitis. Factors governing **leukocytic levels,** migration and chemotaxis may predispose the individual to infection and influence host response. Individuals with a defective immune response as seen in **agranulocytosis** and **neutropenia** are notably susceptible to crestal alveolar bone loss and abscesses. Autoimmune conditions *(e.g., Crohn's disease, rheumatoid arthritis, lupus erythematosus, CREST syndrome)* have been associated with a higher incidence of periodontal disease. Other diseases associated with periodontitis include leukemia and other cancers, tuberculosis, syphilis, Wegener's granulomatosis, amyloidosis, and many genetic disorders.

Agranulocytosis is an abnormal condition of the blood. It is characterized by a severe reduction in the number of granulocytes, resulting in fever, prostration and bleeding ulcers in the mouth, rectum and vagina.

Neutropenia is an abnormal decrease in the **number of neutrophils** in the blood. It is associated with acute leukemia, infection, rheumatoid arthritis, Vitamin **B12 deficiency** and chronic splenomegaly.

Leukemias are malignant neoplasms of immature **white blood** cells. Patients with **acute leukemias** have more **oral complications than** those with chronic. **The gingiva** is grossly **enlarged and is** bluish-red in color. It has a soft, spongy **consistency** and the papillae **are blunted**. **See picture #21 and #22 in booklet.**

The **frequency of maintenance visits** for a patient who has had previous periodontal treatment should be dependent upon which **two factors?**

- On whether or not the patient feels that frequent visits will help maintain his / her periodontium
- On the appearance and clinical condition of the gingival tissues
- On the amount of attachment loss prior to the periodontal treatment
- On the ability and performance in home care

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- **On the appearance and clinical condition of the gingival tissues** → this will determine if the patient is maintaining adequate plaque control
- **On the ability and performance in home care** → this will determine the effectiveness of the patient's oral hygiene

The **main goal** of the dentist, dental hygienist and patient is the maintenance of good oral health. This will **prevent the recurrence of disease,** which is the main objective of the maintenance phase of periodontal therapy. The primary cause of recurrence of disease is the dental team's failure to motivate the patient to practice effective plaque control.

The **first year after treatment** is a critical period, since the patient has already demonstrated susceptibility to periodontal disease, the cause of which tends to be persistent and recurrent. The **appearance and condition** of the gingival tissues will determine if the patient is maintaining adequate plaque control.

Notes:

1. Bleeding during circumferential probing **indicates** that the crevicular epithelium is ulcerated due **to active periodontal disease.**
2. After periodontal treatment the first **recall visit** should be scheduled at three months. With excellent plaque control and **maintenance** of periodontal health, the interval may be lengthened **to 4 - 6** months.

Which of the following cells **participates in the early phase of inflammation,** for example, the early lesion of gingivitis?

- Eosinophils
- Kupffer's cells
- Mast cells
- Epithelioid cells

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• **Mast cells**

These cells are found in connective tissue, contain numerous basophilic granules, and release substances such as **heparin and histamine** in response to injury or inflammation of bodily tissues. The major storage sites of histamine are **mast cells, platelets, and basophils.** Mast cell histamine is released when tissues are damaged. Histamine is important in the **vascular phase** of acute inflammation *(causing vasodilation and increased vascular permeability).*

The mast cell content in **human gingiva is high.** The mast cell content of inflamed gingiva increases as the severity of inflammation increases.

Remember: The anaphylactic response is characterized by the degranulation of mast cells as a result of antigen-antibody complexes affixed to cell surfaces.

The first leukocytes to arrive as a **result of inflammation** caused by plaque formation in the initial lesion of gingivitis are the:

- Eosinophils
- Lymphocytes
- Neutrophils *(polymorphonuclear neutrophils)*
- Basophils
- Monocytes

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• **Neutrophils** ***(polymorphonuclear neutrophils)***

***They are also called neutrophilic leukocytes.

The **initial lesion** begins within 2-4 days after deprivation of oral hygiene. It exhibits neutrophils in the junctional epithelium, which are in the process of egressing into the gingival crevice. They are the **first line of defense** and migrate toward the injured site *(gingiva)* as a result of the chemotactic properties of plaque.

As the **gingivitis develops** *(early lesion – 4 to 7 days)*, the area of destruction becomes larger with persistence of inflammation. Leukocytes invade connective tissue and are dominated by lymphocytes *(3/4 of all cells)*, macrophages, plasma cells *(secrete IgG)*, and mast cells *(release histamine)*.

As the gingivitis reaches the **chronic stage** *(established lesion, time varies from weeks to months or years)*, there is an increase in plasma cells *(still secreting IgG)* and B lymphocytes. These cells invade deep into the connective tissue. ***This stage may persist for years with or **without going** to the advanced lesion or periodontitis.

When gingivitis progresses to **periodontitis** *(advanced lesion)*, lymphocytes, plasma cells, and macrophages continue to invade the connective tissue along vascular pathways and destroy the gingival fibers. Changes within the supporting bone occur as the inflammatory process continues.

Remember: Neutrophils are the **most numerous cells** in the **inflammatory** exudate of an **acute periodontal abscess.**

The **vascular phase** of acute inflammation involves all of the following cells **except:**

- Platelets
- Tissue mast cells
- Eosinophils
- Basophils

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- **Eosinophils**

***Eosinophils are the predominant inflammatory cells in **allergic reactions and parasitic infections.** The **two phases** of acute inflammation are:

1. **Vascular phase**
   - **Vasoconstriction***(temporary)* → narrowing of blood vessels caused by contraction of smooth muscle in the vessel walls, which can be seen as blanching *(whitening)* of the skin.
   - **Vasodilation**→ widening of the blood vessels to increase the blood flow to the infected area.
   - **Increased vascular permeability** → which allows diffusible components to enter the site.
2. **Cellular phase**
   - **Leukocytes** *(predominantly polymorphonuclear neutrophils)* are the first defense cell to migrate to the injured tissue by chemotaxis. These leukocytes engulf particulate matter by phagocytosis. This engulfed matter becomes a **phagosome** and then combines with lysosomal granules to form a **phagolysosome,** in which digestion of the engulfed particle occurs.

Note: $C5_a$ and leukotriene B_4 *(LTB_4)* have been identified as chemotactic factors for neutrophils.

Cells involved in **acute** inflammation include:

- Basophils, tissue mast cells, and platelets are important in the **vascular** phase. These cells release histamine.
- **PMNs** predominate in the **cellular phase.** Macrophages appear late in the cellular phase and represent a **transition** between acute and chronic inflammation.

Which local sign of acute inflammation is caused by the **dilation of capillaries?**

- Redness
- Heat
- Swelling
- Pain

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• **Redness**

Acute inflammation is the **initial response** of tissue to injury. It involves vascular and cellular responses. Polymorphonuclear neutrophils are the **most abundant cells initially.**

The four local signs of acute inflammation are:

1. **Redness** *(rubor)* → due to **dilation of capillaries**
2. **Heat** *(calor)* → capillary dilation permits **increased blood flow** through vessels with the associated high metabolic activity of neutrophils and macrophages
3. **Swelling** *(tumor)* → caused by **increased capillary permeability**
4. **Pain** *(dolor)* → **due to lysis of blood** cells that triggers the production of bradykinin and prostaglandins

***The above signs are usually accompanied by a **loss of function.**

Note: Systemic effects include fever, tachycardia, and leukocytosis *(especially neutrophils).*

Chronic inflammation is the tissue response to injury that persists longer than several days. There is an accumulation of lymphocytes, plasma cells, and **mostly macrophages.** Fibrosis occurs which causes progressive tissue damage and loss of function.

Notes:

- **Inflammation** is the protective response **of the tissues** of the body to irritation or injury.
- **Pain,** which is a cardinal symptom of **inflammation,** may result from histamine and bradykinin release **as well as** from direct **nerve damage** or **pressure by the** tissue exudate.

The characteristic cell components of **chronic inflammation** include all of the following **except:**

- Lymphocytes
- Plasma cells
- Polymorphonuclear leukocytes
- Macrophages

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• **Polymorphonuclear leukocytes**

***Remember:** PMNs *(polymorphonuclear neutrophils)* are the most prominent inflammatory cells in acute inflammation. They predominate in the **cellular phase.**

Acute inflammation is the initial response of tissue to injury, particularly bacterial infections, involving vascular and cellular responses.

Three **major phenomena** occur:

1. **Increased vascular permeability** → inflammatory **tissue exudate** is formed because of the increased permeability of the blood vessels at the inflammatory site *(edema).*
2. **Leukocytic cellular infiltration** → **neutrophils** mostly, caused by chemotactic agents $C5_a$ and $C3_a$. They function to phagocytize bacteria and elaborate proteolytic enzymes.
3. **Repair** → either by **regeneration or replacement.**

Note: → **Chronic inflammation** compared to acute inflammation:

- **More proliferative** than exudative
- **Macrophages** predominate ***(with some** plasma cells)*
- **Necrosis** occurs and recurs ***(as opposed to regeneration)***

Extrinsic dental stains include:

- Green to brown stains caused by erythroblastosis fetalis
- Brown, black, green, or orange stains caused by chromogenic bacteria in plaque
- Red to brown stain caused by congenital porphyria
- Gray or brownish stains caused by tetracycline

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- **Brown, black, green, or orange stains caused by chromogenic bacteria in plaque**

Stains can be identified by **location:**
- **Intrinsic** – stains that occur within the tooth substance
- **Extrinsic** – stains that occur on the external surface of the tooth

Stains can be identified by **sources** of the discoloration:
- **Endogenous** - stain that originates from within the tooth. Endogenous stains are always intrinsic and frequently are discolorations of the dentin reflected through the enamel. **Examples of sources:** drugs *(tetracycline, systemic fluoride)*, changes in pulp tissue of pulpless teeth, imperfect tooth development *(amelogenesis imperfecta)*
- **Exogenous** - stain that originates from an external source.

Brown, black, green and orange stains are generally seen on the labial surface of anterior teeth and are usually caused by **poor oral hygiene.** Some theories attribute the change in color to by-products of some bacteria. Black stain is generally seen in the cervical portion of molar teeth in children. Green and orange stains are supposedly associated with an increased amount of caries and actually they represent pigmentation of dental plaque. Black line, tobacco, orange and green stains are all **exogenous extrinsic stains,** initially at least. After a period of time, both green and tobacco stains may become incorporated with the tooth. At this point, their classification changes. They become **exogenous intrinsic stains.**

Silver amalgam and topical fluoride are **also examples** of exogenous **intrinsic stains.** An exogenous **intrinsic stain** is one that originates **from a** source outside the tooth and subsequently becomes incorporated **within** the tooth **structure.**

All of the following are diagnostic of occlusal trauma **except:**

- Thickening of the lamina dura
- Periodontal pocket formation
- Widening of the periodontal ligament space
- Angular bone destruction
- Increase in tooth mobility
- Fremitus
- Wear facets

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- **Periodontal pocket formation**

***Periodontal pockets **are not caused** by occlusal trauma. A local irritant and inflammation are necessary to cause apical shift of the epithelial attachment.

The **most common** clinical sign of occlusal trauma is **tooth mobility.** Other clinical signs of occlusal trauma include migration of teeth and the tenderness of teeth to percussion.

Radiographic signs of trauma from occlusion:

- Widening of the periodontal ligament space
- Sometimes thickening of the lamina dura
- Angular bone loss and infrabony pocket formation
- Root resorption
- Hypercementosis

Trauma from occlusion is **reversible,** that is, the body can repair the damage if the excessive occlusal forces are eliminated.

Other findings **associated** with excessive occlusal forces:

- Alternating areas of resorption and repair of the alveolar bone
- Fibrosis of the alveolar bone marrow spaces
- Cemental resorption leading to dentinal resorption
- Cemental tears
- Possible ankylosis
- Occasional pulpal necrosis and calcification

Radiographic changes that may be seen on teeth that are no longer in function:

- Reduced trabeculation of bone
- Narrowing of the periodontal ligament space

The purpose of using **rosin** in the periodontal dressing is to:

- Serve as an astringent
- Improve the taste
- Act as a filler for strength
- Provide an antibacterial property

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- **Act as a filler for strength**

Purpose of the periodontal dressing:

- Provide mechanical protection for the surgical wound and therefore facilitate healing
- Prevents post operative bleeding by keeping the initial clot in place
- Supports mobile teeth during healing
- Helps in shaping or molding the newly formed tissue
- Provide patient comfort by isolating area from external irritations or injuries

Characteristics of acceptable dressing material:

- Should be nontoxic or nonirritating to the tissue
- Conveniently prepared, place and removed with minimal discomfort to patient
- Should maintain adhesion to itself and to the teeth and tissues
- Should not damage or stain any restorative materials

Types of Dressings:

- Eugenol dressing *(hard pack)*
  - Basic ingredients: powder & liquid *(eugenol)*
  - Brand names: PPC, Wards
- Light-cure
  - Basic ingredients: syringe
  - Brand names: Barricaid
- Non-eugenol *(soft pack)*
  - Basic ingredients: base & accelerator
  - Brand names: Coe-Pak

Notes:

1. Periodontal dressings have **no well-defined effect on the** processes of wound healing or on surgical outcomes *(i.e., gains of periodontal attachment or the reduction in probing depths).*
2. For any type of dressing, the first rule is to **follow the** manufacturer's instructions.
3. Dressings **should be** removed within 7 to **10 days**.
4. Before **removing, make** sure sutures are **not embedded** in the dressing **and that the** dressing is not locked **interproximally.**

Which **one** of the following will reduce the **abrasive action** of a polishing agent?

- Using an agent with nice large particles
- Using an agent containing particles that are dull and round
- Using a thick, viscous mixture of polishing paste
- Applying the polishing agent with firm pressure and increasing to a heavy constant pressure
- Polishing at a high speed with heavy pressure

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- **Using an agent containing particles that are dull and round**

***Using a **thin, watery mixture** of polishing paste or polishing at a **low speed with light pressure** will also reduce the abrasive action of a polishing agent.

The use of abrasive polishing agents and / or a rotary polishing instrument may be **contraindicated** in the following cases:

- **Patient with a communicable disease** → the production of aerosols is likely to occur, which may disseminate the disease
- **Patient with respiratory problems**
- **Patient with "green stain"** → usually the tooth surface under the stain is demineralized
- **Patient with newly erupted teeth** → have not mineralized completely yet
- **Any patient who is at risk for dental caries** → those with xerostomia, amelogenesis imperfecta, rampant caries, or receiving radiation therapy to the head

***The use of a porte-polisher *(manual polisher)* may be helpful in some of the cases list above.

***Dental tape and finishing strips may be used to polish interproximal tooth surfaces.

Notes:

1. When selecting a polishing agent to remove generalized staining, **consider the following:** tooth sensitivity, type of stain present, **type of restorations** present and the condition of the tooth surface.
2. Flexing the **polishing cup** into proximal **areas increases its effectiveness.**

The collagen found in the gingiva:

- Is mostly of type I
- Is biochemically similar to the collagen found elsewhere
- Has a turnover rate which is as rapid as in the periodontal ligament
- Accounts for 90% of the gingival protein

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• **Type I**

***All other statements are **incorrect.**

Notes:

1. Most of the fibers in gingival connective tissue are composed of collagen. The bulk of the collagen is **type I** collagen, the most abundant form of collagen in the human body.
2. The type I collagen of gingiva, however, is **not the same** biochemically as found in other parts of the body, including the skin.
3. The collagen turnover in normal gingiva **is not as rapid as in the periodontal** ligament but significantly greater than in other tissues such as the skin, tendons, and the palate.
4. Collagen accounts for **about 60%** of gingival protein. The other gingival proteins are oxytalin, elaunin, and elastin.

Remember: Vitamin C is needed for hydroxylation of proline and lysine essential for collagen formation.

Type I collagen fibrils are normally **organized** into bundles of fibrils, or fibers. They are found throughout the lamina propria *(**the dense** connective tissue beneath the masticatory mucosa)*. **Type III** collagen fibers **are thinner** than the type I fibers and tend to be found close to basal **laminas** of vascular **channels** and epithelial **tissues.** They are also known as **reticular fibers.**

The most important procedure in the treatment of a localized periodontal abscess is to:

- Prescribe an antibiotic
- Create drainage of the inflammation through the pocket or by an incision
- Have the patient rinse twice daily with warm saline
- Leave it alone and let it clear up by itself

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• **Create drainage of the inflammation through the pocket or by an incision**

Periodontal abscesses *(lateral periodontal abscess)* can be acute or chronic. Acute abscesses are painful, edematous, red, shiny, ovoid elevations of the gingival margin and / or attached gingiva. After their purulent content is partially exuded, they become chronic. Chronic abscesses may produce a dull pain and may at times become acute.

Treatment for an **acute periodontal abscess is** determined initially by whether there is localization of the abscess. If there is, then the treatment is to **provide drainage.** If it is **not localized,** the patient should be placed on antibiotics *(Pen V or Clindamycin)* and instructed to rinse with warm saline.

The tissue destruction at periodontal abscess formation is rapid and it is therefore important treatment is undertaken as soon as possible. The principle for treatment is to establish drainage of the inflammatory process and to eliminate the abscess provoking agent. Often drainage of the lesion can be obtained by scaling and root planing through the periodontal pocket.

The **most prevalent symptom** that the patient will report with a periodontal abscess is one of acute pain. The nature of the pain is a constant, severe, dull throbbing. **Thermal changes do not seem to elicit or modify the discomfort.** The onset of this discomfort is rapid and becomes progressively more intense. The patient may also notice an increase in mobility of an individual tooth. The patient probably will say that it is difficult to close his / her teeth together without striking the involved tooth first, causing increased pain.

The radiographic findings associated with this abscess are not specific. There may be no change radiographically in the early acute lesion. Often, however, there will be a localized discrete radiolucency lateral to the root or in a furcation. Note: it can cause rapid alveolar bone destruction.

All of the following are clinical signs of bruxism **except:**

- TMJ symptoms
- Muscle soreness
- Periodontal pocket formation
- Cracked teeth or fillings
- Wear facets on teeth
- Widened PDL spaces on radiographs

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- **Periodontal pocket formation** → periodontal pockets are inflammatory lesions that are not caused by occlusal trauma or bruxism

Bruxism is defined as aggressive, repetitive, or continuous grinding, gritting, or clenching of the teeth during the day and / or night in other than functional activities such as chewing or swallowing.Opinions differ as to the **primary cause of bruxism,** but occlusal prematurities, muscle tension and emotional factors have been implicated. Bruxism **can be treated** through behavioral, emotional and interceptive modalities.

Occlusal splints / night guards can be an effective, inexpensive treatment for patients that are bruxers. Uses of a night guard include: **(1)** To stabilize the dentition, **(2)** To modify the nature of the habit *(bruxism),* **(3)** To act as a splint after periodontal surgery, **(4)** To assist in controlling recurrence of drug-induced gingival enlargement. **Note:** Night guards are usually worn at night, but they may also be worn during the day.
***The **primary purpose** of a night guard in periodontal trauma is to modify and control bruxism or to redirect forces into a non-traumatic pattern.

Night guards should have the following characteristics:
- They should be made of **hard acrylic.**
- There should be **no sharp edges** → **needs to be** comfortable.
- They should **fit well and cover the occlusal surfaces** of the maxillary teeth → do not cover the palate.
- They should **occlude evenly** with **mandibular** teeth and permit the patient to move the mandible **freely in all** excursions.

Which of the following are **contraindications** to selective grinding in the natural dentition?

- When pulp chambers are large
- In the presence of tooth sensitivity
- When major occlusal discrepancies may require orthodontics or full mouth reconstruction
- In patients who are poor candidates for full mouth reconstruction because of psychologic factors
- All of the above

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- **All of the above**

General objectives in **selective grinding** in the natural dentition:

- To achieve a **more favorable** direction and distribution of forces
- To coordinate the median occlusal position with the **terminal hinge position of the mandible**
- To **eliminate prematurities in excursive movements** to either gain a group function or canine protected occlusion
- To direct occlusal forces centrally along the **long axis of the tooth**
- To **improve or maintain** masticatory performance
- To accomplish the occlusal adjustment **without reducing vertical dimension and by retaining** an acceptable interocclusal distance
- To **reduce** or **eliminate** fremitus

The **usual steps** of occlusal adjustment:

- Eliminate prematurities in **centric relation**
- Eliminate prematurities in **protrusive movements**
- Eliminate prematurities in **lateral excursive movements**
- **Re-establishment** of **physiologic occlusal anatomy** and careful polishing of all ground surfaces

Which of the following is **not correctly matched** with regards to a periodontal treatment plan?

- Preliminary Phase → Plaque Control
- Phase I → Mouth Preparation
- Phase II → Periodontal surgery
- Phase III → Restorative Phase
- Phase IV → Maintenance Phase

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- **Preliminary Phase → Plaque control**

***Plaque control** is a part of Phase I or initial therapy. The preliminary phase includes treatment of **emergencies only** whether they be pulpal, periodontal or any other.

Phase I *(initial therapy)* of periodontal treatment planning includes:

- **Extraction of hopeless teeth**
- **Mouth preparation**
  - Initial scaling *(full mouth)*
  - Definitive root planing
- **Oral hygiene instructions:** This is the most important aspect of initial therapy. The dental hygienist or dentist **teaches, motivates, and guides** the patient in the performance of measures for disease control. During a prophy appointment, the hygienist or dentist can **establish a rapport** with the patient and individualize instruction for the particular patient. Show the proper brushing and flossing techniques and have them repeat these techniques to see if they understand what you are showing them.
- **Occlusal adjustment**
- **Night guards** *(if bruxism exists)*
- **Splinting** (*stabilizing loose teeth)*
- **Re-examination** *(charting with probings)*

Remember: If oral hygiene is poor, surgery is contraindicated!!!

When assessing **tooth mobility,** a score of 1 indicates:

- No detectable movement
- Barely distinguishable tooth movement
- Any movement up to 1 mm
- Any movement more than 1 mm or teeth that can be depressed or rotated in their sockets

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• **Barely distinguishable tooth movement**

Tooth mobility is defined as movement of a tooth in a horizontal or vertical plane of space. All teeth have some degree of mobility related to the width of the periodontal ligament, root attachment area, elasticity of the alveolar process, and function of the tooth. Teeth normally have a certain range of mobility. Single-rooted teeth are more mobile than multirooted ones, and incisors have the most mobility. Mobility is measured by the examiner pushing the tooth gently in a faciolingual direction using the blunt ends of two metal instruments. The use of a finger **is not acceptable** when assessing mobility.

Mobility is scored from 0 to 3:

- A mobility of 0 indicates a tooth has no mobility at all.
- A mobility of 1 indicates barely distinguishable tooth movement.
- A mobility of 2 indicates any movement up to 1 mm.
- A mobility of 3 indicates any movement more than 1 mm or teeth that can be depressed or rotated in their sockets.

Mobility beyond the physiologic range is termed **abnormal or pathologic.** It is pathologic in that it exceeds the limits of normal mobility values; the periodontium may not necessarily be diseased at the time of examination.

Increased tooth mobility may be caused by a variety of factors, including pregnancy, diseases *(local and systemic)*, trauma *(including orthodontic movement)*, and hypofunction or hyperfunction. The two factors most often seen would be plaque induced inflammatory disease and excessive occlusal forces *(bruxism)*.

All of the following statements **concerning B cells** are true **except:**

- They **mature** in the bone marrow and migrate to lymphoid organs
- They are found in the **germinal centers** of the spleen and lymph nodes
- They are **progenitors** of plasma cells
- They are **involved in** humoral *(antibody-mediated)* and cell-mediated immunity

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• **They are involved in humoral** *(antibody-mediated)* **and cell-mediated immunity**

***This is **false;** B cells **are not** involved with cell-mediated immunity.

B cells *(lymphocytes)* are white blood cells that complete maturation in the bone marrow and migrate to lymphoid organs. These lymphocytes are committed to differentiate into antibody-producing plasma cells involved in **antibody-mediated immunity.** When an immature B cell is exposed to a specific antigen *(they recognize antigen by membrane-bound immunoglobulin),* the cell is activated. It then travels to the spleen or the lymph nodes, differentiates, and rapidly produces plasma cells and memory cells.

Remember: Plasma cells synthesize antibodies.

T cells *(lymphocytes)* are white blood cells that complete maturation in the thymus and become thymocytes. They are important in **cell-mediated immunity,** Type 4 hypersensitivity reactions *(contact dermatitis),* and in the modulation of antibody-mediated immunity. **Major classes include** helper T cells, suppressor T cells, and cytotoxic *("killer")* T cells.

Note: Patients with periodontitis **often have** T-lymphocytes sensitized to plaque bacterial antigens.

Cellular cementum*(contrasted with acellular cementum)* is:

- Cementum that is resorbing
- Newly formed cementum
- That part of the cementum that is adjacent to the enamel
- Cementum that contains cementocytes

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- **Cementum that contains cementocytes**

Cementum may be classified in the following ways:

1. **By location:**
   - **Radicular cementum** → the cementum that is found on the root surface.
   - **Coronal cementum** → the cementum that forms on the enamel covering the crown.

2. **By cellularity:**
   - **Cellular cementum** → cementum containing cementocytes in lacunae within the cementum matrix. It occurs more frequently on the **apical third of the root** and in **furcations.** It is usually the thickest to compensate for attritional wear of the occlusal / incisal surface and passive eruption of the tooth.
   - **Acellular cementum** → cementum without any cells in its matrix. Usually predominates on the **coronal two-thirds** of the root. It is thinnest at the CEJ. It plays a **major role** in tooth anchorage.

If vital cementum is **resorbed or nicked in surgical procedures,** the defect is repaired by the deposition of new cementum. Repair cannot occur where pockets are present or where the gingiva has receded and the cementum **is exposed.** Cementum that has become exposed, forming part of the clinical crown, is often **removed** in the course of scaling, root planing, or brushing.

Note: The thickness of radicular cementum increases with age. It is thicker apically than cervically. Thickness may range from 0.05 to 0.6 mm.

Which of the following is **not** true of cementum?

- It is produced by cells of the periodontal ligament
- The **main** function is to compensate for tooth wear
- The deposition of new cementum continues periodically throughout life whereby root fractures may be repaired
- The cementum is indistinguishable on radiographs
- It is lighter in color than dentin, contains 40 to 50% inorganic substance, and is permeable

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• **The main function is to compensate for tooth wear**

***This is **false;** the main function is the attachment of principal fibers of the PDL.

Other functions include:

1. **Compensates** for the loss of tooth surface due to occlusal wear by apical deposition of cementum throughout life.
2. **Protects** the root surface from resorption during vertical eruption and tooth movement.
3. Has a **reparative function,** allows reattachment of connective tissue following periodontal treatment.

In the cementum there are **two types** of collagen fibers:

1. **Sharpey's fibers:** are the terminal portions of the principal fibers of the PDL that are embedded in the cementum *(run perpendicular to the cementum)* on one end and alveolar bone on the other end. They serve to attach the tooth to surrounding bone.
2. **Type I collagen fibers:** are within the cementum itself and run parallel to the surface of the cementum.

Note: Unlike bone, which is continuously remodelled, the cementum grows slowly in thickness, throughout life, by apposition of new cementum at the surface. While cementum can be resorbed, it is not continuously remodelled like bone.

Which of the following is **least** affected by occlusal trauma?

- Alveolar bone
- Gingival attachment
- Periodontal ligament
- Cementum

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***This is **false;** there is **significant** probing depths.

***Important:** The signs and symptoms **are not always** consistent. Each case may or may not present with all of these signs and symptoms. In the presences of both a chronic endodontic and periodontal lesion, both lesions must be treated in order to achieve complete healing.

Treatment may include:

- **Root canal** therapy *(RCT)* → the pulp is treated first and the the periodontal condition is re-evaluated 2-3 months after the completion of endodontic therapy.
- Course of **antibiotic therapy**
- **Scaling** and **root planing**
- Periodontal **surgery if needed** is performed 2-3 months after the completion of RCT.

Remember:

1. A **periodontal cyst** is usually asymptomatic and without grossly detectable changes, but it may present as a localized tender swelling. Radiographically, an interproximal periodontal cyst appears on the side of the root as a radiolucent area bordered by a radiopaque line. Its radiographic appearance **cannot be differentiated** from that of a periodontal abscess.
   The **apical periodontal cyst** has a predilection for the **mandibular canine-premolar area.**
2. Teeth are **vital,** usually asymptomatic **but may be** slightly uncomfortable. There are **no periodontal pockets;** radiographs reveal **a small, well-**defined oval to round radiolucency on the lateral surface of the root. Treatment **includes** surgical removal **without** extraction of surrounding teeth.

PERIODONTICS

Which of the following are **primary reasons** for splinting teeth with a fixed prosthesis?

A. To stabilize loose teeth in a favorable occlusal relationship

B. To distribute occlusal forces so periodontically weakened teeth do not loosen

C. To prevent a natural unopposed tooth from migrating

D. To prevent maxillary central incisors from separating after closure of diastema

- A, B, C
- A, C, D
- B, C, D
- A, D

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abilization is achieved by splinting one or more mobile teeth to one another and to other more
ble teeth in a position that facilitates a more axial and even distribution of occlusal forces. This is
generally performed on teeth with reduced periodontal support. The rationale for this is improved patient
comfort, function and plaque control, better distribution of occlusal forces and improved tooth stability dur-
ing clinical procedures.

There is **no reason** for splinting non-mobile teeth as a preventive measure. Splinting is only one measure
used in the treatment of periodontal disease. Splinting should be used with other necessary measures such
as root planing, oral hygiene instructions, pocket elimination, and occlusal adjustment. **Loose teeth splint-
ed to adjacent teeth** may become stabilized. When many teeth are loose, adjacent sextants should be
included in the splint. Teeth tend to loosen buccolingually yet may remain firm mesiodistally. Even when
teeth do not tighten, the splint serves as an orthopedic brace that permits useful function of loose teeth. A
variety of means may be utilized to achieve temporary stabilization. Whatever means are used, special
attention should be paid to making the splint amenable to oral hygiene procedures and instructing the
patient on plaque control around the splint.

Types of splints:

- **External:** ligatures, tooth-bonding plastic splints, welded band splints, continuous clasps, and night guards. These devices may be unesthetic or unhygienic. They lack durability, rigidity, and / or fit. However, they are prepared easily and economically. **Tooth structure is not removed** *(very conservative).*
- **Intracoronal:** include acrylic or amalgam with an embedded wire and acrylic or acrylic-and-gold provisional splints. **Tooth structure is removed.** These devices tend to be more serviceable than the external splints. However, the materials tend to wear and break and are dependent on the strength of the bonding medium. They tend to create plaque harbors, which leads to caries, calculus deposition, and inflammation.

per Floss® is ideal for cleaning braces, bridges and wide gaps between teeth. Its three unique components – a stiffened end, spongy floss, and regular floss – all work all together for maximum benefits.

Three components:

- Stiff-end threader so you can floss under appliances
- Spongy floss cleans around appliances and between wide spaces
- Regular floss removes interproximal subgingival plaque

Indications for use of Super Floss® include plaque removal around the following:

- Isolated teeth
- Teeth separated by a diastema
- Wide embrasures where interdental papillae have been lost
- Fixed partial dentures *(bridgework)*
- Orthodontic appliances
- Implants

PERIODONTICS

OH

The effectiveness of toothbrushing is **best** measured by:

- The amount and location of plaque
- The caries experience
- The toothbrushing frequency
- The condition of the toothbrush

involves different combinations of horizontal, vertical, rotary, and vibratory motions. All the techniques described in the table below can be used to clean facial, lingual and occlusal surfaces, but are less effective for interproximal areas. Only the **Modified Bass** technique *("sulcular technique")* effectively cleans the **sulcus**, while the Modified Charters technique is useful for cleaning fixed orthodontic appliances. No matter what toothbrushing method is chosen, the manual toothbrush should have soft nylon bristles and a small head.

Toothbrushing Techniques

Technique	Bristle Position	Motion	Effect
Horizontal Scrub	90° to tooth	Horizontal strokes	• Supragingival cleaning • Gingival stimulation
Fones	90° to tooth	Large circles over teeth and gingiva	• Supragingival cleaning • Gingival stimulation
Leonard	90° to tooth	Vertical strokes	• Supragingival cleaning • Gingival stimulation
Smith-Bell	At occlusal surface	Sweep gingivally	• Supragingival cleaning
Rolling Stroke	Apically at attached gingiva	Sweep in arc occlusally	• Supragingival cleaning • Gingival stimulation
Stillman	45° to apex; part on gingival margin, part on cervix of tooth	Vibratory pulsing	• Gingival stimulation
Modified Charters	90° to tooth	Sweep occlusally, circular vibratory	• Gingival stimulation • Interproximal cleaning
Modified Bass	45° to apex, in sulcus	Sweep occlusally, vibratory horizontal jiggle	• Supragingival cleaning • Gingival stimulation

PERIODONTICS

OH

Water irrigation devices *(oral irrigators)* have been shown to:

- Eliminate plaque
- Dislodge food particles from between teeth
- Disinfect pockets for up to 12 hours
- Prevent calculus formation

Water irrigation devices are useful for loosening debris from the gingival sulcus and dislodging debris from between teeth. These devices will disrupt bacterial plaque, but will not remove it.

These devices can remove **non**-adherent bacteria from subgingival as well as supragingival sites. This can be done at home as well as in office *(also called "flushing" or "lavage" of periodontal pockets)*. **Note:** When used with water only, it can reduce gingivitis but to a **lesser** extent than when used with chlorhexidine gluconate 0.12%.

Beneficial effects from irrigation include:

- Reduction of gingivitis
- Reduction or alteration of microbial flora
- Subgingival access *(penetrates below gingival margin)*
- Delivery of antimicrobial agents including chlorhexidine gluconate, stannous fluoride, and sanguinaria

Water irrigation devices may be **contraindicated** in patients requiring antibiotic premedication prior to dental treatment since these devices have the potential for causing a bacteremia. The patient's physician should be consulted.
Note: Fluoride, antibiotics, and chlorhexidine have been demonstrated to be effective in inhibiting microbial plaque.

Chlorhexidine gluconate 0.12%, in an alcohol containing vehicle:

1. Stains teeth, tongue, and resin restorations

2. Impairs taste perception

3. Causes teratogenic alterations

4. Has a high systemic toxicity

5. Has retention properties that are concentration and time dependent and is not influenced by the temperature or pH of the rinsing

A. 1 only

B. 1, 2, and 3

C. 1 and 4

D. 1, 2, and 5

E. All of the above are true

F. None of the above are true

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• **D 1,2, and 5**

***The stains *(yellowish-brown to brown color)* are reversible and impairment of taste perceptions is transient. It has a low systemic toxicity and is not known to be associated with teratogenic alterations.

To date, two basic agents have been shown to have a significant therapeutic effect on gingivitis over a 6-month period. **Chlorhexidine gluconate, 0.12%,** in an alcohol-containing vehicle and phenolic compound / essential oil-based mouthrinses can be used to help control gingivitis. Chlorhexidine kills bacteria in situations when used for 30 seconds twice daily. It is the most effective anti-microbial agent for reducing plaque and gingivitis over long term. **Its effectiveness** may be explained by the fact that it leaves the greatest residual concentration in the mouth after its use. It is rapidly absorbed onto the teeth and the pellicle; and it is slowly released. It is approved by the ADA as an antimicrobial and antigingivitis agent. Examples include: **Peridex** and **PerioGard.**

The essential oils, thymol, menthol, and eucalyptol, along with methyl salicylate for flavoring, constitute the active ingredients in most phenol-based mouthrinses. These rinses also contain between 20% to 27% alcohol in the vehicle. Examples include: **Listerine.**

Other topical antimicrobial agents include:

- **Stannous fluoride** – antimicrobial action appears to be related to the stannous *(tin)* ion rather than to the fluoride ion. It is available in gel form *(e.g., Stop, Gel-Kam)*. The ADA accepts stannous fluoride in anticaries activity **but not for anti-plaque** or antigingivitis purposes.
- **Quaternary ammonium compounds – not as effective** as others in reducing plaque or gingivitis, seem to be best at eliminating bad breath *(halitosis)* contains cetylpyridinium. Examples include: **Scope and Cepacol.**

PERIODONTICS

OH

Which of the following oral hygiene aids is appropriate for cleaning a **Class II** furcation?

- Stim-U-Dent®
- Interproximal brushes
- Interdental stimulator
- Perio Aid®
- Oral irrigator

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• **Perio Aid®**

A tapered, round toothpick is inserted into the hole in the carrier and is then broken off. The tip is left in and is used in a tracing motion along the gingival margins. It is also helpful in cleaning furcations that are accessible.

Stim-U-Dent® *(balsa wood wedges)* → these are of primary importance in **gingival massage.** They are also good for patients with interdental recession. These picks *(triangular in cross section)* are small enough to fit into most interdental spaces. As a supplement to brushing, they are useful for dislodging interproximal debris often missed by meticulous brushing and for massaging the underlying interproximal gingiva.
Interproximal brushes *(proxabrush)* → are used for interdental cleansing when the interdental space is wide. The brushes are replaceable.
Interdental stimulator → consists of a rubber tip of smooth or ribbed conical shape attached to a handle or to the end of a toothbrush. Its action **massages and stimulates circulation** of the interdental gingiva and may increase the tone of the tissue. It is not recommended for areas in which the papillae are normal and fill the interproximal spaces. It may cause injury to the gingival tissue.
Water irrigation devices → are useful around fixed bridgework and orthodontic appliances where debris tends to accumulate. The water pressure helps remove food debris, dilutes bacterial by-products and even some plaque, but it **does not** remove all dental plaque. These devices may be injurious if used forcefully in deep periodontal pockets.

Remember:

1. **Nothing replaces** brushing and flossing for **removal of or** disruption of plaque.
2. Frequent **brushing** and flossing helps to prevent **calculus** formation by breaking up the **matrix of plaque.**
3. New **plaque growth occurs shortly after brushing** and flossing *(starts interproximally and works its way around the tooth).*

Which of the following controlled-release delivery systems for site-specific antimicrobial therapy is impregnated with 12.7 mg of tetracycline HCL?

- Atridox®
- Actisite®
- Perio Chip®
- All of the above

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• **Actisite®**

Perio Chip®, Atridox®, or Actisite® are disinfectant or antibiotics that are used as an adjunct to scaling & root planing *(non-surgical periodontal therapy)*. These products are placed subgingivally in deep periodontal pockets to control periodontal inflammation. These products can enhance the positive results obtained from non-surgical therapy and may delay or eliminate the need for periodontal surgery.

Actisite® is an ethylene vinyl acetate flexible fiber impregnated with 12.7 mg of **tetracycline HCL**. It is placed subgingivally into the periodontal pocket, where the tetracycline is released slowly over 7 to 10 days. It is nonresorbable and must be removed at a follow-up appointment.

Atridox® is a biodegradable controlled release *(over a period of 7 days)* gel containing **doxycycline.** It is delivered via a syringe system to the diseased pocket for use in the treatment of chronic periodontitis to promote attachment level gain, to reduce pocket depths, and to reduce bleeding on probing.

Perio Chip® is a gelatin chip that contains 2.5 mg of **chlorhexidine gluconate** and is placed into a periodontal pocket following scaling and root planing. It is bio-adsorbable over 8 days and provides a continuous, sustained release of antibiotic into the site.

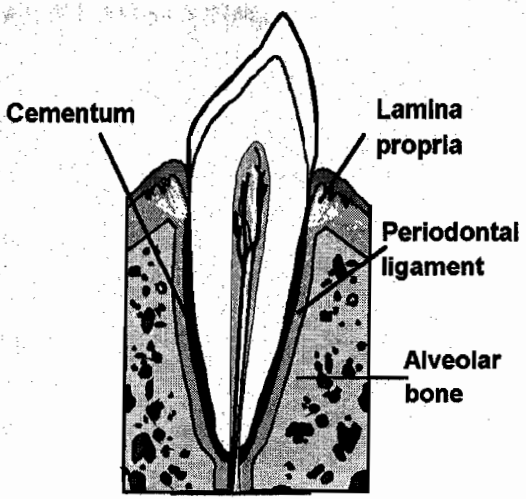
Periostat® is a twice daily, orally administered tablet containing 20 mg of **doxycycline** that has been approved as an adjunctive treatment to scaling and root planing *(SRP)*. This drug promotes attachment level gain and reduces pocket depth in patients with adult periodontitis.

PERIODONTICS

Pdl / G

The periodontium is a collective term for the supporting structures of the teeth. All of the following are considered to be a part of the periodontium **except:**

- Gingiva
- Periodontal ligament
- Cementum
- Alveolar bone
- Pulp



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• **Pulp**

The periodontium refers to the functional unit of tissues that surrounds and supports the teeth. Their main functions are to support, protect, and provide nourishment to the teeth.

The periodontium consists of **two** sections:

1. **Gingival unit:** composed of the free and attached gingiva and the alveolar mucosa.
2. **Attachment apparatus** which attaches the root to the alveolar bone:
    - **Periodontal ligament**
    - **Cementum**
    - **Alveolar bone** *(consists of alveolar bone proper and supporting bone)*

In health, **gingival crevicular fluid** *(GCF)* is a transudate that emerges from the gingival sulcus. The gingival crevicular fluid may contain a variety of enzymes and cells, particularly desquamating epithelium and neutrophils, that are being shed through the sulcus. An increase in gingival crevicular flow is the **first detectable sign** of inflammation. Once inflammation has occurred, the GCF is referred to as an **inflammatory exudate.** This exudate contains a higher level of serum proteins and leukocytes.

Notes: Nutrient materials for the cells of **the gingival** epithelium are supplied primarily by way of **capillaries** in subjacent connective **tissue.**

Of the choices listed below, which one describes the boundaries that define the **attached gingiva?**

- From the **gingival margin** to the **interdental groove**
- From the **free gingival groove** to the **gingival margin**
- From the **mucogingival junction** to the **free gingival groove**
- From the **epithelial attachment** to the **cementoenamel junction**

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• **From the mucogingival junction to the free gingival groove** *(base of the sulcus)*

***Remember:** The **free gingiva** extends from the **free gingival groove** *(base of the sulcus)* to the **gingival margin.** The **free gingival cuff** is lined by sulcular epithelium which is continuous with the oral gingival epithelium at the gingival margin. A **free gingival groove** demarcates the junction between the free gingiva and the attached gingiva.

The **attached gingiva** is normally **coral pink** in color and is firmly bound down to underlying tooth structure, periosteum, and bone. The color may vary depending on the following factors: degree of keratinization, thickness of epithelium, presence of melanin and the number of blood vessels.

The **gingivae** are lined by a masticatory type mucosa. The epithelium is **keratinized** and is interlocked with the underlying lamina propria by tall rete ridges and connective tissue papillae, respectively. Collagen fibers of the dentogingival group of fibers extend from the cementum into the lamina propria of the gingiva.

Important: The **width of facial attached gingiva** ranges from 1-9 mm and is greatest on the facial surface of the maxillary lateral incisor and narrowest on the facial surfaces of the mandibular canine and first premolar.

"Stippling" of the attached gingiva refers to the irregular surface texture of the attached gingiva, similar to the surface of an orange peel. Stippling occurs at the intersection of epithelial ridges that causes the depression and the interspersing of connective tissue papillae between these intersections giving rise to the small bumps.

All of the following may result in an **absence of "stippling"** in areas of the attached gingiva:

• **Edema of the underlying** connective tissue • **Inflammatory degradation** of gingival collagen fibers
• **Normal variation in gingival** topography

Gingival fibers are found within the:

- Attached gingiva
- Free gingiva
- Mucogingival junction
- Attached and free gingiva

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• **Free gingiva**

The **collagen fibers** *(composed of Type I collagen)* that extend from the cervical cementum of the tooth into the gingiva are called **gingival fibers.** Also known as the supracrestal connective tissue fibers, these fibers are designated by their orientation: Dentogingival, dentoperiosteal, circular, and alveologingival fibers. **Note:** Some literature includes the transseptal fibers as a separate group of gingival fibers *(instead of being a group of principal fibers of the PDL).*

The **function** of the gingival fibers is to support the gingiva, keeping it closely adapted to the tooth surface and sustaining it against forces placed on it during mastication.

Notes:

1. The **gingival apparatus** is a term used to describe these gingival fibers and the epithelial attachment.
2. The **gingival ligament** includes the dentogingival, alveologingival and circular fibers.
3. The **indifferent fiber plexus** is found in the PDL. These are small collagen fibers associated with the larger principal collagen fibers and run in all directions.

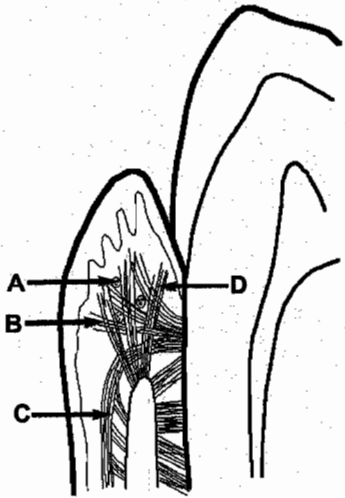
The drawing below is a frontal view of the **gingival fiber** groups. Which group arise from the alveolar crest and fan out coronally into the adjacent gingival connective tissue?

A. Circular fibers

B. Dentogingival fibers

C. Dentoperiosteal fibers

D. Alveologingival fibers



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- **(D) Alveologingival fibers**

Gingival fibers are collagen fibers that provide support for the marginal gingiva including the interdental papilla. **Note:** Even though they are found in the free gingiva, they are continuous with the connective tissue fibers of the PDL and are often considered to be part of the periodontal ligament.

The **gingival fibers of the free gingiva** are subdivided into four groups:

- **Circular fibers** → encircle the tooth around the most cervical part of the root and insert into the cementum and lamina propria of the free gingiva and the alveolar crest. **They resist rotational forces.**
- **Dentogingival fibers** → extend from the cementum apical to the epithelial attachment and course laterally and coronally into the lamina propria of the gingiva.
- **Dentoperiosteal fibers** → extend from the cervical cementum over the alveolar crest to the periosteum of the cortical plates of bone.
- **Alveologingival fibers** → insert in crest of alveolar process and spread out through the lamina propria into the free gingiva.

Remember: Transseptal fibers are sometimes classified as a separate group of gingival fibers.

Which group of **principal fibers** of the periodontal ligament help to prevent tipping and dislocation of the tooth?

A. Transseptal

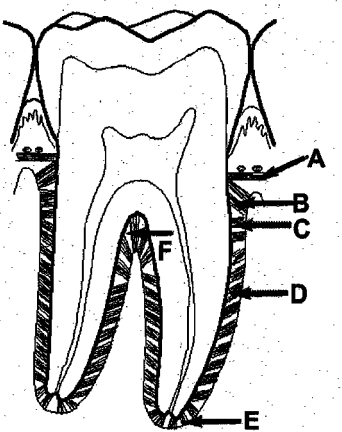
B. Alveolar crest

C. Horizontal

D. Oblique

E. Apical

F. Interradicular



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• **(E) Apical**

The principal fibers of the PDL are primarily composed of bundles of **type I collagen fibers** grouped according to the direction they extend from the cementum of the root to the alveolar bone.

Transseptal fibers → extend from tooth to tooth, coronal to the alveolar crest and are embedded in the cementum of adjacent teeth. Not found on the facial aspect and have no attachment to alveolar crestal bone. These fibers keep all the teeth aligned *(they maintain the integrity of the dental arches).*

Alveolar crest fibers → extend from the cervical cementum of the tooth to the alveolar crest. Their function is to counterbalance the occlusal forces on the more apical fibers and resist lateral movements.

Horizontal → run perpendicular from the alveolar bone to the cementum and **resist lateral forces.**

Oblique fibers → slant occlusally from the cementum to the alveolar bone. These fibers are more resistant to forces along the long axis of a tooth *(masticatory forces).* Most likely to be found in the apical two-thirds of the root. They are the most numerous **(approximately one-third of all principal fibers are in this group).**

Apical fibers → radiate apically from cementum of tooth to bone. They offer the **initial resistance** to tooth movement in an **occlusal direction.**

Interradicular fibers *(found only on multi-rooted teeth)* → **extend** from the cementum in the furcation area to the bone **within** the furcation.

Important: The **terminal portions** of these **collagen fibers** that insert into **the cementum** and alveolar bone are called **Sharpey's fibers.**

Which structure is the **inner layer of cells** of the junctional epithelium and attaches the gingiva to the tooth?

- Mucogingival junction
- Free gingival groove
- Epithelial attachment
- Gingival col

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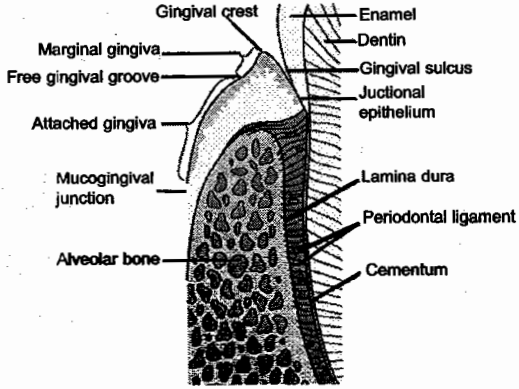
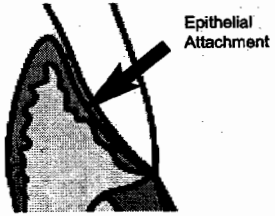
• **Epithelial attachment**

The **epithelial attachment** is the structural complex that mediates the attachment of the reduced enamel epithelium or junctional epithelium to the tooth, namely the **internal basal lamina and hemidesmosomes.** The **primary** epithelial attachment refers to the attachment of reduced enamel epithelium to the tooth. The **secondary** epithelial attachment refers to the attachment of the junctional epithelium to the tooth.

Remember: The **junctional epithelium** is that portion of the gingival epithelium that is attached to the tooth on one side and to the gingival connective tissue on the other. The **sulcular epithelium** *(crevicular epithelium)* is the stratified squamous epithelium lining the inner aspect of the soft tissue wall of the gingival sulcus extending from the gingival margin to the junctional epithelium.





Cervical line contours are closely related to the attachment of the gingiva at the neck of the tooth. The **greatest contour of the cervical lines and gingival attachments** occur on:

- The distal surface of anterior teeth
- The distal surface of posterior teeth
- The mesial surface of anterior teeth
- The mesial surface of posterior teeth

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• **The mesial surface of anterior teeth**

***The mesial surface of the **maxillary central** has the greatest curvature.

All teeth generally have a **greater proximal cervical line** *(CEJ)* **curvature on the mesial** than the distal. Also, the proximal cervical line *(CEJ)* curvatures **are greater on the incisors** and tend to get smaller when moving toward the last molar, where there may be no curvature at all.

The **cemento-enamel junction** of all teeth curves in two directions:

• **Towards the apex** on the facial and lingual surfaces
• **Away from the apex** on the mesial and distal surfaces

In the **absence of periodontal disease,** the configuration of the crest of the interdental alveolar septa is determined by the position of the CEJ on adjacent teeth.

The **width** of the interdental alveolar bone is determined by the tooth form present. Relatively flat proximal tooth surfaces **call for narrow** septa, whereas in the presence of an extremely convex tooth surface, wide **interdental** septa with flat crests are found.

The **narrowest band of attached gingiva** is found:

- On the lingual surfaces of **maxillary incisors** and the facial surfaces of **maxillary first molars**
- On the facial surfaces of **mandibular second premolars** and the lingual surface of canines
- On the facial surfaces of the **mandibular canine** and **first premolar** and the lingual surfaces adjacent to the **mandibular incisors** and **canines**
- None of the above

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- **On the facial surfaces of the mandibular canine and first premolar and the lingual surfaces adjacent to the mandibular incisors and canines**

Narrow gingival zones may occur also at the **mesiobuccal root of maxillary first molars,** associated with prominent roots and sometimes with bony dehiscences and at the mandibular third molars. The **greatest width** of attached gingiva is generally found in the incisor regions *(especially the lateral incisor)*. Compared to the mandible, the maxilla usually exhibits the broader area of attached gingiva.

A **"functionally adequate"** zone of gingiva is defined as one that is keratinized, firmly bound to tooth and underlying bone, about 2 mm or more in width, and resistant to probing and gaping when the lip is distended.

Histologic Comparison of Attached Gingiva & Alveolar Mucosa

Attached gingiva	Alveolar mucosa
Keratinized	Nonkeratinized
Stippled	Unstippled
Deep rete pegs	Short, wide rete pegs
Thick lamina propria	Thin lamina propria
Few elastic fibers	Numerous elastic fibers
Indistinct submucosa	Distinct submucosa
Firmly attached	Movable

Notes:

1. The **"attached" gingiva** is structured to **withstand frictional** stresses of mastication and brushing.
2. The **alveolar mucosa** appears to be **well-adapted** to permit **movement but is not able to withstand frictional stresses.**

Which of the following types of oral mucosa is **not** keratinized under normal conditions?

- Buccal mucosa
- Vermillion border of the lips
- Hard palate
- Gingiva

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• **Buccal mucosa**

Normal **keratinized** areas of the oral mucosa include the vermillion border of the lips, the hard palate, dorsum of the tongue, and the gingival tissues. The buccal mucosa, floor of the mouth, lateral and ventral surface of the tongue, gingival col, sulcular epithelium, alveolar mucosa, and soft palate are **non-keratinized** oral mucosa under normal conditions.

***Important:** All oral mucosa, whether keratinized or nonkeratinized, is of the **stratified squamous** type of epithelium.

Functional types of oral mucosa:

1. **Masticatory** → composed of free and attached gingiva and the mucosa of the hard palate. The epithelium of these tissues is **keratinized** and the lamina propria is a dense, thick, firm connective tissue containing collagenous fibers. **Remember:** The surface epithelium of the gingiva is **highly impermeable,** which makes it resistant to bacterial invasion.
2. **Lining or reflective mucosa** → the mucosa that lines most of the oral cavity, with the exception of the gingiva, anterior palate and dorsum of the tongue. The lining mucosa is a thin, movable tissue with a thin, **non-keratinized epithelium** and a thin lamina propria. ***The junction of the **lining** mucosa with the masticatory mucosa is referred to as the **mucogingival junction.**
3. **Specialized mucosa** → covers the **dorsum** of the tongue and the taste buds. The epithelium is **keratinized.**

The **alveolar process** is that part of the maxilla and mandible that houses the teeth. It consists of two main parts which are called:

- Alveolar bone proper and supporting alveolar bone
- Cortical plate and spongy bone
- Compact lamellar bone and a layer of bundle bone

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- **Alveolar bone proper and supporting alveolar bone**

The alveolar process **consists of** two main parts:

1. **Alveolar bone proper** → that part of the alveolar process which immediately surround the root of the tooth and to which the fibers of the PDL are attached. It is a perforate cribriform plate through which vessels and nerves pass between the PDL and marrow. **It consists of two layers of bones:**
   - **Compact lamellar bone**
   - **Layer of bundle bone** → this is the layer that the periodontal fibers insert into.
2. **Supporting alveolar bone** → that bone which surrounds the alveolar bone proper and gives support to the socket. It consists of:
   - **Cortical plate** *(compact lamellar bone)* → forms the outer and inner plates of the alveolar processes. It is thicker in the mandible than in the maxilla.
   - **Spongy bone** *(cancellated bone)* → fills in area between cortical plates of bone. This type of bone is **not present** in the anterior region of the mouth – here the cortical plate is fused to the cribriform plate. This is also true over the radicular buccal bone of the maxillary posterior teeth.

Note: The **alveolar bone proper** is the **only essential** part of the bone socket. The supporting alveolar bone is not always present.

The periodontal ligament is made of large collagen fibers that course between the cementum and the alveolar bone. These fibers are embedded in the outer layer of cementum and are called:

- Purkinje's fibers
- Sharpey's fibers
- Spindle fibers
- Gray fibers

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- **Sharpey's fibers**

***They project into the cementum between groups of cementoblasts, and lie perpendicular to the surface of the cementum. The diameter of these fibers is **considerably greater on the bone side** than on the cementum side.

The **periodontal ligament** surrounds the root of the tooth, separating it from and attaching it to the alveolar bone. It is a soft, fibrous connective tissue that is cellular and vascular. Its functions include:

- **Physical** → attachment of the tooth to the bone via principal fibers and the absorption of occlusal forces.
- **Formative** → formation of connective tissue components by activities of connective tissue cells *(cementoblasts, fibroblasts and osteoblasts).*
- **Remodeling** → by activities of connective tissue cells that are able to form as well as resorb cementum *(cementoblasts or cementoclasts),* the periodontal ligament *(fibroblasts or fibroclasts)*, and the alveolar bone *(osteoblasts or osteoclasts).*
- **Nutritive** → through blood vessels that maintains the vitality of its various cells.
- **Sensory** → carried by the trigeminal nerve, proprioceptive and tactile sensitivity is imparted through PDL (*sensation of contact between teeth).*

The **PDL** tends to become **thinner** with **age,** probably as a result of the deposition of cementum and bone *(this happens primarily in the region of the mandibular canine).* The tissues immediately adjacent to the PDL are cementum and alveolar bone.

Note: Remnants of the epithelial root sheath that remain following its disintegration during root formation are called **epithelial rests of Malassez** *(groups of epithelial cells).* These are located in the periodontal ligament. Some rests degenerate, others become calcified *(forming cementicles).*

The **periodontal ligament** consists predominantly of:

- Bundles of collagenous and elastic fibers
- Fibrous elements that are formed by the fibroblasts
- Regularly arranged bundles of collagenous fibers
- Large bundles of oxytalan fibers

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- **Regularly arranged bundles of collagenous fibers → PDL fibers are principally made of collagen type I**

The periodontal ligament **does not** contain mature elastin. Two immature forms are found, oxytalan and eluanin. The oxytalan fibers run parallel to the root surface in a vertical direction and bend to attach to cementum in the cervical third of the root. They are thought to regulate vascular flow. **Note:** The main type of cell in the periodontal ligament is the **fibroblast.**

The **periodontal ligament** is a specialized form of connective tissue derived from the dental sac and lies between the cementum and the alveolar bone. The space it occupies is the periodontal space. This extends coronally to the most apical part of the lamina propria of the gingiva.

The tooth is anchored to the surrounding alveolar bone by the periodontal ligament. The alveolar bone directly surrounding the tooth cavity is called **cribriform plate.** The layer of cribriform plate into which the collagen fibers of the ligament are anchored is called **bundle bone.** The bone underlying the gingiva is called **cortical plate**. Cribriform and cortical plates are both **compact bone**, and are separated by **spongy bone** in the center of alveolar bone. **Interstitial spaces** are regions of loose connective tissue located between periodontal fiber bundles. These regions contain fibroblasts, blood vessels, and nerves and are responsible for providing nutrients to the periodontal ligament and cells of the cementum.

In the **absence of function** of the tooth, the periodontal ligament becomes narrow and looses the organization of the fiber bundles.

The periodontal ligament in an adult is about:

- 0.002 mm wide
- 0.2 mm wide
- 2.0 mm wide
- 20 mm wide

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- **0.2 mm wide**

The **thickness of the PDL** varies depending on:

- The person's age→ it decreases to 0.1 mm in old age, this happens most likely as a result of the deposition of cementum and bone *(this is most noticeable in the region of the mandibular canine).*
- Stage of eruption
- Function of the tooth → it is thicker in functioning than in non-functioning teeth and in areas of tension as opposed to areas of compression.
- History of any trauma → PDL may be non-existent due to ankylosis *(fusion)* of tooth to the bone.

Four features of the human dentition which **directly affect** the health of the PDL and its hard tissue anchorage in terms of **resisting occlusal force:**

1. **Anterior teeth** have slight or no contact in the intercuspal position.
2. The **occlusal table** is less than sixty percent of the overall faciolingual width of the tooth.
3. The **occlusal table** of the tooth is generally at right angles to the long axis of the tooth.
4. Crowns of **mandibular molars** are inclined about 15-20° toward the lingual. For this reason, the root apices of mandibular molars are positioned more facially and the crowns are positioned more lingually.

Note: Orthodontic treatment is possible **because the PDL** continuously responds and changes as the result of the functional requirements imposed upon it by externally applied forces.

Surrounding the root of each tooth is a **specialized epithelium** known as a:

- Connective tissue attachment
- Periodontal ligament attachment
- Junctional epithelium
- Nasmyth's membrane

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• **Junctional epithelium**

The **dentojunctional epithelium** is the gingival epithelium that faces the tooth. It is composed of **non-keratinized stratified squamous epithelium** and is divided into:

1. **Sulcular epithelium** → lines the sulcus; it connects directly with the junctional epithelium.
2. **Junctional epithelium** → begins at the base of the sulcus; it is a collar-like band of stratified squamous epithelium that is firmly attached to the tooth by hemidesmosomes. At its beginning it is approximately 15-30 cell layers thick, and at its **apical end** it is only a few cell layers thick. The junctional epithelium consists of two layers: **a basal and suprabasal layer.**

***In ideal gingival health,** the junctional epithelium is located entirely on enamel above the cementoenamel junction.

The **epithelial attachment** is the structural complex that mediates the attachment of the reduced enamel epithelium or junctional epithelium to the tooth, namely the **internal basal lamina and hemidesmosomes.** The **primary** epithelial attachment refers to the attachment of reduced enamel epithelium to the tooth. The **secondary** epithelial attachment refers to the attachment of the junctional epithelium to the tooth.

Note: Histologically, the best way to distinguish the free gingiva from the epithelial attachment is the fact that the epithelium of the epithelial attachment **does not contain rete pegs** and the free gingiva does. **Rete pegs** are epithelial projections that extend into the gingival connective tissue. Connective tissue projections that extend into the overlying epithelium are called **connective tissue papillae.**

Which of the following cells are **most abundant** in the periodontal ligament?

- Fibroblasts
- Osteoblasts
- Cementoblasts
- Osteoclasts

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• **Fibroblasts**

***Fibroblasts are the **most common cells** in the periodontal ligament. They appear as ovoid or elongated cells orientated along the principal fibers and exhibit pseudopodia-like processes.

The periodontal ligament contains a unique assortment of cells that are capable of generating and maintaining three distinct tissues, namely the ligament itself as well as the mineralized tissues on either side of it, i.e. the cementum and the alveolar bone.

The **major cell types** of the periodontal ligament include the following:

- Fibroblasts, macrophages and undifferentiated **ectomesenchymal** cells that are able to give rise to fibroblasts, cementoblasts and osteoblasts. These pluripotent cells are unique to the periodontal ligament. Therefore, the periodontal ligament serves as the primary source of new cells when attempts are made at regenerating lost periodontal attachment.
- Cementoblasts and cementoclasts
- Osteoblasts and osteoclasts
- Cell rests of Malassez
- Vascular and neural elements

Two types of **nerve endings** are found in **the PDL**:
1. Free, **non-myelinated** nerve endings → **convey pain**
2. Encapsulated ***(myelinated)*** nerve endings **with** specialized **receptors** → convey **pressure**

Parts of the **free gingiva** include all of the following **except:**

- Gingival margin
- Free gingival groove
- Mucogingival junction
- Gingival sulcus
- Interdental *(interproximal)* gingiva

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- **Mucogingival junction**

Components of the gingiva:

1. **Free gingiva** *(unattached or marginal gingiva)* → the collar of tissue that is not attached to the tooth or alveolar bone. It is approximately 1 to 3 mm wide and forms the soft tissue wall of the gingival sulcus next to the tooth. Other structures of the free gingiva include:
   - **Gingival margin** → the 1 mm narrow band of gingiva that forms the immediate collar around the base of the tooth. This area is the first to show symptoms of gingivitis.
   - **Free gingival groove** → separates the free gingiva from the attached gingiva. It is only present in about one-third of adults. When present, it is located 1 to 1.5 mm apical to the gingival margin.
   - **Gingival sulcus** → a shallow groove between the marginal gingiva and the tooth surface, bound by sulcular epithelium laterally and junctional epithelium apically.
   - **Epithelial attachment** *(part of the junctional epithelium)* → joins the gingiva to the tooth surface.
   - **Interdental** *(interproximal)* **gingiva** → the gingiva that occupies the interdental spaces coronal to the alveolar crest.
2. **Attached gingiva** → that part of the gingiva that is attached to the underlying periosteum of the alveolar bone, to the cementum by connective tissue fibers and the epithelial attachment. It is present between the free gingiva and the more movable alveolar mucosa.

Notes:

1. The **mucogingival junction** separates the attached gingiva from the alveolar mucosa.
2. The **free gingival groove** separates the free gingiva from the attached gingiva.

Supragingival plaque:

- Is unattached or loosely adherent
- Is dominated by gram-negative organisms
- Acquires nutrition from saliva and host diet in the oral cavity
- Is dominated by anaerobes

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- **Acquires nutrition from saliva and host diet in the oral cavity**

Supragingival plaque → saliva and **diet** alter bacterial composition:

- Is attached or tooth associated
- Consists primarily of **gram-positive facultative cocci**. Filaments and slender rods are also seen.
- Streptococcus sanguis, mutans & Actinomyces viscosus **predominate**

Subgingival plaque → saliva and diet **do not alter** bacterial composition:

- Is unattached or loosely adherent *(more closely associated with the wall of the subgingival tissues than is the attached plaque)*
- Is dominated by **gram-negative anaerobic rods and spirochetes**
- Bacterial species found include: Porphyromonas gingivalis, Fusobacterium nucleatum, Prevotella intermedia, Bacteroides forsythus, Campylobacter rectus and Treponema denticola as well as other Treponema species. Capnocytophaga species, Eikenella corrodens, and Actinobacillus actinomycetemcomitans can also be found.

Note: Bacteria produce low molecular weight compounds such as hydrogen sulfide and butyrate that are capable of inducing some of the early signs of inflammation but the chronic **signs of periodontitis,** including inflammation, loss of collagen, and loss of bone, are primarily the result of bacterial products entering the tissue and activating inflammatory and immune processes.

Important: The overall pattern observed in **dental plaque** development is a very characteristic shift from the **early predominance of Gram-positive facultative** microorganisms to the **later predominance of Gram-negative anaerobic microorganisms,** as the plaque mass accumulates and matures. This developmental **progression** is also **reflected in the** shifts in **predominant** microorganisms that are observed in the **transition from** health to **disease.**

PERIODONTICS

P / C

The key etiologic agent in the **initiation** of gingivitis and periodontal disease is:

- Calculus
- Plaque
- Toothbrush abrasion
- Saliva

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• **Plaque**

Dental plaque is a soft deposit that accumulates on the teeth. Plaque can be defined as a complex microbial community with greater than 10^{10} bacteria per milligram. In addition to the bacterial cells, plaque contains a small number of epithelial cells, leukocytes, and macrophages. The cells are contained within an extracellular matrix ***(dextran matrix)***, which is formed from bacterial products and saliva. The extracellular matrix contains protein, polysaccharide and lipids. **Dextrans are insoluble and sticky,** which contributes to the ability of plaque to adhere to teeth. Inorganic components are also found in dental plaque; largely calcium and phosphorus which are primarily derived from saliva. The inorganic content of plaque is greatly increased with the development of calculus. The process of calculus formation involves the calcification of dental plaque.

Selected Bacterial Species Found in Dental Plaque		
	Facultative	Anaerobic
Gram-positive	Streptococcus mutans Streptococcus sanguis Actinomyces viscosus	
Gram-negative	Actinobacillus actinomycetemcomitans Capnocytophypa species Eikenella corrodens	Porphyromonas gingivalis Fusobacterium nucleatum Prevotella intermedia Bacteroides forsythus Campylobacter rectus
Spirochetes		Treponema denticola *(Other Treponema species)*

Notes:

1. The **goal of the clinician** and of a regimen **in oral** hygiene is to **remove this etiologic agent.**
2. Following a **prophy, plaque** is most likely to **accumulate** on the **interproximal** tooth surfaces first.

All of the following statements concerning bacterial plaque are true **except:**

- It is the **key etiologic agent** in the initiation of gingivitis and periodontal disease
- It is an accumulation of a **mixed bacterial community** in a dextran matrix
- It forms on a cleaned tooth **within minutes**
- It is composed of bacteria and an intermicrobial matrix. Organic and inorganic solids constitute approximately 80% *(bacteria make up at least 70 to 80% of the solid matter)*, and water accounts for 20%
- There are two categories: **supragingival** and **subgingival plaque**
- Different bacteria may be found in plaque *(cocci, rods, and filaments)* and their proportions change with **time, diet and location**

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- **It is composed of bacteria and an intermicrobial matrix. Organic and inorganic solids constitute approximately 80%** ***(bacteria make up at least 70 to 80% of the solid matter)*****, and water accounts for 20%**

***This is **false;** Organic and inorganic solids constitute approximately **20%** *(bacteria make up at least 70 to 80% of the solid matter)*, and water accounts for **80%.**

Stages in plaque formation:

- **Formation of the pellicle** *(acquired pellicle)* → the pellicle is derived from **salivary** constituents which are selectively adsorbed onto the tooth surface. Components of the dental pellicle include albumin, lysozyme, amylase, immunoglobulin A, proline-rich proteins and mucins. It is essentially structureless and bacteria-free. It will form on a clean tooth surface within a matter of minutes because of its salivary origin. It also will **form** on crowns, dentures, porcelain teeth, etc.
- **Bacterial colonization** → within a short time of cleaning a tooth, the pellicle-coated tooth surface is colonized by Gram-positive bacteria such as Streptococcus sanguis, Streptococcus mutans, and Actinomyces viscosus. These organisms are examples of the **"primary colonizers"** of dental plaque. The **"secondary colonizers"** include Gram-negative species such as Fusobacterium nucleatum, Prevotella intermedia, and Capnocytophaga species. These organisms would typically be found in plaque after 1 to 3 days of accumulation. After one week of plaque accumulation, other Gram-negative species may also be present in plaque. These species represent what is considered to be the **"tertiary colonizers",** and include Porphyromonas gingivalis, Campylobacter rectus, Eikenella corrodens, Actinobacillus actinomycetemcomitans, and the oral spirochetes *(Treponema species).*
- **Maturation stage** → saliva continues to provide agglutinating substances and other proteins to the intercellular matrix, and bacterial intercellular adhesion results. The crystalline structure will increase and eventually will calcify *(calculus).*

PERIODONTICS

P / C

Early or young plaque consists primarily of:

- Filaments
- Cocci
- Rods
- Vibrios

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• **Cocci**

Plaque formation begins immediately after a tooth surface is cleaned. The rate of plaque formation is affected by diet, age, salivary flow, oral hygiene, tooth alignment, systemic disease and host factors. Changes in the types of organisms occur within plaque as the plaque matures.

Days 1 to 2 → young plaque consists primarily of cocci *(i.e., Streptococcus mutans and sanguis)*

Days 2 to 4 → cocci still dominate but there are increasing numbers of filamentous forms and slender rods. Gradually the filamentous forms replace many of the cocci.

Days 4 to 7 → filaments increase in numbers, and a more mixed flora begins to appear with rods, filamentous forms, and fusobacteria.

Days 7 to 14 → vibrios and spirochetes appear, and the number of white blood cells increases. More gram-negative and anaerobic organisms appear. The signs of inflammation are beginning to be observable in the gingiva.

Days 14 to 21 → vibrios and spirochetes are prevalent in older plaque, along with filamentous forms. Gingivitis is evident clinically.

As plaque ages:
- The number of cocci **decreases** and the **number of rods**, fusiform, filaments, and spirochetes **increases.**
- The number of aerobic bacteria **decreases and the number** of anaerobic bacteria **increases**
- The number of **gram-positive** organisms **decreases** and the number **of gram-negative** organisms **increases.**

PERIODONTICS

P / C

Which of the following crystalline salts is most prevalent in calculus?

- Whitlockite
- Brushite
- Octocalcium phosphate
- Hydroxyapatite

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• **Hydroxyapatite**

Inorganic components of calculus *(70-90%)*: Calcium and phosphates with small amounts of magnesium and carbonate *(these are derived almost entirely from saliva)*. At least two-thirds of the inorganic matter in calculus is crystalline, principally appatite, predominating is **hydroxyapatite**. Calculus also contains octocalcium phosphate, tricalcium phosphate *(whitlockite)* and brushite.

Organic components of calculus: Microorganisms *(same as plaque)*, desquamated epithelial cells, leukocytes and mucin.

Three phases of the formation of calculus:
1. **Pellicle formation** → it begins to form within minutes
2. **Plaque maturation** → microorganisms grow together to form a cohesive plaque layer
3. **Mineralization of the plaque** → both supragingivally and subgingivally

*** The average time for this **whole process** to take place is 12 days.

Note: Calculus is **mineralized plaque.** It is formed by bathing the plaque in a highly concentrated solution of calcium and phosphorus *(i.e., saliva)*. **Subgingival calculus** is darker *(due to pigments from blood breakdown)*, harder and more dense than supragingival calculus.

Remember: The primary reason for removing calculus from teeth is because it harbors plaque organisms.

Periodontal osseous defects *(infrabony or intrabony pockets)* are generally **classified by:**

- The number of bony walls that **were destroyed** by periodontal disease
- The number of bony walls **left surrounding** the tooth
- The number of bony walls that **will remain** after surgery
- **Periodontal probe** readings

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• **The number of bony walls left surrounding the tooth**

Infrabony or intrabony pockets are associated with **vertical bone loss** and may be classified as one-walled *(a **hemiseptum** if only the proximal wall is present or a **ramp** if only a facial or lingual wall is present)*, two-walled *(an interdental crater)*, three-walled *(intrabony defect)*, or four-walled *(circumferential or moat defects)*. Three-walled intrabony defects and four-walled moat defects **offer the best opportunities** for bone graft containment and periodontal regeneration procedures.

Remember: Infrabony defects are **contraindications** for mucogingival surgery.

Notes:

1. **Osseous craters** are concavities in the crest of the interdental bone confined within the facial and lingual walls. Craters have been found to make up about one third *(35.2%)* of all defects and about two thirds *(62%)* of all mandibular defects. They are more common in posterior segments than in anterior segments. They are best treated with osseous surgery *(recontouring)*.
2. Zero-wall defects are alveolar **dehiscences** and **fenestrations.** Osseous surgery is not the treatment of choice.
3. **Suprabony pockets** are associated with **horizontal bone loss.** They **are not** intraosseous.
4. The pattern of bone loss from periodontitis may be either **horizontal** *(i.e., the pattern of interproximal bone loss tends to parallel the CEJ's of adjacent teeth)* or **vertical** *(i.e., the pattern of interproximal bone loss does not parallel the CEJ's of adjacent teeth)*.
5. Horizontal patterns of bone loss are usually **generalized** *(i.e., they involve multiple teeth in a segment)*, and vertical bone loss patterns **may be** found around **isolated teeth.**

When evaluating an osseous defect, the **only way** to determine the **number of walls left surrounding the tooth** is by:

- Periodontal probing
- Radiographs
- Exploratory surgery
- Testing for mobility

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- **Exploratory surgery**

***This is because a dense buccal and/or lingual plate of bone will tend to mask the defect, blocking it out on the radiographs. This information can only be determined by **exploratory surgery.**

Important: Radiographs **will not** show:

1. The number of walls left surrounding the tooth
2. The exact configuration of the defect
3. The location of the epithelial attachment

Remember: The two most critical parameters for the prognosis of a periodontally involved tooth are **mobility** and **attachment loss** *(which is most critical).* Periodontal osseous defects are classified on the basis of the number of osseous walls present or remaining at the time of their surgical exposure and may have one, two, three, or four walls. **Note:** A **combination defect** is a combination lesion that is more complex and has more walls apically than coronally.

To measure attachment loss:

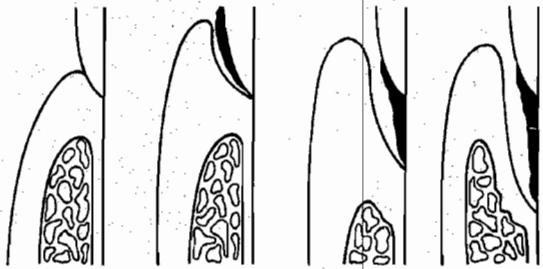
- Use periodontal probe from an **established reference point** (*CEJ or restoration margin)* for initial measurement.
- Probings are repeated after appropriate **intervals** of time and the changes are noted.
- **Example:** If probing depth is 4 mm and recession is 3 mm, the **total attachment loss is 7 mm.**

PERIODONTICS

Poc / Fur

Match the following **terms** to the appropriate **picture of pockets.**

- Gingival pocket
- Periodontal pocket
- Normal
- Periodontal infrabony pocket



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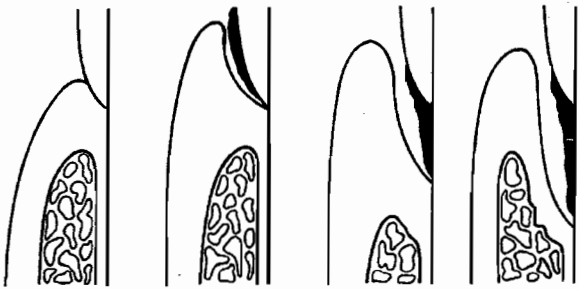

- **Gingival pocket:** A pocket formed by gingival enlargement without apical migration of the junctional epithelium. All gingival pockets are suprabony *(the base of the pocket is coronal to the crest of the alveolar bone)*.
- **Periodontal pocket:** A pocket formed as a result of disease or degeneration that caused the junctional epithelium to migrate apically along the cementum. May be suprabony or infrabony.
- **Periodontal Infrabony pocket:** A pocket in which the base of the pocket is below or apical to the crest of the alveolar bone.
- **Periodontal suprabony pocket:** A pocket in which the base of the pocket is coronal to the crest of the alveolar bone.

In a suprabony pocket, the epithelial attachment is located:

- Within basal bone
- Coronal to alveolar bone crest
- Apical to alveolar bone crest
- None of the above

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- **Coronal to alveolar bone crest**

Remember: In the **suprabony pocket** the base of the pocket is **coronal** to the alveolar crest. This type of pocket may be **further classified** as:

- A **gingival pocket** *(also called a relative or pseudopocket)* → *a* condition in which there is expansion of the marginal tissue coronally **rather than apical movement of the epithelial attachment.** Pocketing occurs **without** attachment loss. *(***See photo below)*
- A **periodontal pocket** *(also called a true pocket)* → a deepened gingival sulcus in which the epithelium has undergone ulceration. It is characterized by **the apical migration of the epithelial attachment** *(which is the inner layer of cells of the junctional epithelium)* **beyond its physiological level** *(which is normally at or near the CEJ).*

***Photo of patient who would demonstrate gingival pockets.

Remember: In periodontal osseous defects ***(infrabony pockets)*** the base of the pocket is **apical** to the crest of the bone. In order for a **defect to have** a bony wall, it must be intraosseous *(i.e., partially or completely within alveolar bone)*. Examples include **ramps,** hemiseptums, interdental craters, intrabony **defects,** and moat defects.

PERIODONTICS

Poc / Fur

In infrabony pockets:

- The bone loss is angular or vertical in nature
- The bone loss is horizontal in nature
- Transseptal fibers are horizontal
- Supracrestal fibers follow the normal bone contour

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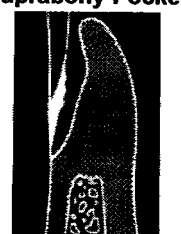
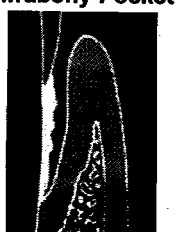
- **The bone loss is angular or vertical in nature**

Features of Suprabony & Infrabony Pockets	
Suprabony Pocket	**Infrabony Pocket**
• The base of the pocket *(epithelial attachment)* is coronal to the crest of the alveolar bone. • Destruction of the underlying bone is horizontal in nature. Further classified as gingival or periodontal pocket. • Transseptal fibers are horizontal. • Supracrestal fibers follow normal bone contour.	• The base of the pocket (epithelial attachment) is apical to the crest of the alveolar bone so that there is actually a defect or hole within the bone *(intraosseous)*. • Bone loss is angular or vertical in nature creating holes or defects within the bone. • Transseptal fibers are oblique • Supracrestal fibers follow angular pattern of osseous defect.

Suprabony Pocket



Infrabony Pocket



A furcation invasion in which bone loss allows the probe to extend more than 1 mm horizontally but not completely through the furcation is classified as:

- Grade I
- Grade II
- Grade III
- Chronic

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• **Grade II**

Note: In **trifurcated teeth,** the probe can be passed from one aspect to another *(from the mesiolingual to the buccal)*. In these cases, the furcation is still covered by gingiva.

Horizontal classification of furcations *(Glickman Classification)*

Grade I → incipient bone loss. The furcation probe can feel the depression of the furcation opening.

Grade II → partial bone loss (lesion is *Cul-de-sac, not tunnel)*. The furcation probe tip enters under the roof of the furcation.

Grade III → total bone loss with through-and through opening of the furcation *(essentially a tunnel)*. The furcation entrance is not visible.

Grade IV → a Grade III furcation where the furcation entrance is visible.

Findings that **complicate furcation involvement** and account for some painful symptoms include caries of the cementum and dentin, tooth resorption in the furcation, abscess formation in the furcation, and involvement of the pulp via lateral canals in the furcation. **Definitive** diagnosis of furcation involvement is made by careful clinical probing. X-rays are helpful but only as an adjunct to the clinical examination.

The **major principle** of treatment of involved furcas is to eliminate the involvement whenever possible. A variety of methods are available for treatment. Not all of them provide for elimination of the furcation; some provide only for increased accessibility for plaque removal. **Bone grafts have relatively little effectiveness in treating furcations. However, guided tissue regeneration** is used to treat Grade II furcations with good success. Furcation involvement of maxillary second molars have the poorest prognosis following therapy.

All of the following drugs can be associated with gingival hyperplasia **except:**

- Phenytoin *(Dilantin)*
- Cyclosporine A *(Sandimmune)*
- Paroxetine HCl *(Paxil)*
- Nifedipine *(Procardia)*
- Diltiazem *(Cardizem)*

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• **Paroxetine HCl** ***(Paxil)***

Remember: The highest incidence of drug induced hyperplasia is reported for Phenytoin *(Dilantin).*

Pseudopocketing is a condition in which pocketing occurs without attachment loss. A pseudopocket is also referred to as a gingival or relative pocket since it is caused by expansion of the marginal tissue coronally rather than an apical movement of the epithelial attachment beyond its physiological level. A classical example is **Dilantin hyperplasia.**

Dilantin hyperplasia is a progressive proliferation response to the gingiva associated with the use of sodium Dilantin *(phenytoin).* Recent studies have shown that the administration of this medication **does not** cause significant increase in fibroblastic activity. The occurrence of the gingivitis **is not automatic** with the drug therapy if the level of oral hygiene is maintained and no pre-existing gingival disease exists. ***Overgrowth appears to be related to the level of plaque accumulation. Studies have shown that between 50-60% of persons taking Dilantin will develop hyperplasia. A **significant difference** in comparing this hyperplasia to **hereditary gingivofibromatosis** is the finding that **there is an increased** accumulation of inflammatory cells in Dilantin-induced hyperplasia.

It has been estimated that 20% of patients on calcium channel blockers *(for example Nifedipine and Diltiazem)*, and 20% to 30% of patients on Cyclosporine A *(an immunosuppressant)* are susceptible to drug-induced gingival overgrowth.

Note: Gingivectomy may be used to correct **the** gingival contours **for these** overgrowth.

When using the periodontal probe to measure pocket depth, the measurement is taken from the:

- Base of the pocket to the CEJ
- Free gingival margin to the CEJ
- Junctional epithelium to the margin of the free gingiva
- Base of the pocket to the mucogingival junction

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• **Junctional epithelium to the margin of the free gingiva**

A periodontal pocket is a pathologic space between a tooth and pocket wall and its depth measured from the gingival margin to the clinical attachment level. The depth of a periodontal pocket is measured by using a **calibrated periodontal probe.**

Periodontal probing provides the **most accurate assessment** of periodontal pocket depth. The true topography of vertical osseous defects **cannot** be determined by radiographic examination alone. Bone levels may be high, yet pockets may be deep. Extensive bone loss may exist and yet be unaccompanied by pockets if the gingiva has receded. **The most important reason for using the periodontal probe is that it determines the loss of attachment.**

The probe is inserted along the long axis of the tooth into the pocket with gentle *(approximately 10 to 20 grams)* of force until resistance is met. The probe is walked around each surface of the tooth. This method is less painful and more efficient, **provides a complete and accurate assessment of the depth of the epithelial attachment and will detect bony defects better.**

Notes:

1. Bleeding scores → bleeding is still the **most reliable indicator** of the presence of gingival or periodontal inflammation.
2. Plaque score → with plaque disclosing solution, used to help visualize plaque for the patient and clinician. Also used at multiple visits to show patients improvement in their level of oral hygiene.
3. **Recession is measured** in millimeters **from the** CEJ to the marginal gingiva of each tooth root.

How should a **periodontal probe** be adapted in an interproximal area?

- It should be parallel to the long axis of the tooth at the point angle
- It should be parallel to the long axis of the tooth at the contact area
- It should touch the contact area and the tip should angle slightly beneath and beyond the contact area
- It should be perpendicular to the long axis of the tooth in front of the contact area

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- **It should touch the contact area and the tip should angle slightly beneath and beyond the contact area**

***The periodontal probe may be angled **approximately 10°** on each interproximal surface so that the tip of the probe is placed apical to the contact point of adjacent teeth and may detect any interdental crater but, in most instances, the direction of the probing is parallel to the long axis of the tooth.

Periodontal measurements are taken by inserting the probe under the marginal gingiva and gently moving it down to the junctional epithelium *(feels soft, elastic and resilient)*. In a healthy site, the tip of the probe stops within the junctional epithelium and in a diseased site it penetrates into the connective tissue. In severe disease, the probe tip may penetrate to the alveolar bone.

Notes:

1. The clinical probing depth is always greater than the histologic sulcus or pocket depth. Probing accuracy is only within +/- 1 mm.
2. The calibrated periodontal probe should have a tapered shaft approximately 0.5 mm in diameter at the tip. It is important to have uniform instruments throughout the practice in order to ensure as much standardization as possible.
3. A Naber's 2N or Hamp probe are used to detect and clinically diagnose furcation involvement.

PERIODONTICS

Prob

How should the periodontal probe be inserted into the sulcus?

- Perpendicular to the long axis of the tooth
- With a firm pushing motion
- With a short oblique stroke
- Parallel to the tooth surface

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• **Parallel to the tooth surface**

The tip of the probe should **always** be kept in contact with the tooth, thus preventing soft tissue injury. The probe is gently "walked" along the junctional epithelium in an up and down motion *(called circumferential probing)* always remaining under the gingival margin. It is imperative that the probe be walked along the entire gingival sulcus since the depth of the epithelial attachment varies. **Six measurements are recorded** – three from the buccal and three from the lingual: disto-buccal, buccal, mesio-buccal, disto-lingual, lingual and mesio-lingual. These measurements are the distance in millimeters from the base of the pocket *(junctional epithelium)* to the margin of the free gingiva.

Notes:

1. The most important reason for using the periodontal probe is that it **determines the loss of attachment.** These measurements are taken both before and after scaling and root planing procedures to evaluate the tissue response and the effectiveness of treatment.
2. Probing is performed with gentle pressure. The correct probe force *(approximately 10 to 20 grams)* depresses the thumb pad approximately 1-2 mm.

If you should meet resistance after inserting the periodontal probe into the sulcus, you should:

- Remove the probe and reinsert it in a different spot
- Lift the probe away from the tooth and attempt to move it apically
- Force the probe beyond the obstruction
- Remove the probe and select one with a narrower diameter
- Record the measurement where the probe stopped

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• **Lift the probe away from the tooth and attempt to move it apically**

Often when probing, the passage of the probe may be blocked by a hard, unyielding ledge. This is usually calculus. Gently lift the probe away from the tooth, placing it against the tissue wall of the pocket and attempt to proceed apically again. If the obstruction was indeed calculus and it has now been bypassed, then the probe should now move deeper into the pocket until the junctional epithelium is reached. The tip of the probe should be placed back against the tooth once the obstruction has been bypassed. **Remember:** The probe should be inserted parallel to the vertical axis of the tooth and **"walked" circumferentially** around each surface of each tooth to detect the areas of deepest penetration.

Notes:

1. **Recession** is the measurement of the migration of the free gingival margin apical to the CEJ of the tooth. Recession is measured as a positive value. The recession measurement added to probe depth at a particular site indicates the amount of periodontal attachment that has been lost at that site. When the gingival margin is coronal to the CEJ, the recession measurement has a negative value.
2. To measure the **amount of attached** gingiva: **(1)** Place the probe on the external surface of the gingiva and measure from the mucogingival junction to the gingival margin to determine the width of the total gingiva, **(2)** Insert the probe and measure probing depth, **(3)** Subtract the probing depth from the total gingival measurement to get the width of the attached gingiva.

Which of the following is the **most common error** when performing periodontal probing?

- Using the **wrong type** or probe
- **Incorrectly reading** the periodontal probe
- **Excessively angling the probe when inserting it interproximally** beyond the long axis of the tooth
- Forgetting to also **probe the lingual** of every tooth

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- **Excessively angling the probe when inserting it interproximally beyond the long axis of the tooth**

***This will give **greater probe readings** than are actually present.

Tilting the probe could affect the accuracy of the measurements. If the probe is angled too much, it will extend beyond the contact area and if it isn't angled enough, then it will be at the line angle instead of under the contact area. Both mistakes will result in inaccurate readings. The **tip should be flat** against the tooth near the gingival margin with the probe approximately **parallel with the long axis** of the tooth for insertion.

Note: In the **presence of inflammation,** the probe may extend apical to the most coronal extent of the junctional epithelium *(bottom of pocket)* and give a slightly greater depth than is actually present.

Important: If a patient is at risk for subacute bacterial endocarditis, he or she needs to be premedicated **before** performing periodontal probing.

Remember: Bacteremia can occur even **with** mastication or brushing. However, it does not last long. **The** important consider**ation is the** presence or absence of periodontal inflammation. **The** presence of inflamm**ation leads** to a longer duration of bacteremia with resultant risks **for patients** at risk of acute **bacterial** endocard**itis.**

When **extensive scaling and root planing** must be performed, the best approach would be:

- A series of appointments set up to scale and root plane a segment or quadrant of teeth at a time *(thoroughly and completely)*
- Gross debridement *(sub and supragingival)* of the entire mouth, followed by a series of appointments for fine scaling and polishing
- Perform everything in a single appointment
- None of the above

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- **A series of appointments set up to scale and root plane a segment or quadrant of teeth at a time** *(thoroughly and completely).*

Notes:

1. There is potential for abscess formation in a deep pocket when only a superficial scaling is performed.
2. OHI may be more effective if a patient can see healing tissue in an area which has been completely debrided and compare it to tissue in an untreated area.
3. A patient who has had a gross debridement will see a marked visual improvement of the oral cavity and may not understand the importance and necessity of the deep scaling and root planing appointments. This may cause the patient to not follow through with the scheduled treatment, and the patient's periodontal condition will be allowed to deteriorate further.
4. Post-evaluation therapy should take place approximately 4-6 weeks *(the time for repair of the dentogingival junction)* after periodontal therapy has been completed.
5. If any bleeding or swelling is noted in localized areas of the mouth during the re-evaluation appointment, check for and remove any residual calculus deposits that might remain.
6. In root planing, ideally, the **working stroke** should begin at the **apical edge of the junctional epithelium** *(the base of the pocket).*

PERIODONTICS

Scal / Rp / Gc

Which of the following presents the most difficulty in performing a thorough scaling and root planing on?

- Mesial surfaces of **maxillary premolars**
- Proximal surfaces of **mandibular incisors**
- Trifurcations of **maxillary molars**
- Distal surfaces of **mandibular molars**

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• **Trifurcations of maxillary molars**

Mesial surfaces of **maxillary premolars** and the proximal surfaces of **mandibular incisors** are most likely to have flutings. Also, root proximity is a major problem when performing scaling and root planing on mandibular incisors. **Trifurcations on maxillary first molars are the most difficult of all to root plane.**

***Key point** → anatomic features of teeth frequently limit the effectiveness and efficiency of calculus removal.

Remember: If while root planing you find only a thin ring of calculus in the bottom third of a deep pocket, you can assume that the calculus previously extended the full length of the pocket but the top part was previously removed. Likewise, if after scaling and root planing, the patient returns in one week with hard, black deposits of calculus around the gingival margin, this indicates that a reduction in inflammation occurred after the procedure and old calculus is now exposed.

Notes:

1. **The best criterion** to evaluate the success of scaling and root planing is no evidence of bleeding upon probing. **Remember:** Bleeding upon probing indicates active inflammatory periodontal disease.
2. Cementum, dentin, and calculus **are removed during** root planing.
3. **Tactile sensitivity** refers to the ability to **distinguish** degrees of roughness and smoothness on **the tooth surface**.

PERIODONTICS

Scal / Rp / Gc

Incidental gingival curettage is performed during:

- Polishing
- Scaling and root planing
- Ultrasonic scaling
- All of the above

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- **Scaling and root planing**

Incidental *(inadvertent, accidental or coincidental)* **gingival curettage** occurs during scaling and root planing. This incidental curettage includes the debridement of the lining of the sulcus or pocket. Incidental gingival curettage is caused by the outer or unused side of the curet blade. **Note: Non-definitive gingival curettage** may be indicated to improve tissue health in preparation for another definitive procedure.

Gingival curettage removes the diseased soft tissue lining the periodontal pockets. It is a manual procedure and permits a deeper and more complete cleaning than ultrasound. It does not add any significant benefits for shallow pockets. Local anesthesia is often used. Fine scaling instruments, called curets, serve two functions: **(1)** They scrape and clean the root surfaces. **(2)** They also plane the surfaces in an attempt to smooth and remove the outer layer of diseased material.

Objective of gingival curettage: Removal of chronically inflamed connective tissue, diseased sulcular epithelial lining and microorganisms from the pocket in order to reduce edema and pocket depth.

When performing gingival curettage:

- **Instruments & Angulation** → definitive curettage may be performed with a Gracey curet or a universal curet. The angulation of the instrument against the tissue should be 45° to 90°.
- **Digitally support the free gingival / pocket wall** → this increases the effectiveness of the cutting stroke
- **Strokes** → long and horizontal with moderate **pressure**
- **Irrigate frequently** while performing procedure
- **Post-operative healing** → most common response is healing by **shrinkage** of the marginal and papillary gingiva.

PERIODONTICS

Scal / Rp / Gc

The **main objective** of root planing is:

- To remove chronically inflamed tissues
- To change the bacterial microflora
- To provide optimally smooth root surfaces
- To eliminate pockets

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• **To provide optimally smooth root surfaces**

***By providing smooth root surfaces there will be a reduced potential for bacterial accumulation, which is done in an attempt to achieve soft-tissue re-attachment.

Scaling and root planing are techniques of instrumentation applied to the root surface to **divest it of plaque, calcified deposits and softened or roughened cementum.** When thoroughly performed, these techniques produce a smooth, clean, hard polished root surface. Scaling and root planing are the **primary treatment** for periodontal inflammation. In simple cases, this treatment is useful in reducing shallow pockets and reducing the number of bacteria within these shallow pockets and may be the only treatment necessary. In severely advanced periodontal disease where surgery may not be possible, scaling and root planing are the only treatment feasible. Since the removal of plaque and deposits is the **definitive treatment** for periodontal inflammation, root planing and scaling **are more frequently** used than any other type of therapy. Commonly observed clinical changes one week after scaling and root planing include a reduction in pocket depths and a reduction in gingival inflammation.

The **most effective instrument** for subgingival scaling and root planing is a **sharp curet.** They are generally smaller than scalers and are designed to permit atraumatic entry to the subgingival space. The tactile sensitivity of most curets is greater than scalers, and, as such, curets are well suited for subgingival calculus detection, calculus removal, and root planing. The **working angulation** with a curet is less than 90° but more than 45°. The best clinical aid in determining whether subgingival calculus has been removed is using an **explorer** and **bite-wing x-rays** *(will show the presence of interproximal calculus).*

Remember: A **chisel** is best designed for **removing supra**gingival calculus deposits in interproximal areas, particularly **on anterior** teeth. A chisel **has a** single, straight **cutting edge.** The end of the blade is flat and beveled **at a 45° angle.**

Maximum shrinkage after gingival curettage can be expected from tissue that is:

- Fibrotic
- Edematous
- Fibroedematous
- Formed within an infrabony pocket

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- **Edematous**

Indications for gingival curettage:
- When a gingival inflammation persists **after** careful and thorough root planing
- In the treatment of **shallow** *(up to 4 mm)* **pockets** where the gingival tissues are **highly edematous**

Contraindications of gingival curettage as a definitive procedure include:
- Acute periodontal inflammation
- Firm, fibrotic tissue
- Infrabony pockets
- Mucogingival involvements
- When the lateral gingival wall is extremely thin

Notes:

1. Patients with edematous and granulomatous inflammation **respond better** to curettage **than do those** with conditions of fibrous hyperplasia.
2. For a **new attachment** to occur: **(1)** An adequate number of undifferentiated mesenchymal cells must be present, **(2)** Complete removal of junctional and pocket epithelium must be accomplished, as well as, **(3)** The complete removal of calculus and / or altered cementum.

Important: It is **recommended that** all students **read** The American Academy of Periodontology Statement Regarding Gingival Curettage. This can be found on the internet at: **http://www.perio.org/resources-products/pdf/38-curettage.pdf**

The ultrasonic tip **should not** be allowed to remain on the tooth surface for **too long** because it will:

- Burnish the calculus onto the tooth surface
- Stop the vibrations of the tip
- Clog the water that circulates through the handpiece
- Damage the tooth surface

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- **Damage the tooth surface**

Indications for the use of ultrasonic devices:

- **Supragingival** calculus removal
- **Subgingival** debridement
- **Initial debridement** of a patient with ANUG
- **Gross scaling** prior to extractions
- **Removal of** orthodontic cement, bonding material, and overhanging restorations

When to use ultrasonic scaling devices **cautiously:**

- **Creates an aerosol spray** → watch with patients with known communicable diseases
- **Compromised patients** who are susceptible to infection
- Patients with a **respiratory risk**
- Patients prone to **gagging** *(excessive water)*
- **Do not use ultrasonic devices** of any sort on or around patients with **cardiac pace makers** unless pacemaker is shielded *(newer models).* **Recommend consultation with cardiologist first.**
- **Children**
- **Demineralized areas**
- **Exposed cementum**
- **Restorative materials** *(i.e., porcelain crowns, titanium implants)*
- Patients with **sensitive areas**
- Be careful **subgingivally** → lack of visibility / **narrow** pockets

Note: Subgingival root surface roughness **does not** seem to interfere with healing after scaling and root planing. **Thus it does** not appear useful to reinstrument root **surfaces with** hand instruments after a clinically detectable **smooth** surface has **been** created **with sonic or** ultrasonic scaler.

PERIODONTICS

Scal / Rp / Gc

Which of the following instruments is considered **least traumatic** and **most effective** for performing nonsurgical root planing procedures?

- An ultrasonic instrument
- A hoe
- A periodontal curet
- Periodontal files

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• **A periodontal curet**

The curet is a narrow, delicate instrument having either one or two cutting edges *(or working surfaces)*. **Only one** cutting edge is used and the cutting edges are **parallel and curved** to the side. Most often curets are in paired forms allowing for access to opposite surfaces. They are generally smaller than scalers and are designed to permit atraumatic entry to the subgingival space. The tactile sensitivity of most curets is greater than scalers, and, as such, curets are well suited for subgingival calculus detection, calculus removal, and root planing.

Important: When root planing, **vertical strokes** should be used first, followed by **oblique and then horizontal strokes.** Light pressure should be utilized with root planing strokes to maximize tactile sensitivity.

Notes:

1. Using curets with short, even working strokes followed by longer ones is the most effective and efficient way of performing root planing. The **correct cutting edge** can be seen as a larger, outer curve.
2. Final root planing strokes are longer and lighter than scaling strokes.
3. Root planing strokes become lighter as the cementum becomes smoother.
4. Exploratory scaling and root planing strokes differ in angulation, pressure, length, and direction.

Remember: To establish the correct working **angle once** a curet is inserted subgingivally, the shank must be moved away from the tooth in order to **open the** angle of the blade to the tooth surface. At proper working angulation *(less than 90° but more than 45°)*, the lower shank of a **Gracey curet** is **parallel** to the **tooth surface** being scaled. **The** lower shank of a **universal curet** would be tilted **slightly toward the tooth.**

Indications or objectives of gingival or subgingival curettage include all of the following **except:**

- To eliminate the cause of periodontal inflammation
- As maintenance treatment for areas of recurrent inflammation and where pocket reduction surgery has previously been performed
- To reduce inflammation prior to pocket elimination
- To remove chronically inflamed periodontal tissues

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• **To eliminate the cause of periodontal inflammation**

***This is **not an objective of curettage** → the goals and objectives scaling and root planing include the elimination of the cause of inflammation. **Remember,** in addition to the removal of plaque, stain, and calculus, scaling and root planing also includes the removal of "disease affected" cementum containing embedded calculus, whole bacteria, and toxic bacterial debris such as endotoxin.

Gingival curettage is performed when the cutting edge of the curet is directed against the soft tissue wall of the pocket. The **term curettage** refers only to treatment of the pocket wall *(removal of sulcular epithelium and inflamed connective tissue)*, whereas **root planing** is the instrumentation applied to the tooth surface to divest it of deposits and to smooth it. Gingival curettage is frequently **performed with root planing** to promote soft-tissue attachment *(re-epithelialization)*, which usually occurs within 7-10 days.

Notes:

1. The **most important factor** in determining the amount of shrinkage is the **degree of edema** in the tissue. Healing begins with formation of the blood clot. Neutrophils predominate immediately following curettage *(for the first 12 hours)*.
2. During **gingival curettage,** complete **removal of all** diseased tissue may be difficult **when the tissue** is very soft, loose, and **edematous.** In such a case, applying finger pressure to support the marginal gingiva **will help** in the removal of the diseased tissue.

All of the following statements regarding sonic and ultrasonic instruments are true **except:**

- Sonic instruments do not release heat the way ultrasonic instruments do
- Magnetostrictive units of ultrasonic scalers have a linear pattern of vibration of the tip
- Ultrasonic instruments may be used for scaling, curetting as well as removing stains
- Ultrasonic scalers can remove calculus from both supragingival as well as subgingival sites
- Ultrasonic scaling is based on the principle of high-frequency sound waves

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• **Magnetostrictive units of ultrasonic scalers have a linear pattern of vibration of the tip**

Remember: Magnetostrictive units have an **elliptical** pattern of vibration of the tip, which means that all sides of the tip are active and will work when adapted to the tooth. In **piezoelectric** units the pattern of vibration of the tip is **linear, or back and forth,** meaning that only the two sides of the tip are active and will work when adapted to the tooth.

Power-driven scalers use either ultrasonic or sonic energy for debridement. **Ultrasonic** scaling devices use either magnetostrictive or piezoelectric technology to convert electrical energy to physical energy at the instrument tip. The instrument tips vibrate from **25,000-40,000 cycles per second** at an amplitude between 10-13 μm. When the tip of the instrument is held against calculus, stain, cement and other substances adhering to the tooth surface, they are broken up and removed. The **side of the tip**, not the pointed end, should be applied so that the tooth surface is not gouged or injured in any way. The heat generated by magnetostrictive instruments requires copious amounts of water for cooling. In the cooling process, water **"cavitation"** occurs which releases dissolved gases. The resulting spray, along with the water itself, facilitates the removal of dislodged debris.

Sonic instruments are classified as air-turbine instruments. They use air pressure from high-speed handpiece lines to produce tip vibrations from **2,000-6,000 cycles per second.**

Important: Power-driven instruments must **be adequately** cooled, be kept in constant motion, and the side *(not the tip)* of the instrument must **be used** against the tooth structure at all times. Many ultrasonic units **may be used** with a variety **of irrigants** such as **chlorhexidine** or providone-iodine.

PERIODONTICS

Scal / Rp / Gc

If a patient experiences **sensitivity** while being scaled with an **ultrasonic scaling device,** all of the following actions will be appropriate to counter this problem **except two.** Which two are inappropriate?

- **Proceeding to another tooth** and then returning to the sensitive tooth later in the appointment
- Moving the **instrument slower**
- Making necessary **adjustments** to the **water spray**
- Turning **up** the **power** of the device
- Using **less pressure**

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- **Moving the instrument slower**
- **Turning up the power of the device**

***The opposite of these is true!! All of the rest are appropriate actions to be considered in that situation.

If sensitivity is encountered during use:

- Lighten the pressure
- Adjust the water spay → increase water to cool tip
- Move to another tooth and then return later to the sensitive tooth
- Decrease the power

Power driven instruments **work best** with quick hand movement → **rapid, controlled movements.**

Remember: ***The ultrasonic principle is based on the use of high-frequency sound waves.

Air is used to deflect the free gingival margin in order to detect:

- Supragingival calculus
- The CEJ
- Smooth root surfaces
- Subgingival calculus
- Inflammation

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• **Subgingival calculus**

One of the uses of the **air syringe** is to detect calculus, both supra and subgingival. Supragingival calculus is often seen more easily when it is dry *(saliva often conceals it)*, deflecting the free gingival margin slightly makes subgingival calculus easier to detect.

When using the **explorer** to detect calculus, a light instrument grasp should be utilized to increase tactile sensitivity. The lateral side of the tip of the instrument should be placed in contact with the tooth surface when exploring for calculus. Dried calculus is easier to detect than wet calculus with the explorer because it is less slippery.

PERIODONTICS

Scal / Rp / Gc

The **primary function** of the periodontal file is to:

- Root plane
- Remove heavy supragingival calculus
- Crush or fracture heavy tenacious calculus
- Completely remove heavy subgingival calculus

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• Crush or fracture heavy tenacious calculus

Periodontal files and hoes are designed primarily to **crush or fracture accessible supragingival calculus.** Because of the thickness of their blades, they lack adaptability and tactile sensitivity. They may be used subgingival for gross calculus removal **only if** the tissue is flexible and easily displaced. Since they are for gross calculus removal only, they should always be followed by a curet. *** Their cutting edges are designed to function at right angles to the tooth surface.

Hoes → their wide, straight cutting edge is unable to be adapted to curved tooth surfaces *(injury to both tooth and tissue could occur).* The entire length of the cutting edge must be kept against the tooth and only **vertical pull-type strokes** should be used. Angle the cutting edge at 90° to the tooth surface. Hoes are most effective on **buccal and lingual surfaces** and mesial and distal adaptation is poor, if not impossible. Maintaining a two-point contact with the tooth *(cutting edge and side of shank)* increases instrument stability. The hoe has a single, straight cutting edge. The blade is turned at a 90°-100° angle to the shank and the cutting edge is beveled at a 45° angle to the end of the blade. **This angle should be maintained when sharpening the hoe.**

Periodontal files → the **primary function** of the file is to crush or fracture heavy calculus deposits, however, the entire deposit is not removed. The file should always be followed by root planing with a curet. Files work best on **buccal and lingual surfaces** and next to edentulous areas. Although their size and lack of adaptability make interproximal use difficult, they work well on the distal surface of the last molar, where access is often limited. Vertical pull-type strokes are used with this instrument. The file is also used to reduce amalgam overhangs.

While scaling **subgingivally** the tip of the curet breaks off. All of the following are appropriate actions to take to try and remove this tip **except:**

- Use a push stroke to force the tip out of the sulcus
- Gently examine the gingival sulcus
- Take a periapical radiograph of the area
- Place the patient in an upright position

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• **Use a push stroke to force the tip out of the sulcus**

***Never use a push stroke,** this could force the tip further into the sulcus. Gently examine the sulcus using a curet in a spoon-like stroke, attempting to pull the fragment out.

The procedure should be **stopped immediately** and the patient placed in an upright position. Before proceeding subgingival, check the floor of the mouth and the mucobuccal fold for the fragment. A periapical radiograph should then be taken before going any further to locate the fragment. Try not to alarm the patient. The last thing you want is for the patient to become frantic.

The **best way to prevent** curet breakage is to use proper sharpening techniques to maintain the original instrument design and discard instruments when the blade starts to thin out. A thinner blade is weaker and will break more easily.

Comparison of Gracey and Universal Curets

Characteristic	Graceys	Universal
Area of use	Area specific — set of many designed for specific areas and surfaces.	Universal — one curet designed for all areas and surfaces.
Blade angle	Offset blade — face of blade beveled at 60° - 70° to shank.	Not offset — face of blade beveled at 90° to shank.
Use of cutting edges	Only one cutting edge used — work with larger outer cutting edge only.	Both cutting edges used — work with either cutting edge.
Curvature of cutting edges	Curved in two planes — blade curves up and to the side.	Curved in one plane only — blade curves up, but not to the side.

Regarding instrument sharpening:

A. A wire edge is produced when the direction of the sharpening stroke is **away** from, rather than into or toward, the cutting edge

B. Oil should be used for synthetic stones and water for natural stones

C. Sharpen instruments at the first sign of dullness

D. The angle between the face of the blade and the stone is kept at 100° to 110°

E. The technique to sharpen Gracey curets is essentially the same for universal curets

- (A) only
- (A), (B), and (C) only
- (B) and (E) only
- (A), (C), (D), and (E) only

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- **(A), (C), (D) and (E) only**

***The only **incorrect** statement is (B). Oil should be used for natural stones and water for synthetic *(artificial)* stones.

Principles of sharpening:

- Choose sharpening stone appropriate for instrument *(flat, cone, coarse, etc.)*
- Make sure sharpening stone is sterile when sharpening any instrument, while working on a patient
- Establish proper angle between stone and specific instrument
- Maintain firm grasp of both stone and instrument
- Avoid excessive pressure
- Avoid producing a "wire edge" by always finishing with a **down** stroke
- Lubricate stone
- Sharpen instruments at **first** sign of dullness
- The technique for sharpening a universal curet can be used to sharpen a Gracey curet, however, bear in mind that the Gracey's cutting edge is **offset** and it also curves, unlike the universal's cutting edge
- Arkansas and India stones are examples of natural while Carborundum, ruby, and ceramic stones are synthetic *(artificial)*

Important: A properly sharpened instrument with no rounded surface area will not reflect light.

When sharpening, a wire edge is produced:

- Only when using a coarse artificial stone
- When using a mounted ruby stone only
- When no oil is used for lubrication of the stone
- When the last stroke of the stone is drawn away from the cutting edge

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• **When the last stroke of the stone is drawn away from the cutting edge**

***Avoid producing a "wire edge" by always finishing with a **down** stroke.

The **lubricant allows** the metallic particles *(removed from the instrument during sharpening)* to become suspended in the lubricant. This helps prevent scratching and "glazing" of the stone. A sludge of metal shavings and oil that develops on the face of the blade indicates that the cutting edge may be sharp.

The sharpening technique used is an important consideration when it comes to maintaining the instrument's original shape. **Manual sharpening methods are preferred over the use of mandrel mounted stones.** Manual sharpening helps to prevent the unnecessary reduction of the blade which may occur when using a rapid cutting mounted stone. When sharpening instruments with a flat stone, the angle between the instrument and the stone should be between 100° and 110°.

Notes:

1. Instruments **must be sharp** if scaling and root planing are to be completed efficiently with minimal trauma to the tissues. A sharp instrument will require fewer strokes, provide greater control, and increase tactile sensitivity.
2. With a **dull instrument,** the handle must be grasped much more firmly and with more pressure applied to the tooth surface. Dull instruments result in fatigue due to heavy-handedness, burnished calculus, slipping, decreased tactile sensitivity, and inefficient use of time.
3. The **optimal internal angle** between the face of **the blade** and the lateral surface of a universal curet and a Gracey curet is 70° to 80°.
4. An instrument **whose cutting edge is 90° or more will slip over** calculus deposits and requires heavy lateral pressure to remove calculus deposits.
5. The best grasp **to use when** holding an **instrument** to be sharpened **is the palm grasp.**

PERIODONTICS

Scal / Rp / Gc

A curet designed to scale and root plane anterior teeth with deep pockets will have a:

- Short, straight shank
- Long, straight shank
- Short, angled shank
- Long, angled shank

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• **Long, straight shank**

How an instrument shank is designed influences the intended use of the instrument. It is recommended that an instrument with a rigid shank be used for removal of heavy calculus deposits. ***Straight shanks are used in the anterior areas and contra-angled shanks are used in the posterior areas.

Rigid, thick shank:
• Stronger
• Less flexible
• Provides **less** tactile sensitivity
• **Stronger instruments are needed for heavy calculus removal**

Less rigid, more flexible shank:
• Provides **more** tactile sensitivity
• **Used for removing fine calculus and for root planing**

Important: The **ideal working angulation** with a curet is less than 90° but more than 45°. If the angulation of the curet were more than 90° the lateral surface would be against the tooth, calculus would not be effectively removed and the free cutting edge would remove soft tissue. The **objective of proper adaptation** of the curet blade throughout the scaling and root planing process is to insure maximum effectiveness of instrumentation and to avoid trauma to the soft tissues.

Notes:

1. The **blade of a universal curet** is honed **at a 80° to 90°** angle to the lower shank.
2. Design features **of a universal** curet include: **(1) Both** cutting **edges are used**, (2) Cutting edges are parallel and **straight**.

PERIODONTICS

Scal / Rp / Gc

Match each Gracey curet with its intended area of use by filling in the space with the appropriate letters. *(Letters may be used more than once).*

- Gracey #1 and #2 ____
- Gracey #3 and #4 ____
- Gracey #5 and #6 ____
- Gracey #7 and #8 ____
- Gracey #9 and #10 ____
- Gracey #11 and #12 ____
- Gracey #13 and #14 ____
- Gracey #15 and #16 ____

A. Anterior teeth

B. Posterior teeth - mesial surfaces

C. Posterior teeth - distal surfaces

D. Posterior teeth - buccal and lingual surfaces

E. "Universal" curets

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Gracey curets:

- (#1 and #2) **A** → have a short shank distance and are utilized on the interproximal surface of anterior teeth as well as the buccal and lingual-palatal surfaces in posterior teeth.
- (#3 and #4) **A** → are designed with a short modification of the shank and are used in the same region as #1 and #2.
- (#5 and #6) **A** → are available in two different shank lengths and are used in the same region as #1 and #2.
- (#7 and #8) **E** → are referred to as universal because of their application to several areas and tooth surfaces. They have limitations on the distal surfaces of posterior teeth.
- (#9 and #10) **D** → are of a long contra-angle design to instrument the buccal and lingual surfaces of molar and premolar teeth.
- (#11 and #12) **B** → are easily applicable to the mesial surfaces of posterior teeth from both the buccal and palatal.
- (#13 and #14) **C** → provide excellent access to the distal surfaces of posterior teeth.
- (#15 and #16) **C** → provide excellent access to the distal surfaces of posterior teeth.

***Universal curets** *(Crane-Kaplan 6, McCall's 17s / 18s, Columbia 4R / 4L)* have two cutting edges at 90° to the root surface and may be used in any region of the mouth. **Gracey curets** have a single cutting edge at 60° to the root surface and are designed to instrument specific tooth surfaces in specific regions of the mouth. Curets may be used with vertical, oblique, horizontal, or circumferential pull-type stokes. **Purposes and use of curets** → subgingival scaling and root planing, supragingival calculus removal and gingival curettage.

The **push** stroke:

- Is directed in the same plane as the long axis of the tooth
- Is likely to injure the junctional epithelium
- Is good for posterior teeth where vertical strokes are difficult
- Is directed circumferentially around the tooth

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- **Is likely to injure the junctional epithelium**

There are **three basic strokes** used during instrumentation. Any of these basic strokes may be activated by a pull or a push motion, although the pull motion is more widely used and is considered safer for calculus removal.

1. **Exploratory or assessment stroke** → used to assess the smoothness or roughness of the tooth surface and the effectiveness of instrumentation. **Instrument handle is grasped lightly to increase tactile sensitivity.**
2. **Scaling stroke** → short, powerful **"pull"** stroke used to remove calculus *(more pressure)*. The scaling motion is preferably initiated in the forearm and transmitted from the wrist to the hand with a slight flexing of the fingers. Rotation of the wrist is synchronized with movement of the forearm.
3. **Root planing stroke** → long, overlapping strokes for final smoothing of the root surface, usually in a **"pull"** motion *(less pressure)*.

Notes:

1. "Pulling" strokes are safer than "pushing" strokes *(pushing strokes can cause calculus to become embedded in soft tissue)*.
2. Strokes can be directed vertically, horizontally, or obliquely *(diagonally)*. Strokes in a vertical or oblique direction are used most frequently.
3. The pressure applied becomes lighter as the surface becomes smoother.
4. Probing stroke → upward and downward movement within a periodontal pocket.
5. The modified pen grasp is the most useful grasp for periodontal instrumentation.

PERIODONTICS

Rt Sens / Rec

Toothbrush trauma *(abrasion)* usually occurs on:

- Centrals and laterals
- Canines and premolars
- Second and third molars
- First and second molars

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• **Canines and premolars**

***Toothbrush trauma** *(abrasion)* usually occurs on teeth that are the **most prominent in the dental arch.**

The maintenance of oral health is enhanced by the use of a soft-bristle toothbrush with a dentifrice of optimum abrasiveness and a dental floss. A hard-bristle toothbrush is capable of causing gingival recession and abrasion of enamel and / or cementum and should be avoided. The abrasive quality of dentifrices affects enamel, but abrasion is more of a concern for patients with exposed dentin because dentin is abraded **25** times faster and cementum **35** times faster than enamel. This can lead to root surface abrasion and root sensitivity. Hard tissue damage from oral hygiene procedures is mainly due to **abrasive dentifrices,** whereas gingival lesions can be produced by the **toothbrush alone.**

Trauma from toothbrushing may result in the following:

- Recession of the **marginal gingiva**
- **Lacerations of the soft tissues** including the attached gingiva and the alveolar mucosa
- **V-shaped notches** in the cervical areas of teeth
- Gingival clefts → which are narrow grooves that extend from the crest of the gingiva to the attached gingiva

Note: The location of the above alterations **is frequently inversely** related to the right- or left-handedness of the patient.

Remember: In **gingival atrophy or recession, the** tissue appears to be otherwise normal. The gingiva is **thin, finely** textured and **pale pink** in color with **normal papillae.** The gingival sulci is very shallow. **Plaque is minimal.**

The most accepted theory as to the **cause of root sensitivity** is the:

- Bayer's theory
- Chemoisomotic theory
- Hydrodynamic theory
- Quantum theory

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• **Hydrodynamic theory**

The **Hydrodynamic theory** postulates that the pain of root sensitivity results from indirect innervation caused by **dentinal fluid movement in the tubules,** which stimulates mechanoreceptors in the pulp.

Dentinal hypersensitivity is a particular problem in patients immediately after periodontal surgery that results in the clinical exposure of root surfaces *(dentinal tubules are not adequately sealed).* **The primary symptom is cold sensitivity.** Plaque and food debris, if allowed to remain on exposed root surfaces, often lead to increased sensitivity. **Note:** To reduce the sensitivity to thermal change after removal of a periodontal dressing, it is best to keep the roots **free of plaque.**

Numerous forms of treatment have been used:

- **Topical fluorides** → sodium fluoride *(2.0%)* and stannous fluoride *(8%).* Acidulated phosphate fluoride is contraindicated due to the high acid pH.
- **Fluoride mouth rinses** → over the counter sodium fluoride *(0.05%)* or prescription acidulated phosphate-fluoride *(0.04%)* and sodium fluoride *(0.2%).*
- **Desensitizing toothpastes** → contain either strontium chloride *(found in Sensodyne toothpaste),* potassium nitrate or sodium citrate. Recommend to patient a low abrasive dentifrice.
- **Iontophoresis** → electroplating fluoride to **root surfaces.**
- **Dentin bonding agents** applied to root **surface** → **All** Bond, Scotchbond, Gluma, Amalgambond.
- **Root coverage with gingival surgery**

***None of the above have been totally effective. **Often diligent oral hygiene alone solves the problem of sensitivity.**

The most important factor in the control of hypersensitive roots among patients with periodontal disease after gingival recession has exposed the cervical portions of teeth is:

- Prescription of home fluoride rinses
- Minimal removal of tooth structure during root planing
- The application of desensitizing agents that contain fluoride
- Thorough daily plaque control

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• **Thorough daily plaque control**

The acids and toxins produced by the plaque organisms are very irritating to the pulp by way of the odontoblastic processes. This irritation of the pulp heightens its sensitivity to other stimuli. No attempt to reduce hypersensitivity will be successful unless the roots are consistently kept free of plaque.

The rationale of in-office desensitization procedures is not fully understood. Some procedures may depend on denaturation of the superficial end of Tomes' fibers or of nerve endings in dentin. Other procedures are designed to deposit an insoluble substance on the ends of the fibers or nerves to act as a barrier to stimuli. Still others are designed to stimulate secondary dentin formation *(reparative dentin),* thus insulating the pulp from external stimuli.

The **most common cause** of gingival recession is tooth injury *(abrasion)*. This type of recession is common on the left canines of right-handed persons *(or right canines of left-handed persons).*

The hygienist or dentist **should evaluate** the brushing technique and monitor hard and soft tissue conditions at each recall visit. Faulty placement, overaggressive movement or pressure, or the use of a hard toothbrush can lead to hard and soft tissue damage.

Sometimes these areas will become sensitive if the root is exposed. **The patient will complain of cold sensitivity.** The hypersensitivity will sometimes subside in time with **daily plaque removal using a soft brush** *(this will help desensitize the root surface by allowing remineralization of the root surface).* **Remember:** Gingival recession **can also occur,** secondary to periodontal therapy. This may have additional significance in the older patient, namely, increased risk for cervical abrasion, dentinal sensitivity, and most importantly, predisposition to root caries.

The bacteria that form plaque and calculus release toxins that stimulate the immune system to over produce powerful infection-fighting factors called:

- Free radicals
- Cytokines
- Amides
- Lymphokines

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• **Cytokines**

Evidence now suggests that periodontal disease may be an autoimmune disorder, in which immune factors in the body attack the person's own cells and tissues.

The bacteria that form plaque and calculus release toxins that stimulate the immune system to over produce powerful infection-fighting factors called **cytokines.** Cytokines of particular importance in periodontal disease are known as tumor necrosis factor-alpha *(TNF-alpha)* and interleukin-1beta and interleukin 4 *(IL-1 beta and IL4)*, which are very active in the mouth. Ordinarily, cytokines are important for healing. **In excess,** however, they can cause inflammation and severe damage. These cytokines overproduce an enzyme called **collagenase,** which breaks down proteins, including the connective tissue that supports the teeth. **Note:** Studies suggest that this inflammatory response may have damaging effects not only on the connective tissue that supports the teeth but also in organs throughout the body, including the heart.

A wide variation in host response to bacterial challenge between patients is usually seen. Some individuals with heavy plaque accumulation and high proportions of pathogenic organisms appear relatively resistant to bone and attachment loss. Others develop extensive periodontal destruction in the presence of small amounts of plaque and low proportions of putative pathogens.

Those patients with abnormally exuberant inflammatory responses often have a **hyperinflammatory monocyte / macrophage phenotype.** These individuals secrete significantly increased levels of pro-inflammatory mediators like **IL-1 beta, IL4, and TNF-alpha** in response to bacterial byproducts.

Which of the following is the single major preventable risk factor for periodontal disease?

- Smoking
- Diabetes
- Genetics
- Pathogenic bacteria

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• **Smoking**

Smoking is the **single major preventable risk factor** for periodontal disease, and can cause bone loss and gingival recession even in the absence of periodontal disease. A number of studies indicate that smoking and nicotine increase inflammation by reducing oxygen in gingival tissue and triggering an over-production of immune factors called cytokines *(specifically interleukins),* which in excess are harmful to cells and tissue. The risk of periodontal disease increases with the number of cigarettes smoked per day. Smoking cigars and pipes carries the same risks as smoking cigarettes.

Most systemic diseases and conditions that may affect periodontal diseases generally alter host barrier and host defense mechanisms. Although many conditions cause gingival inflammation and ulcers, **not all** people develop periodontal disease. Certain factors put individuals at higher risk than others.

Much evidence exists on the link between **diabetes type 1 and 2** and periodontal disease. People with these diseases have 15 times the risk of the nondiabetic population. Diabetes causes abnormalities in blood vessels and high levels of specific inflammatory chemicals, such as interleukins, that significantly increase the chances of periodontal disease.

Osteoporosis *(loss of bone density)* has been associated with periodontal disease in postmenopausal women. There is some evidence that some treatments for osteoporosis, such as bisphosphonates, may reduce bone loss, including the bony structures that support the teeth.

Autoimmune conditions *(e.g., Crohn's disease, rheumatoid arthritis, lupus erythematosus, CREST syndrome)* have been associated with a higher incidence of periodontal disease.

In a commercial toothpaste, **sodium benzoate** functions as a (an):

- Humectant
- Preservative
- Surfactant
- Binder or thickener
- Abrasive

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• **Preservative**

Commercial toothpastes contain ingredients that can typically be divided into **9** categories:

1. **Fluoride** *(sodium fluoride or sodium monofluorophosphate)* makes tooth enamel resistant to acids.
2. **Abrasives** *(calcium phosphate or calcium carbonate)* remove stains and plaque.
3. **Surfactants or Detergents** *(sodium lauryl sulfate)* create a foam to keep the toothpaste in the mouth.
4. **Humectants** *(glycerin or water)* give toothpaste texture and allow the mouth to retain moisture.
5. **Binder or Thickener** *(carrageenan or cellulose gum)* adds to the texture.
6. **Preservatives** *(sodium benzoate)* prevent the growth of harmful microorganisms in the toothpaste.
7. **Flavoring agents** improve the taste and mask the taste of the detergents.
8. **Sweetener** *(saccharin)* improves the taste
9. **Coloring agents** *(titanium dioxide to make it white or artificial dyes to make it red / blue / green, etc.)* give the toothpaste an appealing color.

Other active ingredients that may be found in some toothpastes → triclosan *(extremely potent anti-bacterial agent found in Colgate's Total toothpaste*) as an antiplaque agent, pyrophosphate as an anticalculus agent, potassium nitrate as a desensitizing agent, or peroxide compounds as whitening agents.

PERIODONTICS

P / C

The source of minerals for subgingival calculus is:

- Food
- Crevicular fluid
- Blood
- Saliva

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- **Crevicular fluid**

***The source of minerals for **supragingival calculus** is the saliva.

Supragingival calculus occurs above the free gingival margin. It's usually white or pale yellow in appearance and is easily removed by a professional cleaning. It occurs most frequently on the tongue side of the mandibular incisors and cheek side of the maxillary molars. It usually occurs in these two locations due to the presence of salivary ducts which secrete saliva rich in minerals necessary for its formation.

Subgingival calculus occurs below the free gingival margin. It tends to be much darker in color *(due to breakdown products of blood)* and more difficult to remove than supragingival calculus. It's generally distributed evenly throughout the mouth and is formed from gingival fluid secretion.

The most common mechanism of **supragingival calculus** attachment to smooth enamel surfaces is the **salivary pellicle.** The attachment of **subgingival calculus** is complicated by irregularities such as cemental tears, cemental voids once occupied by Sharpey's fibers, resorption bays, and other cemental defects.

Notes:

1. Calculus exerts its pathogenic potential as a contributing factor that fosters plaque formation and promotes its retention on teeth.
2. The rough surface of calculus is usually covered with a layer of plaque biofilm.
3. Calculus tends to "present" plaque via the biofilm to periodontal soft tissues.

Prevotella intermedia was formerly known as:

- Bacteroides melaninogenicus
- Wolinella intermedius
- Bacteroides gingivalis
- Bacteroides intermedius

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• **Bacteroides intermedius**

The purpose of this card is to hopefully clear up any confusion on the recent reclassifications of a number of plaque microorganisms that are important in periodontal disease development. The **bacteria have stayed the same,** but the **names have changed.**

Recent Reclassification's of Periodontal Microorganisms	
Previous Classification	**New Classification**
Bacteroides gingivalis	Porphyromonas gingivalis
Bacteroides endodontalis	Porphyromonas endodontalis
Bacteroides intermedius	Prevotella intermedia
Bacteroides melaninogenicus	Prevotella melaninogenica
Bacteroides denticola	Prevotella denticola
Bacteroides loescheii	Prevotella loescheii
Wolinella recta	Campylobacter rectus
Wolinella curva	Campylobacter curvus

ORAL PATHOLOGY

Legend

Major Topic	Abbreviation	Major Topic	Abbreviation
Abnormalities of Teeth	Abn of Tth	Odontogenic Tumors	Odont Cyst
Connective Tissue Lesions	Con Tis Les	Pigmented Lesions of the Oral Cavity	Pig Les of Oral Cav
Diseases of the Blood	Bld Dis	Pseudocyst	Pseudocyst
Inflammatory Jaw Lesions	Infl Jaw Les	Red-Blue Lesions	R-B Les
Metabolic & Genetic Jaw Diseases	M & G Jaw Dis	Salivary Gland Tumors	SG Tum
Miscellaneous	Misc.	Terms	Terms
Neoplasms	Neo	Ulcerative-Conditions	Ulc Cond
Nerve & Muscle Disorders	Nrv & Mus Disord	Verrucal-Papillary Lesions	V-P Les
Non-Odontogenic Cysts	N-O Cysts	Vesiculo-Bullous Diseases	V-B Dis
Non-Odontogenic Tumors	N-O Tum	White Lesions	W Les
Odontogenic Cysts	Odont Cyst		

The congenital absence of **all teeth** is referred to as:

- Hypodontia
- Anodontia
- Diphyodontia
- Hypsodontia

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• **Anodontia**

Two forms of anodontia:

1. **Complete true** → is a rare condition in which **all of the teeth** are missing. It may involve both the primary and permanent dentitions. It is usually associated with **hereditary ectodermal dysplasia. See picture #1 in booklet.**

2. **Partial anodontia** *(commonly referred to as* **congenitally missing teeth***)* → is rather common. Teeth usually affected include the third molars *(maxillary more often than mandibular),* maxillary lateral incisors and mandibular second premolars.
   Note: As a general rule, if only one or a few teeth are missing, the absent tooth will be the **most distal tooth** of any given type *(if molar, then it would be the third molar).* **See picture #2 in booklet.**

Other terms to be familiar with include:

- **Oligodontia** → refers to the congenital absence of many *(usually six or more)* but **not all,** teeth.
- **Hypodontia** → refers to the absence of **only a few** teeth.
- **Diphyodontia** → is having 2 successive sets of teeth *(as in humans)* as opposed to polyphyodontia *(having more than two sets of teeth in a lifetime).*
- **Hypsodontia** → having teeth with high crowns *(cusps).* Hypsodont dentition is associated with a diet of abrasive foods.

All of the following statements concerning **dentinogenesis imperfecta** are true **except:**

- It is an inherited disorder of the dentin
- Type II is most common *(incidence is 1:8000 newborns)*
- It is autosomal recessive
- Type I is associated with osteogenesis imperfecta
- The teeth are composed of irregularly formed and undermineralized dentin that obliterates the coronal and root pulpal chambers
- Affected teeth are more susceptible to root fractures

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- **It is autosomal recessive**

***This is **false;** it is autosomal dominant and is found in about 1:7,000 children.

Teeth affected with dentinogenesis imperfecta have an amber, gray, or purple **opalescence** *(translucence)* or discoloration. The pulp chamber may be **completely obliterated.** This is due to the continued deposition of dentin. The crowns are generally short and bulbous in appearance, while the roots are narrow. The enamel can chip away within 2-4 years following eruption. This exposes the dentin underneath, which is soft and wears rapidly.
Note: These effects can be seen in both the deciduous and permanent dentition.
See picture #4 in booklet.

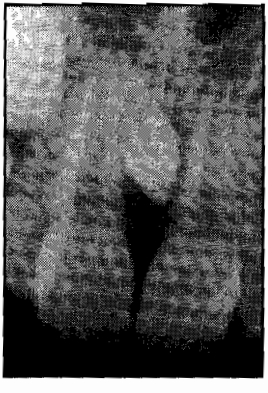
Three types of **dentinogenesis imperfecta:**

- **Type I** → dentin abnormality occurs in patients that have **osteogenesis imperfecta** *(characterized by blue sclera or a history of bone fractures).*
- **Type II** → most common, only the dentin abnormality exists with no bone involvement.
- **Type III** *(Brandywine Type)* → like Type II, only the dentin abnormality exists **however** there are clinical and radiographic variations in this type. They include multiple pulp exposures in the deciduous dentition.

Important: Clinically, dentinogenesis imperfecta is usually easily detected and identified. The teeth exhibit a **translucent or opalescent appearance. See picture #3 in booklet.** Another clinical feature is the abnormal constriction at the enamel-cementum junction that is detected by exploration. **Note:** The enamel in these teeth is structurally and chemically normal.

The permanent maxillary centrals in the x-ray below are **vital.** What is the most probable interpretation of the condition illustrated?

- Mesiodens
- Concrescence
- Fusion
- Dens in Dente



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- **Mesiodens**

Mesiodens is the **most common supernumerary** tooth, appearing singly or in pairs as a small tooth with a cone-shaped crown and a short root between the maxillary central incisors; it may be erupted, impacted or even inverted.

Mesiodens appear situated in the maxilla near the midline and almost always posterior to the normal central incisors. Many of them, therefore, are bypassed by the permanent incisors which are permitted to erupt into their normal position in the arch.

Remember:

- **Fusion** → is a developmental union of two or more teeth in which the dentin and one other dental tissue are united *(may be the root).* **See picture #6 in booklet.**
- **Concrescence** → is a condition in which only the cementum of two or more teeth becomes united. **See picture #7 in booklet.**
- **Dens in dente** *(also called dens invaginatus)* → means **"tooth within a tooth,"** it is caused by a deep invagination of the enamel organ during formation, most likely found associated with a maxillary lateral incisor. **See picture #5 in booklet.**

Hypercementosis is the excessive deposition of secondary cementum on the roots. Any tooth may be involved, however, which teeth are **most frequently** involved?

- Molars
- Incisors
- Premolars
- Canines

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• **Premolars**

Hypercementosis is often confined to the apical half of the root but, in some instances, may involve the entire root. In large majority of instances, it affects **vital** teeth, is not associated with any one particular systemic disease and may be regarded as a dental anomaly. It may be seen when a tooth has lost its antagonist or when there is chronic inflammation of the tooth. The **premolars** are most frequently involved. Next in frequency are the first and second molars.

Hypercementosis produces no **significant clinical signs or symptoms** indicative of its presence. It is seen radiographically as a bulbous enlargement that has surrounding it a **continuous and unbroken periodontal membrane space** and a normal lamina dura.

There is a form of hypercementosis which is a common feature in **Paget's disease** that involves the jaws. On the x-ray in this case, there is **complete absence** of the periodontal membrane space and lamina dura surrounding the hyperplastic cementum. **Note:** In addition to Paget's disease, hypercementosis is associated with supraeruption, apical periodontal infection, occlusal trauma, toxic thyroid goiter, acromegaly, and pituitary gigantism.

Hypercementosis: Maxillary premolars

An enamel defect resulting from the **incomplete formation** of the enamel matrix is called:

- Enamel pearls
- Enamel hypocalcification
- Enamel hypoplasia
- Dentinal dysplasia

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• **Enamel hypoplasia**

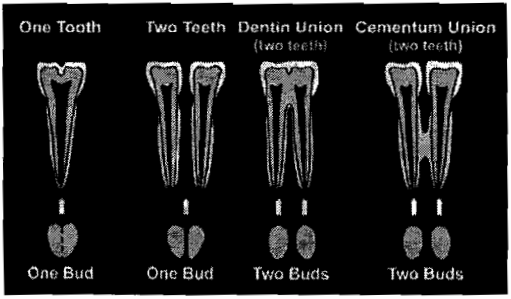
Enamel hypoplasia is a developmental defect in which the enamel of the teeth is hard in context but thin and deficient in amount. It results from **incomplete formation of the enamel matrix with a deficiency** in the cementing substance. Enamel hypoplasia affects both the deciduous and permanent teeth. It is usually due to illness or injury during tooth formation or due to a genetic disorder. **Note:** The genetic forms of enamel hypoplasia are generally considered to be types of amelogenesis imperfecta.

The clinical appearance of enamel hypoplasia includes: **1)** the lack of contact between teeth, **2)** the rapid breakdown of occlusal surfaces, **3)** a yellowish-brown stain that appears where the dentin is exposed. **Note:** If only one permanent tooth is affected, it is usually caused by physical damage to the primary tooth that this permanent tooth replaced.

Remember: Enamel **hypocalcification** is a hereditary dental defect in which the enamel is soft and undercalcified in context yet **normal in quantity.** It is caused by the defective maturation of ameloblasts *(there is a defect in the* **mineralization** *of the formed matrix).* The teeth are chalky in consistency, the surfaces wear down rapidly and a yellow to brown stain appears as the underlying dentin is exposed. This condition affects both the deciduous and permanent teeth as well. **See picture #9 in booklet.**

Match the following alterations in tooth morphology on the left with the correct diagram depicting the alteration on the right.

- Fusion
- Twinning
- Concresence
- Gemination



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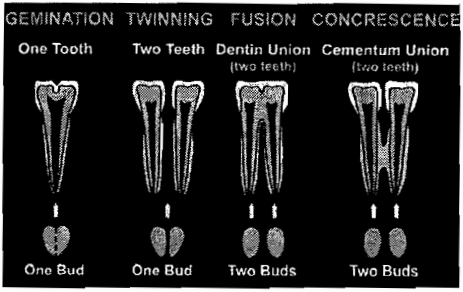

Remember:

- **Gemination** is a division of a single tooth germ by invagination. This results in the incomplete formation of two teeth. Frequently occurs in the incisor region.
- **Fusion** is an abnormally shaped tooth that may appear as an extra wide crown, a normal crown with an extra root, or other combinations resulting from the union of two adjacent tooth germs by dentin during development.
- **Concresence** is the union of the roots of two or more normal teeth caused by confluence of their cemental surfaces.
- **Twinning** is the complete division of a single tooth bud. The divided teeth are seen as completely separate with no connection to each other except each tends to be mirror image of the other.
- **Ankylosis** is the fusion of the cementum or dentin to the surrounding alveolar bone after loss of the intervening periodontal membrane. It is associated with hypodontia. The most commonly ankylosed tooth is the primary second molar; the permanent second premolar is the tooth that fails to develop and erupt. **Clinically,** percussion of the ankylosed tooth produces a dull sound. **Note:** There is a change in the continuity of the occlusal plane which is caused by the continued eruption of the non-ankylosed teeth and the growth of the alveolar process.

All of the following statements concerning **amelogenesis imperfecta** are true **except:**

- It is an inherited condition which is transmitted as a dominant trait
- Because of the enamel malformation, the teeth of individuals with amelogenesis imperfecta are often discolored, sensitive to temperature changes, and painful to brush
- It only affects the permanent teeth
- It causes the enamel of the teeth to be soft and thin
- The teeth appear yellow, because the dentin is visible through the thin enamel
- The teeth are easily damaged and susceptible to decay

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• **It only affects the permanent teeth**

***This is **false;** amelogenesis imperfecta **affects all teeth,** both deciduous and permanent.

Amelogenesis imperfecta is a hereditary **ectodermal** defect, unlike dentinogenesis imperfecta which is a hereditary mesodermal defect. There are three types of amelogenesis imperfecta: hypoplastic, hypocalcified and hypomaturation.

The crowns of the teeth may or may not show discoloration. If present, it varies depending upon the type of the disorder, ranging from **yellow to dark brown.** Contact points between teeth are often open and occlusal surfaces and incisal edges frequently are severely abraded.

The radiographic findings are frequently distinctive and **pathognomonic.** When the enamel is totally absent, the radiographic appearance makes the diagnosis obvious. When some enamel is present, thin radiopaque coverings on the proximal surfaces of the teeth are noted. When the anatomic crown forms are normal or nearly normal, the softness of the defective enamel may not be easily distinguished from the dentin. **In all cases,** however, the dentin, pulp and cementum are unaffected by the disease process itself *(unlike dentinogenesis imperfecta).* **See picture #8 and #10 in booklet.**

Exception: Amelogenesis imperfecta will only show pulp obliteration if there is advanced abrasion with secondary dentin formation.

Loss of tooth surface at the cervical areas of teeth caused by tensile and compressive forces during tooth flexure is referred to as:

- Erosion
- Abfraction
- Attrition
- Abrasion

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• **Abfraction**

Abfraction lesions are cervical erosive lesions that can not be attributed to any particular cause; causing the enamel to "pop" off starting at the base of the tooth and exposing the gum line of the tooth to excessive wear. **See picture #11 in booklet.**

Remember:

• **Abrasion** is the abnormal loss of tooth structure due to non-masticatory physical friction. **See picture #13 in booklet.**

<u>Types of abrasion:</u>

1. **Toothbrush** abrasion → most often results in V-shaped wedges at the cervical margin in the **canine and premolar areas.** It is caused by the use of a hard toothbrush and/or a horizontal brushing stroke and/or a gritty dentifrice.
2. **Occlusal** abrasion → results in flattened cusps on all posterior teeth and worn incisal edges. It results from the chewing or biting of hard foods or objects and chewing tobacco.

- **Attrition** is the wearing away of enamel and dentin due to the **normal function** or most commonly, due to the excessive grinding or gritting together of teeth by the patient *(referred to as* **bruxism***)*. The most noticeable effects of attrition are **polished facets,** flat incisal edges, discolored surfaces of the teeth and exposed dentin. Facets usually develop on the **linguoincisal** of the maxillary central incisors, the **facioincisal** of the mandibular canines and the **linguoincisal** of the maxillary canines. **See picture #14 in booklet.**
- **Erosion** is the loss of tooth structure from **non-mechanical means.** It can result from drinking acidic liquids or eating acidic foods. It is common in **bulimic** individuals as a result of regurgitated stomach acids. It affects smooth and occlusal surfaces. **See picture #12 in booklet.**

All of the following can cause **intrinsic staining** of teeth **except:**

- Dentinogenesis imperfecta
- Erythroblastosis fetalis
- Porphyria
- Fluorosis
- Diabetes mellitus
- Pulpal injury
- Internal resorption
- Tetracyclines

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- **Diabetes mellitus**

Cause	Intrinsic stain
Dentinogenesis imperfecta	Translucent or opalescent hue, usually gray to bluish-brown
Erythroblastosis fetalis	Bluish-black, greenish-blue, tan or brown
Porphyria	Red or brownish
Fluorosis	White opacities or light brown to brownish-black
Pulpal injury	Starts pink and usually becomes orange-brown to bluish-black
Internal resorption	Pinkish
Tetracycline	May vary from light gray, yellow or tan to darker shades of gray

Extrinsic stains can be caused by bacteria, iron, tobacco, foods, beverages, gingival hemorrhage, restorative materials and medications.

Treatment:
- Most extrinsic stains can be removed with abrasives; reduce or eliminate cause of stain
- Intrinsic stains may need bleaching, esthetic restorations, or prosthetic rehabilitation

What is the **most probable diagnosis** of the x-ray below?

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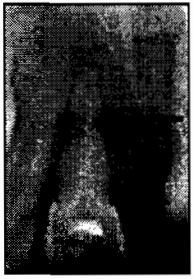
• **Internal resorption**

Internal resorption is an unusual form of tooth resorption that begins centrally within the tooth. Resorption of the dentin of the pulpal walls may be seen as part of an inflammatory response to pulpal injury, or it may be seen in cases in which no apparent trigger can be identified. Most cases of internal resorption present no early clinical symptoms. The **first evidence of the lesion** may be the appearance of a pink-hued area on the crown of the tooth, which represents the hyperplastic, vascular pulp tissue filling the resorbed areas. If the condition is discovered before perforation of the crown or root has occurred, **endodontic therapy** may be carried out with the expectation of a pretty high success rate.

Idiopathic external resorptive lesions are characterized by the invasion of the cervical region of the root by fibrovascular tissue which progressively resorbs dentin, enamel and cementum. The source of the resorption is in the attachment apparatus. This is the key to understanding diagnosis and treatment. The dental pulp remains protected by an intact layer of dentin and predentin until late in the process. Since the source of the lesion is not in the pulp, endodontic treatment by itself is of no assistance in dealing with the source of the problem.



Arrow points to external root resorption of mesial root of maxillary left central incisor

Which type of **amelogenesis imperfecta** is characterized by having enamel so soft that it can be removed during a prophylaxis?

- Type I *(Hypoplastic Amelogenesis Imperfecta)*
- Type II *(Hypocalcified Amelogenesis Imperfecta)*
- Type III *(Hypomaturation Amelogenesis Imperfecta)*

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• **Type II** ***(Hypocalcified Amelogenesis Imperfecta)***

There are three types of **amelogenesis imperfecta:**

1. **Type I** *(Hypoplastic Amelogenesis Imperfecta)* → the enamel **has not formed** to full-normal thickness *(or in some cases may be completely absent)* on newly erupted developing teeth. It results from the **defective formation of the enamel matrix.**
   See picture #15 in booklet.
2. **Type II** *(Hypocalcified Amelogenesis Imperfecta)* → the **quantity of enamel is normal** but the enamel is so soft that it can be removed during a prophylaxis. It results from the **defective mineralization of the enamel matrix.**
   See picture #16 in booklet.
3. **Type III** *(Hypomaturation Amelogenesis Imperfecta)* → the enamel can be pierced by an explorer tip under firm pressure and can be chipped away from the normal appearing dentin. It is characterized by **immature crystallites.**
   See picture #17 in booklet.

Remember:

1. Amelogenesis imperfecta is a hereditary **ectodermal** defect, unlike dentinogenesis imperfecta which is a hereditary **mesodermal** defect.
2. In all three types of amelogenesis imperfecta, the dentin, pulp and cementum are **unaffected** by the disease process itself *(unlike dentinogenesis imperfecta).*

Dentin dysplasia is a hereditary defect in dentin formation in which the coronal dentin and tooth color is _____; the root dentin is _____with a gnarled pattern and associated _____roots.

- abnormal, normal, shortened and tapered
- normal, normal, lengthened and broad
- normal, abnormal, shortened and tapered
- abnormal, abnormal, lengthened and broad

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- **Normal, abnormal, shortened and tapered**

There are two types of dentin dysplasia *(which is also called* **rootless teeth***)*. Type I and Type II. This appears to be a hereditary disease, transmitted as an autosomal dominant characteristic. This condition has not been associated with any systemic connective tissue disorder.

Clinical features:

- Normal eruption pattern
- Bluish in cervical region
- **Two types:**
  - Type I **radicular dysplasia** → more common type
    - Normal crowns of regular or slightly amber translucency
    - Tendency toward complete obliteration of pulp cavities
    - Abnormal spaces between the teeth, malalignment, malposition and severe mobility
  - Type II **coronal dysplasia**
    - Semi-transparent opalescent primary teeth
    - Normal appearance in the permanent teeth
    - Incomplete obliteration of pulp cavities
    - Pulp stones

Radiographic Features:

- Type I **radicular dysplasia**
  - Extremely short roots
  - Obliterated pulp chambers and root canals before eruption
  - Periapical radiolucencies around the defective roots
- Type II **coronal dysplasia**
  - Complete obliteration of pulp chambers and root canals after eruption
  - Pulp stones
  - Absence of periapical radiolucencies

Leukemia is a group of bone marrow diseases involving an **uncontrolled increase** in:

- Red blood cells *(Erythocytes)*
- Platelets
- Plasma cells
- White blood cells *(Leukocytes)*

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• **White blood cells *(Leukocytes)***

Leukemia is a form of cancer that begins in the blood-forming cells of the **bone marrow** *(the soft, inner part of the bones)*. Under normal circumstances, the blood-forming, or hematopoietic, cells of the bone marrow make leukocytes to defend the body against infectious organisms such as viruses and bacteria. But if some leukocytes are damaged and remain in an immature form, they become poor infection fighters that multiply excessively and do not die off as they should.

The leukemic cells accumulate and lessen the production of oxygen-carrying red blood cells *(erythrocytes)*, blood-clotting cells *(platelets)*, and normal leukocytes. If untreated, the surplus leukemic cells **overwhelm the bone marrow,** enter the bloodstream, and eventually invade other parts of the body, such as the lymph nodes, spleen, liver, and central nervous system *(brain, spinal cord)*. In this way, the behavior of leukemia is different than that of other cancers, which usually begin in major organs and ultimately spread to the bone marrow.

There are more than a dozen varieties of leukemia, but the following **four types** are the most common:

1. Acute lymphocytic leukemia *(ALL)* → **most common type in children**
2. Acute myelogenous leukemia *(AML)* → **most malignant** type
3. Chronic lymphocytic leukemia *(CLL)* → **least malignant** type
4. Chronic myelogenous leukemia *(CML)* → 2 distinct phases, invariably fatal

Important:

• Leukemia is classified by the **dominant cell type** and by the **duration from onset to death.**
• Leukemia **can modify** the inflammatory reaction.

All of the following are clinical features of aplastic anemia **except:**

- It results from the production of abnormal hemoglobin *(Hemoglobin A)*
- Unexplained infections, unexpected bleeding and fatigue
- Possible retinal and cerebral hemorrhages
- Hemorrhage, oral mucosal petechiae, purpura, and ecchymoses *(related to thrombocytopenia)*
- Oral mucosa may appear pale due to decreased red blood cell count
- Oral ulcerations, especially in gingival tissues

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• **It results from the production of abnormal hemoglobin *(Hemoglobin A)***

***This is **false;** the etiology is unknown, however, it has been linked to exposure of chemicals and radiation

Aplastic anemia is a form of anemia in which the capacity of the bone marrow to generate red blood cells is defective. **Two types:**

1. **Primary** → unknown cause, affects young adults. The signs and symptoms include pallor, weakness, malaise, dyspnea *(difficulty breathing)*, headache and vertigo. Oral symptoms include spontaneous bleeding, bruising *(petechiae)* and gingival infections. **It is usually fatal.**
2. **Secondary** → caused by exposure to toxic agents, such as radiation, chemicals or drugs *(for example,* **chloramphenicol***)*. It can occur at any age. Symptoms are the same as primary. Prognosis is good once you remove the cause.

***Aplastic anemia is the **most serious** and **life-threatening blood dyscrasia** associated with drug toxicity.

Plummer-Vinson syndrome characterized by an iron-deficiency anemia, atrophic changes in the buccal, glossopharyngeal, and esophageal mucous membranes, koilonycha *(spoon-shaped finger nails)*, and dysphagia. The dysphagia is due to an esophageal stricture or web. Carcinoma of the tongue and postcricoid region are complications. It is most common in middle aged women, rarely in the male. The etiology unknown. Because of the predisposition to the development of carcinoma of the oral mucous membranes, it is essential that the diagnosis be established early so that treatment can be given ASAP. This includes administration of iron, vitamin B complex and a high protein diet.

One of the most common inherited disorders among African Americans is:

- Cystic fibrosis
- Muscular dystrophy
- Polio
- Sickle-cell anemia

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• **Sickle-cell anemia**

Sickle-cell anemia is an inherited disease in which the red blood cells, normally disc-shaped, become **crescent shaped.** As a result, they function abnormally and cause small blood clots. These clots give rise to recurrent painful episodes called "sickle cell pain crises." Sickle-cell anemia *(also called sickle-cell disease)* is the result of the production of **abnormal hemoglobin** *(Hemoglobin S)* due to a genetic defect. It is carried as a trait by 10% of African Americans and 0.2% have sickle-cell anemia. It is more common in females and usually clinically manifests itself before the age of 30. The typical signs of anemia are present. The patient is weak, short of breath and easily fatigued. Muscle and joint pains are common.

Dental radiographs are often of diagnostic value → marrow spaces are markedly enlarged because of the loss of many trabeculae; the trabeculae, which are present, are often abnormally prominent. Occasionally, osteosclerotic areas are noted in the midst of large radiolucent marrow spaces. **However,** the lamina dura and the teeth are unaffected. **See picture #18 in booklet.**

Note: The gene defect is a known mutation of a single nucleotide *(thymine for an adenine)* of the beta-globin gene, which results in glutamic acid to be substituted by valine. Sickle-cell anemia occurs when a person inherits two abnormal genes *(one from each parent)*. If a person inherits one abnormal gene for the disease, they have what is called **sickle cell trait.** The life span of red blood cells is reduced from 120 to 20 days.

When purpura spots are **very small**, they are called:

- Pimples
- Petechiae
- Ecchymoses
- Varicose veins

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• **Petechiae**

***Large purpura spots are called **ecchymoses.**

Purpura spots are purplish discolorations in the skin produced by small bleeding vessels near the surface of the skin. Purpura may also occur in the mucous membranes *(such as the lining of the mouth)* and in the internal organs. Purpura by itself is only a sign of other underlying causes of bleeding. Purpura may occur with either normal platelet counts *(non-thrombocytopenic purpuras)* or decreased platelet counts *(thrombocytopenic purpuras)*. Platelets help maintain the integrity of the capillary lining and are important in the clotting process.

Major kinds of purpura:

- **Thrombocytopenic purpura** *(Werlhof's disease)* → a bleeding disorder characterized by a deficiency in the number of platelets. This results in multiple bruises, petechiae, and hemorrhage into the tissues.
- **Thrombotic thrombocytopenic purpura** *(TTP)* → a severe and frequently fatal form characterized by a low platelet count in the blood due to consumption of platelets by thrombosis in the terminal arterioles and capillaries of many organs.

Oral manifestations of thrombocytopenic purpura:

- Severe and profuse **gingival hemorrhage**
- Petechiae occur commonly on the palate

Important: Tooth extractions are **contraindicated** due to the tendency for excessive bleeding.

All of the following statements are true **except:**

- Acute leukemias have a slow onset and progression
- Acute leukemias are characterized by the appearance of immature, abnormal cells in the bone marrow and peripheral blood and frequently in the liver, spleen, lymph nodes, and other parenchymatous organs.
- The clinical picture of acute leukemias are marked by the effects of anemia, which is usually severe *(fatigue, malaise)*, an absence of functioning granulocytes *(proneness to infection and inflammation)*, and thrombocytopenia *(hemorrhagic diathesis)*.
- The spleen and liver usually are moderately enlarged, while enlarged lymph nodes are seen mainly in acute lymphocytic leukemia. Fever and a very high ESR are found.
- Leukocyte counts vary greatly in the acute leukemias

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• **Acute leukemias have a slow onset and progression**

***This is **false;** acute leukemias have a **rapid onset and progression.**

Acute leukemia is characterized by malignant proliferation of white blood cell precursors *(blasts)* in bone marrow and lymph tissue and their accumulation in peripheral blood, bone marrow, and body tissues. Leukemic cells inhibit normal bone marrow production of erythrocytes, platelets, and immune function.

Other important features of acute leukemia:

- **Abrupt** onset *(few months)* with sudden high fever, weakness, malaise, severe anemia, and generalized lymphadenopathy; bone and joint pain common in children.
- **Principal organ** involved → **bone marrow** *(along with the spleen and liver)*
- **Petechiae** and **ecchymoses** in skin and mucous membranes, hemorrhage from various sites; bacterial infections common.
- Laboratory findings → leukocytosis **30,000-1000,000** per cu.mm. with **immature** forms *(myeloblasts and lymphoblasts)* predominating.
- In 75% of the cases of acute lymphocytic leukemia, the **lymphocytes** are neither B nor T-cells and are called **"null cells"**.
- Untreated patients **die within six months**; with intensive therapy *(chemotherapy, radiation, and bone marrow transplants)* remissions lasting up to five years may be obtained; death is usually due to a hemorrhage *(brain)* or a superimposed bacterial infection.

Agranulocytosis is a marked decrease in circulating granulocytes, particularly neutrophils, attributable to a variety of causes. However, which of the following etiologic agents is / are involved in more than half of the cases?

- Radiation
- Bacterial infection
- Antimetabolic, antibiotic, and cytotoxic drugs
- Smoking

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- **Antimetabolic, antibiotic, and cytotoxic drugs** → this disease is most commonly caused by **ingestion of a drug.**

Agranulocytosis is an abnormal condition of the blood, characterized by a **severe reduction in the number of granulocytes** *(particularly neutrophils)*. **Note:** It may also be caused by the **antithyroid drugs** *(i.e., propylthiouracil, methimazole, and carbimazole)*.

Clinical features:

- Sudden onset of high fever, chills, jaundice, weakness and sore throat
- Oral infection with rapid periodontal destruction
- Oral ulcers and gingival bleeding

The **most characteristic feature** of this condition is the presence of **infection,** particularly in the **oral cavity.** The signs and symptoms develop very rapidly, usually within a few days, and death may occur soon afterward.

The oral lesions are an important phase of the clinical aspects of agranulocytosis. They appear as necrotizing ulcerations of the oral mucosa, particularly the gingiva and palate. These lesions appear as ragged necrotic ulcers covered by a gray membrane. One important aspect is that there **is little or no apparent inflammatory cell** infiltration around the lesions. Histologically, this is pathognomonic of agranulocytosis.

Note: Cyclic neutropenia is an unusual form of agranulocytosis. These patients typically exhibit **severe gingivitis.** The severe ulcerations usually seen in agranulocytosis usually do not occur. **See picture #19 in booklet.**

A form of leukemia characterized by abnormal cells that are very poorly differentiated but that are considered to be precursors of lymphoblasts, myeloblasts, or monoblasts is called:

- Aleukemic leukemia
- Subleukemic leukemia
- Stem cell leukemia

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- **Stem cell leukemia**

***The cells are **too immature** to classify.

Other leukemia terms to know:

- **"Aleukemic"** leukemia is a term used to describe a form of leukemia in which there are leukemic cells present in the bone marrow, but the circulating white blood cells are **neither immature nor increased in number.**

- **"Subleukemic"** leukemia is a term used to describe a form of leukemia in which leukemic cells appear in the blood but there is **no significant increase** in the number of circulating white blood cells.

- A **"leukemoid"** reaction is a term used to describe a marked increase in the number of circulating granulocytes. This condition is seen in a variety of disorders including chronic infections and neoplasms.

A dentist is often consulted first by a patient with pernicious anemia for relief of:

- Denuded gingiva
- Glossitis
- Edematous buccal mucosa
- Severe gingivitis

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• **Glossitis**

Pernicious anemia is a relatively common, chronic, progressive, **megaloblastic anemia.** It is caused by the lack of secretion of the **intrinsic factor** in normal gastric juice. This factor is necessary for adequate absorption of vitamin B_{12}, which is necessary for the **maturation of erythrocytes.** As a result, they produce fewer erythrocytes than normal. The onset of pernicious anemia usually is insidious and vague. As the condition progresses, there will be a sore, painful tongue *(atrophic glossitis)*, angular cheilitis, a tingling numbness of the extremities, difficulty swallowing *(dysphagia)*, painful swallowing *(odynophagia)*. **See picture #20 in booklet.**

A **Schilling 24-hour urine test** is done to evaluate whether vitamin B_{12} is being absorbed by the body and is most commonly used to evaluate patients for pernicious anemia.

Remember: Thalassemia major and minor are **hemolytic anemias** that result from a genetic defect. Both are characterized by a **low level of erythrocytes** and abnormal hemoglobin.

Oral manifestations of thalassemia:

- Oral mucosa may exhibit the characteristic **anemic pallor**
- **Flaring of the maxillary anterior teeth** with malocclusion

In many cases, **severe hemolytic disease** results when the:

- Fetus has Rh-negative blood and the mother has Rh-negative blood
- Fetus has Rh-positive blood and the mother has Rh-positive blood
- Fetus has Rh-positive blood and the mother has Rh-negative blood

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• **Fetus has Rh-positive blood and the mother has Rh-negative blood**

The fetus' blood is Rh-positive because the father passed along an Rh-positive trait, which is a dominant trait. The mother responds to the incompatible blood by producing antibodies against it. These antibodies cross the placenta into the fetus' circulation, where they attach to and destroy the fetus' red blood cells, leading to anemia → this is called **erythroblastosis fetalis.**
Note: It can also result from blood type incompatibilities. For example, the mother may have type O blood and the fetus has type A or B blood.

The most common form of erythroblastosis fetalis is called is **ABO incompatibility,** which can vary in its severity. The less common form is called **Rh incompatibility,** which more often causes a very severe anemia in the baby. The severity of this condition can vary widely. In some instances, the baby has no symptoms of the disease. In other cases, it can lead to death of the baby before or shortly after birth. It can be treated in utero by intrauterine transfusion.

When the child is born, signs may include an enlarged liver or spleen, generalized edema, jaundice, and anemia. After birth, depending on the severity, a transfusion usually needs to be performed. The most severe form of this disease, Rh incompatibility, can be prevented if the mother takes a medicine called Rhogam at certain times during and after pregnancy.

Oral manifestations of erythroblastosis fetalis:
- **Teeth** appear to have a green, blue or brown hue due to the deposition of blood pigment in the enamel and dentin.
- **Enamel hypoplasia** may occur. If it does, it affects the incisal edges of the anterior teeth and the middle portion of the deciduous cuspid and the first molar crown.

Which statement is **true** concerning **chronic** leukemias?

- They have a rapid onset and progression
- They have a shorter, more devastating clinical course than the acute leukemias
- They are characterized by proliferations of lymphoid or hematopoietic cells that are more mature than those of the acute leukemias
- They constitute 75% of all leukemias

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- **They are characterized by proliferations of lymphoid or hematopoietic cells that are more mature than those of the acute leukemias**

Important: Chronic leukemias have a **slower onset and progression.** They also have a **longer, less devastating** clinical course than the acute leukemias, and they constitute about **50%** of all leukemias.

Other important clinical features of chronic leukemias:

- **Insidious onset** with weakness and weight loss → disease may be detected during examination for some other condition, e.g., anemia, unexplained hemorrhages, or recurrent intractable infection.
- Organ involvement **similar to** acute type → massive splenomegaly is characteristic of chronic myelogenous leukemia; lymph node enlargement is main pathologic finding in lymphocytic type.
- **Petechiae and ecchymoses,** recurrent hemorrhages, bacterial infections → lymphocytic anemia may be complicated by autoimmune hemolytic anemia.
- **Laboratory findings** → leukocytosis **above 100,000** per cu. mm. with **mature** forms *(granulocytes and lymphocytes)* predominating; **Philadelphia chromosome** and low levels of leukocyte alkaline phosphatase are common in **chronic myeloid leukemia** *(CML)*.
- Median survival time for patients with chronic myelogenous leukemia *(CML)* is four years with death due to hemorrhage or infection; chronic lymphocytic leukemia *(CLL)* runs a variable course; older patients may survive years even without treatment.

Leukemias are evenly split between the acute and chronic forms, but **among children** one form accounts for about two-thirds of cases. This **one form** is:

- Acute lymphocytic leukemia
- Acute myeloid leukemia
- Chronic lymphocytic leukemia
- Acute monoblastic

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• **Acute lymphocytic leukemia** *(ALL)*

The peak age for ALL is around four years old, and it is the form of acute leukemia that is the **most responsive** to therapy. Current therapies for ALL include chemotherapy with follow-up radiation, and possible bone-marrow transplant after particularly high dose chemotherapy treatment or in cases of recurrence or if non-responsive to other treatments. **See picture #22 in booklet.**

***Acute myeloid leukemia *(AML)* and chronic lymphocytic leukemia *(CLL)* are the most common types in **adults.**

Although the exact cause of most leukemias remains unknown, increasing evidence suggests a combination of contributing factors. These factors include: familial tendency, congenital disorders (Down syndrome, or the presence of **Philadelphia chromosome → chronic myeloid leukemia**), viruses (herpes-like viral particles have been cultured from patients and leukemic patients have high antibody titer to the **Epstein-Barr virus**), ionizing radiation and the exposure to the chemical benzene and cytotoxins such as alkylating agents.

Important: Oral lesions are most likely to be observed in **myelogenous leukemia.** These oral lesions may be the initial manifestation of the disease. The oral lesions include gingivitis, gingival hemorrhage, generalized gingival hyperplasia, petechiae, ecchymoses, and ulcerations. **See picture #21 in booklet.**

All of the following statements concerning **polycythemia vera** are true **except:**

- It is characterized by excessive erythrocyte production
- It usually occurs within the age range of 10-35, with 20 being the mean age of onset
- Polycythemia vera is considered to be one of the chronic myeloproliferative disorders
- Clinical features include headaches, weakness, weight loss, and pruritus
- Hemorrhage and thrombosis may be evident at any time

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• **It usually occurs within the age range of 10-35, with 20 being the mean age of onset**

***This is **false;** polycythemia vera usually occurs within the age range of **20-80**, with 60 being the mean age of onset.

Polycythemia is the condition of **too many red blood cells** in the circulation. The blood can be too thick to pass easily through the small blood vessels of the body. This in turn leads to clot formation and blockage of the small vessels which can lead to a stroke.

There are **two types** of polycythemia:

1. **Primary polycythemia** *(also called* **polycythemia vera** *or erythemia)* occurs when excess erythrocytes are produced as a result of tumorous abnormalities. This occurs in the tissues that produce blood cells. Usually accompanied by leukocytosis. **Splenomegaly**, as a result of vascular congestion, is seen in 75% of patients.
2. **Secondary polycythemia** is an increase in the total number of erythrocytes due to another condition. For example, chronic tissue hypoxia of advanced pulmonary disease, high altitude *(Osker's disease)* or the secretion of erythropoietins by certain tumors.

Oral manifestations of polycythemia:

- Oral mucous membranes *(especially the gingiva and tongue)* appear deep purplish-red.
- The **gingiva** are very **swollen and bleed** very easily.
- Submucosal petechiae *(purplish spots),* ecchymoses *(same as petechiae, but bigger)* and hematomas are common.

Note: Erythromelalgia is a rare syndrome of paroxysmal vasodilation with burning pain, increased skin temperature, and redness of the feet and, less often, the hands.

A specific **chromosome marker** characterizes:

- Acute lymphocytic leukemia *(ALL)*
- Chronic myeloid leukemia *(CML)*
- Acute myeloid leukemia *(AML)*
- Chronic lymphocytic leukemia *(CLL)*

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• **Chronic myeloid leukemia *(CML)***

Almost 90% of patients with **chronic myeloid** *(myelocytic, myelogenous, granulocytic)* **leukemia** have the **philadelphia chromosome,** an abnormality in which the long arm of chromosome 22 is translocated, usually to chromosome 9. Radiation and carcinogenic chemicals may induce this chromosomal abnormality.

CML is characterized by the abnormal overgrowth of granulocytic precursors *(myeloblasts and promyelocytes)* in bone marrow, peripheral blood and body tissues. CML is most common in young and middle-aged adults and is slightly more common in men than in women; it is rare in children.

The typical symptoms of CML include:

- **spongy bleeding gums**
- fatigue
- fever
- weight loss
- moderate splenomegaly
- joint and bone pain
- **repeated infections**

Note: Acute myeloid leukemia *(AML)* is a malignant disease of the bone marrow in which hematopoietic precursors are arrested in an early stage of development. AML is distinguished from other related blood disorders by the presence of greater than 30% blasts in the blood and/or bone marrow. These blasts *(myeloblasts)* contain **Auer rods** in their cytoplasm.

The **traumatic neuroma** results from a hyperplastic response to nerve damage after severance of a large nerve fiber. In the mouth, the traumatic neuroma is **most commonly** commonly seen:

- In the mandibular mucobuccal fold in the region adjacent to the mental foramen in edentulous mouths
- On the maxillary tuberosity
- On the palate
- On the interdental papillae

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- **In the mandibular mucobuccal fold in the region adjacent to the mental foramen edentulous mouths**

A **traumatic neuroma** is a lesion caused by trauma to the peripheral nerve. In the oral cavity, the injury may be in the form of trauma from a surgical procedure such as a tooth extraction, from a local anesthetic injection or from an accident. It is usually a very small nodule *(less than 0.5 cm in diameter).* It is firm, movable and well encapsulated. It is **painful when palpated.** Pressure applied to the neuroma elicits a response often described as an "electric shock." The treatment is surgical excision.

Histologic features:
- Abundant nerve tissue and collagenous fibrous tissue in haphazard arrangement
- Chronic inflammatory cell infiltrate may be present
- Schwann cells will be present

Remember: Multiple neuromas discovered on the lips, tongue or palate may indicate the possibility that the patient has **MEN III** *(multiple endocrine neoplasia syndrome)* — an autosomal dominant condition characterized by numerous mucosal neuromas, marfanoid habitus *(the features of the genetic condition Marfan's syndrome → includes abnormalities in the eyes, skeleton and cardiovascular system),* and endocrine neoplasms.

The most common site in the oral cavity for a **lymphangioma** is the:

- Palate
- Tonsils
- Floor of the mouth
- Tongue

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• **Tongue** → on the dorsal and lateral surface of the anterior portion

Lymphangiomas are benign hamartomas of lymphatic channels that develop early in life with no sex predilection. They may occur on the skin or mucous membrane. In addition to the tongue, they occur commonly on the lips and labial mucosa.

Clinical features:
- Raised, diffuse, bubbly nodules or vesicles
- Range in color from clear to pink, dark red, brown or black
- Asymptomatic
- Soft, fluctuant
- Varies in size
- Usually painless

Histologic features → **four types** of lymphangiomas:
- **Lymphangioma simplex** *(capillary lymphangioma)* – composed of small, thin-walled lymphatics.
- **Cavernous lymphangioma** – comprised of dilated lymphatic vessels with surrounding adventitia.
- **Cystic lymphangioma** *(cystic hygroma)* – consisting of huge, macroscopic lymphatic spaces with surrounding fibrovascular tissues and smooth muscle.
- **Benign lymphangioendothelioma** *(acquired progressive lymphangioma)* – lymphatic channels appear to be dissecting through dense collagenous bundles.

Important: Lymphangiomas **do not** undergo malignant change. Some lymphangiomas, especially congenital types, regress spontaneously during childhood. **Aspiration is mandatory** before surgical excision of a lymphangioma to prevent complications associated with the similar-appearing hemangioma.

A **"pregnancy tumor"** is not a tumor at all but merely a (an):

- Irritation fibroma
- Pyogenic granuloma
- Epulis granulomatosum
- Peripheral fibroma

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- **Pyogenic granuloma** → the soft, red lesion that sometimes occurs in pregnant women is merely a pyogenic granuloma

Entity	Cause	Location	Clinical	Microscopic	Treatment
Pyogenic granuloma	Minor trauma provides pathway for non-specific organisms, calculus	Gingiva most common, lips, buccal mucosa	Elevated mass, often ulcerated, bleeds easily, greater with females, may recur	Exuberant granulation tissue	Surgical excision, may recur
Pregnancy tumor	Possible secondary to altered endocrine state during pregnancy *(first trimester)*	Gingiva most common, lips, buccal mucosa	No justification for continued use of term as it is a pyogenic granuloma	Exuberant granulation tissue	Don't remove until after pregnancy
Epulis granulomatosum	Retained foreign material *(bone, tooth, fragment)* **"latrogenic"**	Post-extraction **socket**, almost always within 10 days of extraction	Soft, non-painful, bleeds easily	Granulation tissue within whieh one may find bone, dentin, cementum or foreign material	Curettage
Fibroma *(also called "Irritation Fibroma" or "Traumatic Fibroma")*	Reactive, most common tumor seen in oral cavity	Buccal mucosa lateral border of tongue and lower lip	Elevated, smooth pink, painless, well demarcated mass. same color or lighter than normal mucosa	Bundles of collagen interspersed with fibroblasts and small blood vessels	Conservative surgical excision

The **peripheral ossifying fibroma** presents as a:

- Raised, bluish, translucent, fluctuant lesion
- Painless, solid, firmly attached, gingival mass
- Raised pink-red, firm, mass arising around a denture flange
- Thickening and crusting of the lower lip

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• **Painless, solid, firmly attached, gingival mass**

The peripheral ossifying fibroma is a **benign reactive lesion** that occurs exclusively on the gingiva. Most authorities believe this entity arises from fibers of the periodontal ligament. The lesion usually presents clinically as a solid, firmly attached, gingival mass. The peripheral ossifying fibroma frequently causes **separation of the adjacent teeth,** and occasionally minimal osseous resorption can be observed subjacent to the lesion. The peripheral ossifying fibroma has been observed in individuals between the ages of 5 and 25 years, with a peak incidence at 13 years. Females are more often affected than males and the majority of the lesions occur anterior to the molar region. There is a slight predilection for the maxillary arch. **Important:** The lesion is treated by surgical excision. Because the peripheral ossifying fibroma probably arises from the periodontal ligament, excision should be deep, down to the periodontium and including the associated periodontal ligament. Thorough root scaling of the adjacent teeth should be performed.

The lesion is quite characteristic, **histologically,** in its high degree of cellularity *(usually exhibiting bone formation)* in contrast to the peripheral fibroma. Vascularity is not a prominent feature as it is in the pyogenic granuloma. **Note:** These lesions tend to recur, **as opposed to** the peripheral fibroma, which does not.

Remember: The **peripheral fibroma** also presents as well-demarcated focal mass of hyperplastic tissue with either a sessile or pedunculated base. It is similar in color to the surrounding connective tissue. It may be ulcerated. The treatment for a peripheral fibroma is local excision. **Recurrence is rare.**

Note: Other variant forms of the **peripheral fibroma** include:

- The **peripheral odontogenic fibroma:** which is gingival mass composed of a well-vascularized, nonencapsulated fibrous connective tissue.
- The **giant cell fibroma:** which is a fibrous hyperplasia composed of multi-nucleated connective tissue cells.

The acronym **MEN** is a group of genetically distinct familial diseases involving adenomatous hyperplasia and malignant tumor formation in several:

- Sebaceous glands
- Intestinal glands
- Endocrine glands
- Salivary glands

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• **Endocrine glands**

***These tumors occur in association with a variety of other pathologic features.

The **multiple endocrine neoplasia syndromes** *(also called* **MEN Syndrome***)* have been classified into three distinct syndromes, each is inherited as an autosomal dominant trait:

- **Men I** → consists of tumors or hyperplasia of the pituitary, parathyroids, adrenal cortex, and of the pancreatic islets.
- **Men II** *(also called* **Sipple's Syndrome** *and subtype A)* → is characterized by parathyroid hyperplasia or adenoma, but no tumors to the pancreas. **However,** in addition, these patients have pheochromocytomas of the adrenal medulla and medullary carcinoma of the thyroid gland.
- **Men III** *(also known as subtype IIB)* → is characterized by **mucocutaneous neuromas,** pheochromocytomas of the adrenal medulla and medullary carcinoma of the thyroid gland.

Important: The most constant feature of **Men III** is the presence of neuromas, particularly of the **oral cavity.** These are most common on the lips, tongue, and buccal mucosa.

Note: The most important aspect of this syndrome is the **medullary carcinoma of the thyroid** because of its ability to metastasize and cause death. Therefore, the detection of the mucosal neuromas may alert the clinician for early diagnosis and treatment.

Which of the following is probably the most common benign peripheral nerve tumor?

- Traumatic neuroma
- Neurilemoma *(Schwannoma)*
- Neurofibroma
- Lipofibroma

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- **Neurofibroma** → this is a tumor of the nerve fibers itself, it commonly appears as a sessile, firm, pink nodule

On occasion a solitary neurofibroma is seen in isolation, however, more frequently they are seen as a part of neurofibromatosis. **Neurofibromatosis** is a genetic disorder that may be associated with multiple neurofibromas. This disease is transmitted through an autosomal dominant mutant gene. There are two types of neurofibromatosis, type 1 and type 2. Type 1 *(von Recklinghausen's disease)* affects 1 in 3000 people; however, the majority of cases are mild with a limited number of tumors. Type 2, which is more severe, affects 1 in 50,000 people.

Tumor	Etiology	Clinical Character	Treatment and Prognosis
Traumatic Neuroma	Trauma to a peripheral nerve	Most common site over mental foramen in edentulous mouths; nodule or swelling, which may be painful to digital pressure	Excision with small proximal portion of involved nerve; recurrence uncommon
Neurilemoma *(Schwannoma)*	It is derived from proliferation of **Schwann cells** of the neurolemma that surrounds peripheral nerves	Encapsulated mass that presents as an asymptomatic lump. The **tongue** is the most common location. Bony lesions may cause pain or paresthesia.	Conservative excision; recurrence rare
Neurofibroma	Some investigators say it is derived from the Schwann cell;	**Two forms:** 1. **Solitary neurofibroma** — asymptomatic nodule, occurs on tongue, buccal mucosa and vestibule 2. **Multiple** lesions as part of the syndrome neurofibromatosis	1. **Solitary:** surgical excision 2. **Neurofibromatosis:** removal is impractical. Watch for high rate of malignant transformation
Palisaded Encapsulated Neuroma	Unknown	Sessile, smooth– surfaced nodule of less than 1 cm in diamcter. Predilection for the face, with nose and cheek most common	Local surgical excision: recurrence rare

Multiple Neurofibromatosis *(Von Recklinghausen's Disease)* is an autosomal dominant hereditary condition consisting of multiple neurofibromas of the skin and mucosa and associated _____with the potential for producing disfigurement and malignant transformation.

- Enlargement of the major salivary glands
- Café au lait spots of the skin
- Facial and trunkal acne
- Swelling of the facial muscles

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• **Café au lait spots of the skin**

Von Recklinghausen's disease is an autosomal dominant disease associated with loss of a tumor suppressor gene *(NF1 or NF2)*. It is characterized by multiple neurofibromas of the oral cavity and on the skin, and pigmentations of the skin *(Café au lait)*, iris *(Lisch nodules)*, and axilla *(axillary freckling or Crowe's sign)*. The presence of **six or more café au lait macules** greater than 1.5 cm in diameter is generally regarded as being **indicative** of this disease until proven otherwise.

Radiographic features:
- Well-demarcated unilocular or multilocular radiolucency
- Possible root disturbance
- Possible jaw enlargement

Treatment:
- Surgical excision but may be impractical due to the number of lesions.
- Best left untreated because multiple recurrences may be associated with malignant transformation to neurogenic sarcoma.

Note: The importance of the lesions is the high risk of **malignant transformation.**

Remember: The single neurofibroma presents at any age, it commonly appears as a sessile, firm, pink nodule that commonly occurs on the **tongue, buccal mucosa and vestibule.** It is removed by surgical excision and rarely recur.

The most frequently encountered intraoral benign neoplasm of **connective tissue origin** is:

- A papilloma
- A fibroma
- A lipoma
- A nodular melanoma

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• **A fibroma**

***Also called an **"irritation fibroma"** or a **"traumatic fibroma."**

Fibromas occur in people of all ages and with equal frequency in both sexes. They may arise from almost any soft tissue in the mouth, although they are found most commonly on the buccal mucosa, lateral border of the tongue and the lower lip. They usually present as pink, painless, smooth, elevated, well-demarcated masses. The history is often helpful in establishing a diagnosis, since, in most cases, the tumor is reported to have been present for months or years and to have a **slow-growing** behavior pattern. **See picture #23 in booklet.**

Note: Some investigators feel that **"true fibromas"** of the oral cavity are rare and that in reality, they are simply examples of localized hyperplasia, resulting from **long-standing irritation or trauma** *(hence the term* **"irritation fibroma"** *or* **"traumatic fibroma"***).* These fibrous nodules are comparable with the hyperplasia from denture irritation *(termed* **"epulis fissuratum"***).* The only difference between the **"true fibroma"** *(which is a true neoplasm)* and the **"irritation fibroma"** *(which is not a true neoplasm)* is that the hyperplastic tissue **may regress after removal** of the irritant, while the true fibroma will not regress. Either way, the treatment is conservative surgical excision.

Remember: A papilloma is the most common benign neoplasm of **epithelial tissue origin.**

Scleroderma is a systemic disease that affects many organ systems. The symptoms result from inflammation and progressive tissue fibrosis and occlusion of the microvasculature by excessive production and deposition of:

- Types II and IV collagens
- Types I and III collagens
- Elastin and reticulin
- All of the above

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• **Types I and III collagens**
***Other levels of macromolecules found in the connective tissue *(e.g., glycosaminoglycans, tenascin, fibronectin)* are also increased.

Scleroderma is an uncommon, chronic disease. It affects the connective tissues which surround the joints, blood vessels and internal organs beneath the affected area of skin. Women are affected three to four times more often than men. The disease usually starts between the ages of 25 and 50. It only occasionally begins in children or in the elderly. Although there is no cure, proper treatment and care can make it possible for people with scleroderma to lead full, productive lives.

Oral radiographs of a patient with scleroderma would show an abnormal widening of the periodontal ligament. This space is created by a thickening of the periodontal-membrane as a result of an increase in size and number of collagen fibers. The enlarged space is almost **uniform in width,** surrounds the entire root of the tooth and makes the tooth appear as if it is being extruded rapidly from its socket. **See picture #31 in booklet. Note:** Other oral radiographic features may include bilateral resorption of the angle of the ramus of the mandible or complete resorption of the condyles and/or coronoid process of the mandible. **Remember:** The abnormal widening of the periodontal-membrane space is also a radiographic finding in **osteosarcomas**.

<u>There are two major types of scleroderma:</u>

1. **Localized scleroderma:** In this condition changes only occur in isolated areas of the skin and the tissues beneath it. It is relatively mild and does not affect internal organs.
2. **Systemic scleroderma:** With this condition changes may occur in the skin and also in a number of internal organs. These might include blood vessels, joints, the digestive system *(esophagus, stomach and bowel)*, and occasionally the lungs, heart, kidneys and muscles. Changes in the connective tissue may affect the function of any of these organs.

The **congenital epulis of the newborn** is composed of cells that are identical to those of:

- A traumatic neuroma
- A schwannoma
- A granular cell myoblastoma
- A lipoma

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• **A granular cell myoblastoma** *(also called a granular cell tumor)*

The **congenital epulis of the newborn** *(also called congenital gingival granular cell)*. It usually occurs in the anterior maxilla and is 10 times more likely to occur in females than in males. The lesion is pink, soft, and compressible. The treatment is surgical excision with little possibility of recurrence. **See picture #24 in booklet.**

The **granular cell myoblastoma** is a rare neoplasm of unknown etiology. It presents as an uninflamed, asymptomatic mass. The most common location in the head and neck region is the **tongue**. It may affect any age group and females seem to be affected more than men. **See picture #25 in booklet.**

Important: Both of these lesions are identical histologically. They both contain **granular cells,** however, the congenital epulis of the newborn **does not exhibit** overlaying pseudoepitheliomatous hyperplasia. The pseudoepitheliomatous hyperplasia of the overlying epithelium is frequently seen in the **granular cell myoblastoma.**

A periapical abscess **usually** arises as a result of:

- Orthodontics
- Trauma
- Infection of the pulp of a tooth
- Periodontal disease

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• **Infection of the pulp of a tooth**

***This infection follows the **carious** involvement of the tooth. The cellular debris and/or infection which caused the tooth pulp to die, slowly filters out of the tip of the root and produces an inflammatory reaction around the root tip.

Note: A periapical abscess can also occur after traumatic injury to a tooth, which results in necrosis of the pulp, and in cases of irritation of the periapical tissues, either by mechanical manipulation or by the application of chemicals in endodontic procedures.

Clinical features:

- If **acute**, presents as an **abscess: See picture #26 in booklet.**
  - Tooth is extremely painful to percussion
  - May feel slightly extruded from its socket
  - Tooth will exhibit mobility
- If **chronic**, presents as a **granuloma or cyst.** There are usually **no clinical** features or symptoms.

Radiographic features:

- If **acute**, only a slight thickening of the periodontal membrane is noticeable
- If **chronic** *(granuloma or cyst)*, there will usually be a radiolucent area at the apex of the involved tooth. **See picture #27 in booklet.**

Treatment: Establish **drainage** either by opening the pulp chamber or extracting the tooth.
Note: If a periapical abscess is **not treated,** it can lead to serious complications such as osteomyelitis, cellulitis and bacteremia.

An 84-year-old woman has had no major medical problems, but she has never been physically active for most of her life. One day she falls out of bed and immediately notices a sharp pain in her right hip. She is subsequently unable to ambulate without severe pain. Radiographs show not only a fracture of the right femoral head, but also a compressed fracture of T10. Which of the following conditions is she **most likely** to have?

- Vitamin D deficiency
- Osteomyelitis
- Osteogenesis imperfecta
- Osteoporosis

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• **Osteoporosis** → also called Albers-Schoenberg Disease and Marble Bone Disease

Osteoporosis is a generalized hereditary condition consisting of excessive bone mineralization, resulting in altered stature, frequent fractures, lack of bone marrow hematopoietic function, and a tendency for severe osteomyelitis of the jaws. It is the **most common type of bone disease.**
Osteoporosis occurs when the body fails to form enough new bone, or when too much old bone is reabsorbed by the body, or both. Calcium and phosphate are two minerals that are essential for normal bone formation. As people age, calcium and phosphate may be reabsorbed back into the body from the bones, which makes the bone tissue weaker. Both situations can result in brittle, fragile bones that are subject to fractures *(pathologic fractures)*, even in the absence of trauma.

The **leading causes of osteoporosis** are a drop in estrogen in women at the time of menopause, and a drop in testosterone in men. Women, especially those over the age of 50, get osteoporosis more often than men. The treatment includes estrogen therapy, calcium supplements and vitamin D.

Osteomyelitis is an acute pyogenic infection of bone, most often caused by Staphylococcus aureus. The infection that causes osteomyelitis often is in another part of the body and spreads to the bone via the blood. Affected bone may have been predisposed to infection because of recent trauma. **In children,** the **long bones** are usually affected. In **adults,** the **vertebrae** and the **pelvis** are most commonly affected. When the bone is infected, pus is produced within the bone, which may result in an abscess. The abscess then deprives the bone of its blood supply. It often presents the following symptoms: pain, redness, and swelling in the affected area. Fever and malaise are usually evident. **Risk factors** are recent trauma, diabetes, hemodialysis, and intravenous drug abuse. People who have had their spleen removed are also at higher risk for osteomyelitis. **Note:** Chronic osteomyelitis results when bone tissue dies as a result of the lost blood supply. Chronic infection can persist intermittently for years.

All of the following statements concerning **condensing osteitis** are true **except:**

- It most often occurs in young patients
- The mandibular second molar is the tooth most commonly involved
- It is most often seen associated with a long-standing periapical infection
- There may be no signs or symptoms of the disease other than mild pain associated with an infected pulp

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• **The mandibular second molar is the tooth most commonly involved**

***This is **false;** the **mandibular first molar** is the tooth most commonly involved.
Chronic focal sclerosing osteomyelitis *(a fancy name for condensing osteitis)* is an unusual reaction of bone to infection, occurring in instances of extremely high tissue resistance, or in cases of low-grade infection.

The periapical x-ray demonstrates the pathognomonic, **well-circumscribed radiopaque mass** of sclerotic bone surrounding and extending below the apex of one or both roots. The **entire root outline is almost always visible,** an important feature in distinguishing it from the **benign cementoblastoma,** which radiographically, it may resemble. The tooth with this lesion may be treated or extracted, since the pulp is infected and the infection has spread past the immediate periapical area. The sclerosing bone constituting the osteomyelitis is not attached to the tooth, and remains after the tooth is treated or removed.

Condensing osteitis

Benign cementoblastoma

Cleft lip results when the medial nasal process fails to fuse with the lateral portions of the:

- Nasal septum
- Secondary palate
- Lateral nasal process
- Maxillary process

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• **Maxillary process**

Cleft lip results when the medial nasal process fails to fuse with the lateral portions of the maxillary process of the first branchial arch. Fusion normally occurs during the **sixth and seventh weeks** of embryonic development. The maxillary lip is most commonly affected. It may be bilateral or unilateral. Clefts of the lip are more frequent in **males.** Lip cleft involvement is more frequent on the **left side** than the right.

Cleft palate occurs in the **eighth to tenth week** of embryonic life. Isolated clefts of the palate are more common in females. It is characterized by a fissure in the midline of the palate, resulting from the failure of the two sides to fuse during embryonic development. The most severe handicap imposed by cleft palate is an impaired mechanism **preventing normal speech and swallowing.**

Notes:

1. Speech problems associated with both of the above are usually the result of the **inability of the soft palate** to close airflow into the nasal area.
2. It is not unusual for teeth especially the **lateral incisor** to be missing in the cleft area.
3. More than 250 syndromes have been identified that may be associated with cleft lip and palate, or cleft palate alone.

A child with **achondroplasia** has a:

- Relatively normal torso and long arms and legs
- Short torso and long arms and legs
- Long torso and long arms with short legs
- Relatively normal torso and short arms and legs

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- **Relatively normal torso and short arms and legs**

Achondroplasia is the most common type of dwarfism. The upper arms and thighs are more shortened than the forearms and lower legs. Generally, the head is large, the forehead is prominent, the nose has a **saddle-like** appearance and the **mandible exhibits prognathism.**

Potential problems in children with achondroplasia include **overcrowding of the teeth,** speech problems *(articulation)*, and frequent ear infections *(otitis media)*. Dental malocclusion is treated with orthodontics. All children with achondroplasia should be evaluated by a speech therapist by two years of age because of possible problems with the development of clear speech. Articulation problems may be caused by orthodontic problems. Due to the abnormal shape of the eustachian tube in an individual with achondroplasia, they are very prone to ear infections *(otitis media)*.

Note: The teeth are of normal size but there is **limited space** within the maxillary and mandibular arches for them to erupt into which causes overcrowding and subsequent malocclusion.

The cause of **osteogenesis imperfecta** is believed to be due to a genetic defect that causes imperfectly-formed, or an inadequate amount of:

- Collagen
- Reticulin
- Elastin
- All of the above

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• **Collagen**

Collagen is the major protein of the body's connective tissue. In osteogenesis imperfecta, a person has either less collagen than normal, or a poorer quality of collagen than normal – **leading to weak bones that fracture easily.**

The characteristic features of osteogenesis imperfecta **vary greatly** from person to person and **not all characteristics** are evident in each case. The chief clinical characteristic of osteogenesis imperfecta is the extreme fragility and porosity of the bones, with a **proneness to fracture.** Other features include: pale blue sclera, deafness due to otosclerosis, abnormal teeth *(***Remember: Type I dentinogenesis imperfecta is associated with osteogenesis imperfecta***)*, loose joints and low muscle tone, a triangular face, and a tendency toward spinal curvature.

Clinically the teeth have:
- Crowns that are bulbous; with a cervical constriction.
- Pulps that are obliterated, either partially or completely.
- Roots that are narrower and shorter.

***The deciduous teeth are **more severely affected** that the permanent dentition.

Types of osteogenesis imperfecta:
- Type I: most common and mildest form
- Type II: most severe form resulting in multiple fractures just from birth process
- Type III: most severe form beyond the perinatal period
- Type IV: mild to moderately severe bone fragility

There is **no known** cure for osteogenesis imperfecta. Treatment is directed toward preventing or controlling the symptoms.

Hypophosphatasia is a genetic metabolic disorder of bone mineralization caused by a deficiency in:

- Acid phosphatase
- Vitamin K
- Alkaline phosphatase
- Phosphorus

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- **Alkaline phosphatase**

Hypophosphatasia is an inherited metabolic *(chemical)* bone disease that results from low levels of an enzyme called **alkaline phosphatase.** This enzyme is essential to the calcification of bone tissue. The severity of hypophosphatasia is remarkably variable from patient to patient. Some patients have blue sclera that resembles **osteogenesis imperfecta.** There may be deformity of the arms, legs and chest. Frequent bouts of pneumonia can occur as well as recurrent fractures.

4 types of hypophosphatasia:

- **Neonatal:** severe manifestations, respiratory failure, marked hypocalcification of the skeletal structures
- **Infantile:** hypercalcemia, premature loss of deciduous teeth, skeletal malformations, failure to grow
- **Childhood:** short stature, frontal bossing, usually normal calcium and phosphate levels
- **Odontohypophosphatasia:** children and adults who have only dental problems

Important: The premature loss of teeth in children and adults is usually characteristic. These teeth also exhibit hypocalcification. **Radiographically,** the teeth display enlarged pulp chambers and pulp canals, deficient root development as well as alveolar bone loss.

Remember: Patient's with Paget's disease also have high levels of serum alkaline phosphatase.

Acromegaly is a hormonal disorder that results when the pituitary gland produces excess:

- ACTH
- Growth hormone
- TSH
- Oxytocin

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• **Growth hormone**

In over 90% of acromegaly patients, the overproduction of GH is caused by a benign tumor of the pituitary gland, called an adenoma. Whether or not the epiphyses of the long bones have fused with the shaft is the main determinant of whether gigantism or acromegaly will occur when there is oversecretion of growth hormone by the pituitary gland.

Remember:
- **Gigantism** → tumor prior to adolescence *(non-fusion of epiphyses)*
- **Acromegaly** → tumor after adolescence *(fusion of epiphyses)*

Oral manifestations of acromegaly and gigantism include: enlarged tongue, mandibular prognathism, spacing of the teeth which are usually tipped to the buccal or lingual side, owing to enlargement of the tongue. Roots may be longer than normal. **See picture #33 in booklet.**

Note: Dwarfism *(pituitary dwarfs)* is characterized by arrested growth. Frequently these people have limbs and features not properly proportioned or formed. It is caused by undersecretion of growth hormone. **Oral manifestations** include: eruption rate and the shedding of the teeth are delayed, clinical crowns appear smaller as do the roots of the teeth, the dental arch as a whole is smaller causing malocclusion and the **mandible is underdeveloped.**

Cone-shaped teeth are characteristic of:

- Pierre Robin syndrome
- Ectodermal dysplasia
- Cleidocranial dysplasia
- Peutz-Jeghers syndrome

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• **Ectodermal dysplasia**

Ectodermal dysplasia is a hereditary condition characterized by abnormal development of the skin and associated structures *(hair, nails, and teeth, and sweat glands).* It involves all structures which are derived from the **ectoderm.** It affects males more than females. Common clinical findings include **hypothrichosis** *(decrease in hair)*, **anhidrosis** *(no sweat glands, leading to heat intolerance),* **anodontia** or **oligodontia** *(complete or partial absence of teeth),* **depressed bridge of nose, lack of salivary glands** and the **child appears much older than what he or she is.** There is no treatment for the disease, however dentures can be fabricated for these patients. Keep in mind that they will need to be replaced periodically to accommodate the patient's jaw growth.
See pictures #28 and #29 in booklet.

Cleidocranial dysplasia is an inherited disorder of bony development characterized by absent or incompletely formed collar bones, a characteristic facial appearance *(a heavy protruding jaw and wide nasal bridge)*, and dental abnormalities which include **retained primary teeth,** malaligned teeth, the presence of **multiple supernumerary** teeth and **unerupted** teeth. **Important:** The dentition itself, as observed by radiographs alone, often suggests the diagnosis.
See picture #30 in booklet.

Pierre Robin syndrome is an inherited disorder that presents the following in the neonate: **micrognathia and retrognathia** of the mandible, **glossoptosis** *(posterior displacement of the tongue),* and **cleft palate.** This condition is characterized by respiratory problems.

Peutz-Jeghers syndrome *(PJS)* is a genetic condition marked by hyperpigmentation *(freckling)* of the lips and sometimes other parts of the face, hands, and feet followed by the development of benign polyps called hamartomas throughout the intestines but primarily in the small intestine.
See picture #35 in booklet.

Radiolucent / radiopaque lesions resembling a **"cotton ball" or "cotton wool"** appearance of the skull is characteristic of:

- Cleidocranial dysplasia
- Paget's disease
- Osteoporosis
- Stevens-Johnson syndrome

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- **Paget's disease**

***Paget's disease of bone is also called **Osteitis Deformans.**

Paget's disease of bone is a chronic bone disorder in which bones become enlarged and deformed. The bone may become dense, but fragile, because of excessive breakdown and formation of bone. The disease is more common in males and is rarely found in people under the age of 40. The cause is unknown *(it appears to be familial).* The signs and symptoms include pain in the affected area, deformity of the bone in the affected area, susceptibility to fractures in the affected area, and headache and hearing loss if the affected area is the skull. **Note:** These symptoms develop **slowly.**

Other important features of this disease include:

- Patients may also give a history of progressively increasing size of hats or new dentures being made at progressively more frequent intervals. **Note:** This is due to bony changes.
- Bones are warm to touch due to increased vascularity.
- X-rays of the skull and the jaws demonstrate the typical **"cotton-wool"** appearance. The teeth have pronounced hypercementosis and, often, the loss of lamina dura.
- **Lab tests:** Drastically **increased serum alkaline phosphatase.** Serum phosphate and calcium are normal. Urinary calcium and hydroxyproline are increased.
- **Treatment:** Calcitonin decreases bone resorption. Antimetabolites may also be used.

Important: These patients are predisposed to developing **osteosarcomas**. **See picture #32 in booklet.**

Cherubism is a familial disease that derives its name from the fact that in severe cases involvement of the maxilla and encroachment on the orbits cause an upward turning of the eyes. **Histologically,** the lesions are characterized by:

- Intact epithelium with pseudoepitheliomatous hyperplasia
- Irregular rete ridges
- Multiple, small, granulomas composed of histiocytes
- Multinucleated giant cells

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• **Multinucleated giant cells**

Cherubism is a benign inherited disease of the maxilla and mandible, usually found in children by 5 years of age *(it affects males 2:1).* The vast majority of cases occur in the **mandible.** The jaws are firm and hard to palpation and regional lymphadenopathy may be present. There are **no associated** systemic manifestations. The deciduous dentition may be spontaneously shed prematurely, beginning as early as three years of age. There is often **delayed eruption** of the permanent dentition which is often defective with the absence of numerous teeth and displacement of those present.

Radiographically, the lesions characteristically appear as multiple, well-defined, **multilocular radiolucencies** of the jaw.

Histologic features:

- **Multinucleated giant cells** with ovoid to spindle shaped cells within a fine fibrillar collagenous stroma
- Numerous small vascular spaces with large endothelial cells within connective stroma

Important: Histologically, the lesions bear a close resemblance to those seen in **central giant cell granulomas.** However, **perivascular collagen cuffing** is regarded as pathognomonic for cherubism. **Note:** Hyperparathyroidism and aneurysmal bone cysts also contain multinucleated giant cells histologically.

The treatment is cautious waiting as there is spontaneous regression of the tumors at around 25 – 30 years of age. With increase in age and size of the patient, the deformity produced is less noticeable.

The most acute and severe type of **hyperthyroidism** is:

- Toxic nodular goiter
- Graves' disease
- Hashimoto's disease
- Addison's disease

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• **Graves' disease**

The term **hyperthyroidism** refers to any condition in which there is too much thyroid hormone *(thyroxin)* in the body. This most commonly results from a generalized overactivity of the entire thyroid gland, a condition also known as **diffuse toxic goiter** or **Graves' disease.** Alternatively, one or more nodules or lumps in the thyroid may become overactive, a condition known as **toxic nodular goiter** or **Plummer's disease.** The primary role of thyroxin is to stimulate cellular metabolism, growth and differentiation of all tissues. **In excess,** it leads to high basal metabolism, fatigue, weight loss, excitability, elevated temperature and generalized osteoporosis. **Oral manifestations are not too remarkable,** but if the disturbance begins in the early years of life, the premature eruption of the teeth and the premature loss of the deciduous dentition are common findings.

Types of hyperthyroidism: (**Note:** Basic symptoms of hyperthyroidism are present as well as additional symptoms).

- **Graves' disease** → is the **most common** form, occurs most frequently in women under 50
  - **goiter** *(enlarged thyroid which may cause a bulge in the neck)*
  - **exophthalmos** is common
  - thickened skin over the shin area
- **Plummer's disease** *(toxic nodular goiter)* → affects both genders usually over 50
  - **Exophthalmos is rare**
  - Often uni-system, may present with only cardiac disease

Important: Thyroiditis causes temporary hyperthyroidism, usually followed with **hypothyroidism.** Thyroiditis is an inflammation of the thyroid gland. There are **three types of thyroiditis:**

- **Hashimoto's** thyroiditis
- Subacute granulomatous thyroiditis
- Silent lymphocytic thyroiditis

Severe hypothyroidism in a **child** is called:

- Dwarfism
- Myxedema
- Cretinism
- Acromegaly

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• **Cretinism**

Hypothyroidism refers to a condition in which the amount of thyroid hormone in the body is below normal. This is the **most common** form of thyroid function abnormality, and is far more common than hyperthyroidism. This condition is considerably more common in **women** than in men. The most common cause of hypothyroidism is **Hashimoto's thyroiditis.** The second most common cause is the treatment of hyperthyroidism. Hypothyroidism is characterized by puffiness of the face and eyelids and swelling of the tongue and larynx. The skin becomes dry and rough and the hair becomes sparse. The individual has a **low basal-metabolic rate** and a low body temperature. The affected individuals also have poor muscle tone, low strength and get tired very easily. Mentally they are very sluggish. The treatment of hypothyroidism is straight-forward and consists of administering thyroid hormone *(thyroxin).*

Severe hypothyroidism in a child is called **cretinism.** Due to a lack of thyroid hormone, there is a retardation of growth and an abnormal development of bones. Mental retardation is caused by the improper development of the CNS. If this condition is recognized early, it can be markedly improved with the use of thyroid hormones. **Note: Extreme hypothyroidism** in adults is called **myxedema.**

Note: Dental findings in a child with hypothyroidism include an **underdeveloped mandible** with an overdeveloped maxilla, **enlarged tongue** which may lead to malocclusion, delayed eruption of teeth and deciduous teeth being retained longer.

Pathologic fracture may be the first symptom of:

- Hyperparathyroidism
- Dentinogenesis imperfecta
- Lupus erythematosus
- Erythema multiforme

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• **Hyperparathyroidism**

Hyperparathyroidism is a metabolic disorder in which the parathyroid glands produce too much parathyroid hormone. Too much parathyroid hormone causes too much calcium to be released from bone. There is a female predilection and it affects middle aged adults. The **symptoms include:** loss of appetite, increasing thirst, frequent urination, lethargy and fatigue, muscle weakness, joint pain and constipation. **Important: pathologic fracture** *(due to the marked resorption of bone)* may be the first symptom of the disorder. **Intraorally,** there is diffuse bone loss causing malocclusion and shifting of the teeth.

The chief **radiographic finding** is the appearance of **well-defined cystic radiolucencies** of the jaw, which may be unilocular or multilocular. Partial loss of the lamina dura is seen around the teeth.
See pictures #34 in booklet.
Types of hyperparathyroidism:

- **Primary Hyperparathyroidism:** Uncontrolled production of PTH as a result of a parathyroid adenoma or parathyroid hyperplasia. Generalized disorder of calcium, phosphate, and bone metabolism due to an increase secretion of parathyroid hormone. Most patients are older than 60 years of age. Classic triad of symptoms: stones *(renal calculi)*, bones *(numerous osseous changes)* and abdominal groans *(duodenal ulcers)*.
- **Secondary Hyperparathyroidism:** Develops when PTH is continuously produced in response to chronic low levels of serum calcium, a physiologic response to renal failure.
- **Tertiary Hyperparathyroidism:** Occurs after secondary hyperparathyroidism when the external factor is corrected but the parathyroid glands remain hyperplastic.

Important: Histologically, **multinucleated giant cells** are found. These cells are also found in cherubism and the aneurysmal bone cyst.

Many cases of **osteomalacia** are related to abnormalities in:

- Vitamin A
- Vitamin D
- Vitamin C
- Vitamin E

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• **Vitamin D**

Osteomalacia involves softening of the bones caused by a deficiency of vitamin D or problems with the metabolism of this vitamin. This softening of the bones occurs because the bones contain osteoid tissue which has failed to calcify due to the lack of Vitamin D. **Note:** Osteomalacia may occur as a complication of steatorrhea secondary to chronic pancreatitis.

Clinical findings:
- Osteopenia
- Bone softening / deformity → hourglass thorax, bowing of long bones
- Increased fractures, biconcave vertebral bodies
- Mottled skull
- Pseudofractures

All bones are effected, specifically their epiphyseal growth plates. Osteomalacia appears to be more common in women. This condition may be asymptomatic until fracture occurs.

Blood tests may show:
- Low levels of vitamin D
- May also show low calcium and phosphorus levels
- Alkaline phosphatase levels can be high

Rickets is osteomalacia in children. It causes skeletal deformities. It is usually accompanied by listlessness, irritability and generalized muscular weakness. A child with rickets may have bowlegs and develop a pigeon breast and a protruding stomach. The **teeth** in a child with rickets are affected as follows: delayed eruption, malocclusion and developmental abnormalities of the dentin and enamel along with a higher caries rate.

All of the following conditions may be seen in a patient with cerebral palsy **except:**

- Difficulty with mastication and swallowing
- Higher incidence of periodontal disease and caries
- They are more susceptible to trauma, particularly to the maxillary anterior teeth
- Attrition of the teeth
- Multilocular radiolucencies of the jaws

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• **Multilocular radiolucencies of the jaws**

Cerebral palsy is a term used to describe a group of disorders affecting body movement and muscle coordination. It is due to an insult to or anomaly of the brain's motor control centers. This damage interferes with messages from the brain to the body, and from the body to the brain. The effects vary widely from individual to individual. Cerebral palsy is characterized primarily by spastic paralysis or impairment of control or coordination over voluntary muscles and is often accompanied by mental retardation, seizures and disorders of vision and communication.

Note: No intraoral anomalies are unique to persons with cerebral palsy. **However,** several conditions *(those listed on the front of the card)* are more common or more severe than in the normal population.

Remember: Down syndrome is a congenital defect caused by a chromosomal abnormality *(trisomy 21)*. It is marked by various degrees of mental retardation and characteristic physical features such as a short, flattened skull, slanting eyes, **a thickened tongue** *(fissured),* broad hands and feet and other anomalies. **Other oral manifestations include** mandibular prognathism, increased incidence of periodontal disease, delayed eruption of teeth, higher incidence of congenitally missing teeth, malocclusion, and enamel dysplasia.

Oral manifestations of **cystic fibrosis** include all of the following **except:**

- Tend to be mouth breathers
- Salivary glands enlarge
- Increase in dental caries
- Increase in calculus deposits
- Intrinsic teeth staining due to tetracycline

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- **Increase in dental caries**

***Important:** There is a significantly **reduced caries rate** in patients with cystic fibrosis. This is **probably** the result of alterations in saliva and the long-term use of antibiotics.

Cystic fibrosis is a congenital metabolic disorder that causes the exocrine glands *(which are glands that secrete fluids into a duct)* to **produce abnormal secretions**, resulting in several symptoms, the most important of which affect the digestive tract and the lungs. In some glands, such as the pancreas and those in the intestines, the secretions are thick or solid *(an excessively* **viscous mucous***)* and may block the gland completely. The mucous-producing glands in the airways of the lungs produce abnormal secretions that clog the airways and allow bacteria to multiply. The sweat glands secrete fluids that have a high sodium and chloride content. **Note:** The staining of the teeth is most likely due to the fact that patients with cystic fibrosis are usually subjected to large amounts of **tetracyclines** during childhood.

Symptoms of CF include:
- Poor growth despite good appetite
- Malabsorption and foul, bulky stools → steatorrhea
- Chronic bronchitis *(COPD)* with cough
- Recurrent pneumonia → respiratory infections
- Clubbing of fingers and toes
- Barrel-chested appearance

Common hepatitis virus infections include Hepatitis A, Hepatitis B and Hepatitis C. Which is the **least serious and most mild** of these diseases?

- Hepatitis A
- Hepatitis B
- Hepatitis C

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• **Hepatitis A** *(also called infectious, viral, or short-incubation hepatitis)*

Hepatitis A is a highly contagious infectious disease involving the liver. It is usually transmitted by the fecal-oral route. However, it may also be transmitted parenterally. Hepatitis A usually results from ingestion of contaminated food, milk, or water. Many outbreaks of this type are traced to ingestion of seafood from polluted water. It is caused by an **RNA enterovirus.** It most often occurs in young adults. The initial symptoms *(fever, malaise, abdominal pain, anorexia, jaundice)* of Hepatitis A appear after an **incubation period of 3-6 weeks.**

Damage to the liver cells, also results in **increased serum levels** of enzymes, such as **transaminases,** normally active in liver cells. The detection of increased serum levels of these enzymes is used in diagnosing this disease. In most cases of Hepatitis A, the infection is self-limiting and recovery occurs within 4 months.

Oral complications: The only oral complication associated with hepatitis is the **potential for abnormal bleeding** in cases of significant liver damage. If surgery is required, it is advisable to:

- Check the prothrombin time. If it is greater than 35, an injection of vitamin K will usually correct the problem. This should, however, be discussed with the patient's physician.
- Monitor the bleeding time to check platelet function. If it is not less than 20 minutes, the patient may require platelet replacement before surgery. This should also be discussed with the patient's physician.

Notes:

1. The presence of **surface antigen** *(A or B)* in a patient's serum indicates that the patient is potentially infectious for Hepatitis *(carrier state).*
2. Hepatitis viruses are very heat-resistant *(more so than the AIDS virus).*
3. Autoclaving properly **will kill** these viruses.

Which type of Hepatitis is found only in patients with acute or chronic episodes of Hepatitis B?

- Hepatitis A
- Hepatitis C
- Hepatitis D
- Hepatitis E

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- **Hepatitis B** → this virus causes "serum" or long-incubation hepatitis

The Hepatitis B virus *(HBV)* is a double-stranded DNA virus with worldwide distribution, transmitted by parenteral and sexual contact. Risk factors include multiple sexual partners, **intravenous drug abuse,** and receipt of blood products. Its incubation period is 40 to 100 days, and it can be recovered from all body fluids, most importantly, blood, breast milk, and amniotic fluid. The signs and symptoms are similar to hepatitis A *(fever, abdominal pain, nausea, etc.)* but there is a **longer incubation period** *(6-8 weeks)*. The symptoms are slower in developing but are of a longer duration. Most patients recover fully, however, some develop chronic liver disease.

Previously, viral hepatitis that was not caused by the type A or type B virus was called "non-A, non-B hepatitis." Recently three more viruses have been identified that cause some of these non-A, non-B infections.

These new viruses include:

- **Hepatitis C** → is a serum hepatitis that is caused by a virus antigenically different from Hepatitis viruses A and B. Most cases of post-transfusion hepatitides are of this type. It is usually much milder than A or B but is otherwise clinically indistinguishable from them. There is a **higher incidence of chronic disease** *(chronic hepatitis)*, cirrhosis and **hepatocellular carcinoma.**
  Note: Hepatitis C is now the most common reason for liver transplantation in the United States.
- **Hepatitis D** → is found **only in patients with acute or chronic episodes of Hepatitis B,** and it makes the Hepatitis B infection more severe. Drug addicts are at relatively high risk.
- **Hepatitis E** → is transmitted enterically, much like Hepatitis A. It causes occasional epidemics similar to those caused by Hepatitis A. So far these epidemics have occurred only in underdeveloped countries.

Which of the following is one of the most important substances that **influences** the rate of healing of wounds in the oral cavity?

- Fat
- Protein
- Iron
- Calcium

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- **Protein**

***Nutritional factors** such as the amount of **protein** a patient is consuming is one of the most important factors which may effect the speed of wound healing. Hypoproteinemia has been shown to delay wound healing, while having a high protein diet has been shown to accelerate wound healing. **Vitamins**, especially Vitamin C, have been shown to be important in proper wound healing.

Other factors that influence the rate of healing:

- **Location of the wound** → wounds in an area in which there is a good vascular bed heal considerably more rapidly than wounds in an area which is relatively avascular.
- **Physical factors** → severe trauma to tissue is a deterrent to rapid wound healing. The local temperature in the area of a wound influences the rate of healing. In environmental hyperthermia, wound healing is accelerated; while in hypothermia, healing is delayed.
- **Circulatory factors** → anemia and dehydration have been found to delay the healing of wounds.
- **Age of patient** → wounds in younger persons heal considerably more rapidly than wounds in elderly persons.
- **Infection** → bacterial invasion will retard healing.
- **Hormonal factors** → **ACTH and cortisone** are substances that have been known to interfere with the healing of wounds. **Diabetes mellitus** *(insulin deficiency)* is one of the most widely recognized diseases in which there is significant, clinically evident, retardation in repair of wounds.

The **fixative of choice** commonly used to preserve biopsy specimens for routine histological examination is:

- Saline
- 10% buffered formalin
- Distilled water
- Alcohol

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• **10% buffered formalin**

A biopsy is a procedure performed to remove tissue or cells from the body for examination under a microscope. Biopsies are usually performed to determine whether a tumor is malignant or to determine the cause of an unexplained infection or inflammation.

When the entire tumor is removed, it is called **excisional biopsy** technique. If only a portion of the tumor is removed, it is called **incisional biopsy** technique.

An **incisional biopsy** *(also called diagnostic biopsy)* is done when lesions are **too large to excise** initially without having established a diagnosis or are of such a nature that excision would be inadvisable.

An **excisional biopsy** is preferred if the size of the lesion is such that it may be removed along with a margin of normal tissue and the wound can be closed primarily. **Example:** A 1-cm exophytic mass *(which is a lesion that grows outward from an epithelial surface)* on the cheek.

Notes:

1. **Biopsy** is the most reliable technique to diagnose soft tissue lesions.
2. The **scalpel** is the instrument of choice since it cleanly removes the tissue and does not dehydrate it as cautery or the high-frequency cutting knife may.
3. The rationale for surgical removal and biopsy of a large periapical lesion suspected to be of inflammatory origin is that a clinical diagnosis can be confirmed microscopically.

Remember: This is the only way **to distinguish** between a granuloma and a cyst.

Which of the following is the cause of **infectious mononucleosis?**

- Streptococci
- Epstein-Barr virus
- Rubella viruses
- Paramyxovirus

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• **Epstein-Barr virus**

The Epstein-Barr virus *(EBV)* is a member of the herpes virus group. It causes infectious mononucleosis and has been associated with the subsequent development of two forms of cancer: **Burkitt's lymphoma** and **nasopharyngeal carcinoma**. EBV is also associated with **hairy leukoplakia,** a whitish, nonmalignant lesion on the tongue seen especially in AIDS patients. The virus specifically **infects B-lymphocytes** and some epithelial cells. It is associated with the production of atypical lymphocytes and **IgM heterophile antibodies** identified by the heterophile test *(also called the mononucleosis spot test)*. This antibody eventually appears in the serum of more than 80% of the patients with infectious mononucleosis, hence it is **highly diagnostic** of the disease.

Notes:

1. There are no specific oral manifestations of infectious mononucleosis, although secondary lesions do occur.
2. Neck swellings are characteristic of infectious mononucleosis, Hodgkin's disease and tuberculosis.

Remember:

- **Rubella** viruses cause German measles *(rubella)*, which present with a characteristic rash *(flat, pink spots on the face and then spreads to other body parts).*
- **Paramyxoviruses** can cause measles *(rubeola)* and mumps. Rubeola is characterized by the formation of **Koplik's spots** in the oral cavity. These spots are small, bluish-white lesions surrounded by a red ring. They cannot be wiped off and occur opposite the molars. **Mumps** cause enlargement of the parotid glands. Serious complications include deafness in children and orchitis *(inflammation of the testis)* in males past puberty.

Epithelioid cells and giant cells are derived from macrophages and are important in the development of:

- Initial inflammation
- Granulomatous inflammation
- Acute inflammation
- Subacute inflammation

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• **Granulomatous inflammation**

Granulomatous inflammation is a subtype of **chronic** inflammation and is characterized by granulomas, which are nodular collections of specialized macrophages referred to as **epithelioid** cells. A rim of lymphocytes usually surrounds granulomas. Granulomas are produced by **multinucleated giant cells** *(Langerhans giant cells and foreign body giant cells)*. All the other cell types characteristic of chronic inflammation, including plasma cells, eosinophils, and fibroblasts, may also be associated with granulomas.

Note: Granulomatous inflammation is characteristically associated with areas of **caseous necrosis** produced by infectious agents, particularly **Mycobacterium tuberculosis.**

Etiologic agents associated with granulomatous inflammation:

- **Infectious agents**
  - TB and leprosy, which are mycobacterial diseases
  - Fungal infections *(blastomycosis, histoplasmosis, and coccidioidomycosis)*
  - Spirochetes *(Treponema pallidum, which causes syphilis)*
  - Cat scratch disease *(caused by an unnamed gram-negative organism)*
- Foreign material (*e.g., suture or talc)*
- Sarcoidosis *(unknown etiology; it is **non-necrotizing**)*
- Crohn's disease *(it is **non-caseating**)*

Ludwig's angina is a severe and spreading infection that involves the:

- Submental and sublingual spaces only
- Submandibular, submental, and sublingual spaces unilaterally
- Submandibular and sublingual spaces only
- Submandibular, submental, and sublingual spaces bilaterally

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• **Submandibular, submental, and sublingual spaces bilaterally**

Ludwig's angina often results from an **odontogenic infection.** As a result, the bacteriology of theses infections generally involves oral flora, particularly anaerobes. Other recognized etiologies of Ludwig's angina include poor oral hygiene, IV drug abuse, trauma, and tonsillitis.

It is characterized by:

- Rapid onset
- The three facial spaces are involved **bilaterally**
- Board-like swelling of floor of the mouth and no fluctuance is present
- Typical **"openmouthed"** appearance
- Elevation of the tongue
- Drooling, trismus, and fever
- Difficulty eating, swallowing, breathing
- Tachycardia, increased respiration rate
- Can lead to glottal edema; asphyxiation

Airway management, massive antibiotic coverage *(IV)*, and surgical incision and drainage are the mainstays of treatment.

Very important: The **most serious complication** of Ludwig's angina is **edema of the glottis** *(which is a slit-like opening between the true vocal cords).*

The **erythrocyte sedimentation rate** *(ESR)* is a nonspecific screening test that monitors:

- Bleeding rate
- The progression of disease
- Prothrombin time
- Platelet function

• **The progression of disease**

The ESR is the rate at which red blood cells settle out in a tube of unclotted blood expressed in millimeters per hour. Blood is collected in an anticoagulant and allowed to sediment in a calibrated glass column. At the end of one hour, the lab technician measures the distance the erythrocytes have fallen in the tube. Elevated sedimentation rates are not specific for any disorder but **indicate the presence of inflammation**. Inflammation causes an alteration of the blood proteins which makes the red blood cells aggregate, becoming heavier than normal. The speed with which they fall to the bottom of the tube corresponds to the degree of inflammation.

ESR rises during:

- Inflammation
- Tissue degeneration
- Suppuration
- Necrosis

Note: Certain non-inflammatory conditions, such as pregnancy, are also categorized by high sedimentation rates.

The **estimated toxic dose** for fluoride ingestion is:

- 1–2 mg / kg
- 5–10 mg / kg
- 8–10 mg / kg
- 12–15 mg / kg

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- **5 – 10 mg / kg**

***Death may result from ingesting as little as 2 g of fluoride in an adult and 16 mg / kg in children. Symptoms may appear with 3-5 mg / kg of fluoride.

Symptoms of **acute** fluoride poisoning include nausea, abdominal pain, vomiting, diarrhea, convulsions, and hypotension.

The treatment for acute fluoride poisoning includes: **1)** Call poison control center, **2)** Monitor vital signs, **3)** Initiate basic life support as needed and **4)** Get patient to the hospital.

Fluoride poisoning may be acute *(caused by a single large dose of fluoride)* or chronic *(caused by long-term ingestion of fluoride).* The characteristic signs of **chronic** fluoride poisoning are:

1. **Osteosclerosis** of the bones → which results from long-term ingestion of water with 10 to 25 ppm of fluoride.
2. **Dental fluorosis** *(enamel hypoplasia)* → which is due to fluoride intake during the **calcification stage** of tooth development. This can occur in permanent and deciduous teeth.

Note: It has been estimated that the average American diet contains about 0.2 to 0.3 mg of fluoride per day. If 1 ppm of fluoride is added to the drinking water, about 1 to 2 mg of fluoride will be added to the diet daily. Balance studies have shown that when quantities of fluoride ingested **do not exceed 4 to 5 mg daily,** little is retained by the body. The finding indicates the safety of the preventive dentistry programs based on the addition of fluoride to drinking water in concentrations of approximately 1 ppm. **Sodium sillicofluoride** is the type of fluoride often used for fluoridation of the communal water supply.

Important: Fluoride normally accumulates slowly in bones as a person ages. **However,** if ingested in very high amounts, it accumulates rapidly. The intake of calcium in high doses will reduce the absorption of dietary fluoride.

Patients with **xerostomia** are best treated with:

- Fluoride
- Sodium carboxymethylcellulose
- Steroids
- Antibiotics

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• **Sodium carboxymethylcellulose**

Xerostomia *(dry mouth)* is not a disease, however, it can be a symptom of certain diseases. Many times xerostomia is caused by failure of the salivary glands to function normally, but the sensation can also occur in people with normal salivary glands. Xerostomia can cause health problems by affecting nutrition, as well as psychological health. At its most extreme, it can lead to **rampant tooth decay** and **periodontal disease.**

Perhaps the **most prevalent cause** of xerostomia is **medication.** The main culprits are antihistamines, antidepressants, anticholinergics *(eg, atropine and scopolamine)*, anorexiants, antihypertensives, antipsychotics *(eg, chlorpromazine and prochlorperazine)*, anti-Parkinson agents, diuretics and sedatives.

The most common **disease** causing xerostomia is **Sjögren's syndrome** *(SS)*, a chronic inflammatory autoimmune disease that occurs predominantly in postmenopausal women. Sarcoidosis and amyloidosis are other chronic inflammatory diseases that cause xerostomia. Other systemic diseases that can cause xerostomia include rheumatoid arthritis, systemic lupus erythematosus, and scleroderma. **Remember:** Xerostomia is the most common toxicity associated with radiation therapy to the head and neck.

Commercially available saliva substitutes in general, contain an agent to increase viscosity, such as carboxymethylcellulose or hydroxymethylcellulose, minerals such as calcium and phosphate ions and fluoride, preservatives such as methyl- or propylparaben, and flavoring agents. Examples include Xero-Lube®, Salivart® and Optimoist®.

The greatest number of skin cancer–related deaths worldwide is caused by:

- Basal cell carcinoma
- Actinic keratosis
- Squamous cell carcinoma
- Malignant melanoma

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• **Malignant melanoma**

Malignant melanoma accounts for only 4% of all skin cancers; however, it causes the greatest number of skin cancer–related deaths worldwide.

Clinical features:

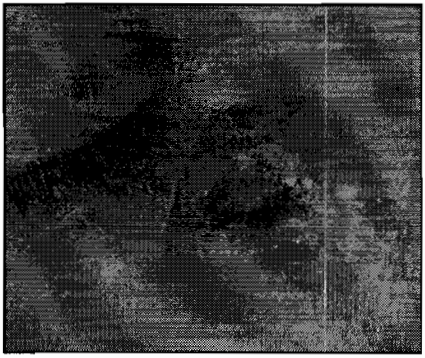
- Males more often than females
- Usually palate or maxillary gingiva
- 5th decade or older
- A, B, C, D's of melanoma:
  - **A** = asymmetry
  - **B** = border irregularity
  - **C** = color variability *(brown, black, blue, gray, pink)*
  - **D** = diameter of *(often)* greater than 1/4 inch

Excessive exposure to UV radiation from the sun may be the primary cause of melanoma. Malignant melanoma has been linked to both a lot of sun exposure over a lifetime and to painful sunburns during childhood.

Malignant melanoma is an **uncommon neoplasm of the oral mucosa.** It exhibits a definite predilection for **the palate and the maxillary gingiva/alveolar ridge.** Unfortunately, oral mucosal melanomas have a dismal prognosis. **See picture #36 in booklet.**

Note: A **nevus** is a mole. Almost all moles are normal. **Atypical** *(dysplastic)* **nevi** are unusual moles that are generally larger than normal moles and are either flat or have a flat part. They have irregular borders and often are variable shades of color, particularly brown. The presence of dysplastic nevi may mark a greater risk of malignant melanoma developing on apparently normal skin. **See picture #46 in booklet.**

Name the malignant, epithelial cell tumor that characteristically begins as a papule and enlarges peripherally, developing a central crater that erodes, crusts and bleeds. **An example of this tumor is shown below.**



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- **Basal Cell Carcinoma**

Metastasis is rare, but the local invasion by direct extension destroys the underlying and adjacent tissue. It frequently develops on the exposed surfaces of the skin, face and scalp in middle-aged or elderly persons. The primary cause of the cancer is excessive **exposure of the sun or to x-rays.**

Clinical features:
- Males are more affected than females
- 4th decade of life or older
- Located primarily on sun exposed areas of head and neck with the nose being the most common site
- **Risk factors:** childhood sun exposure, blistering sunburns, fair skin, blue eyes, blonde or red hair

Important: It is locally destructive and metastases are exceptionally rare.

The treatment for basal cell carcinoma is eradication of the lesion, often by electrodessication or cryotherapy. Recurrence is uncommon if properly treated.

Radiographically, **multiple myeloma** demonstrates:

- Multiple radiolucent / radiopaque lesions of the skull resembling "cotton ball" or "cotton wool" appearance
- Multiple "punched-out" radiolucencies in involved bones
- Bulbous crowns of teeth, obliteration of pulpal chambers and shortened roots
- Multiple, irregular "moth-eaten" radiolucencies of the jaws

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- **Multiple "punched-out" radiolucencies in involved bones**

Multiple myeloma *(also known as "Plasma Cell Myeloma")* is a primary malignant neoplasm of bone characterized by progressive destruction of the marrow with replacement by neoplastic **plasma cells.**

Clinical Features:

- **Men 2:1** → 40-70 years old. Vertebrae, ribs, and skull are most frequently involved; **pain in lumbar or thoracic region** is a common early symptom.
- Jaws are rarely a primary site, but become involved in 70% of the cases, **molar-ramus area most common site.** Symptoms include swelling, pain, loosening of the teeth, and paresthesia.

Radiographic features → variable; slight demineralization to extensive destruction, characteristic finding is multiple, small, discreet **"punched out"** radiolucencies in involved bones. In a patient suspected of having multiple myeloma, a **lateral skull** radiograph is best to confirm the diagnosis.
Laboratory findings → important in establishing diagnosis; **hypergammaglobulinemia** *(especially* **IgG** *in 70% of the cases).* **Bence Jones proteinuria** is present in 60-85% of the cases.
Treatment and Prognosis → chemotherapy, radiation; **prognosis poor** with median survival time 2-3 years.

Note: Plasmacytoma is a localized collection of monoclonal plasma cells. The disease is divided into primary plasmacytoma of the bone and extramedullary plasmacytoma. The importance of the diagnosis rests with the **potential** for these disorders to progress to multiple myeloma.

The most common type of **malignant melanoma** is:

- Superficial spreading
- Lentigo maligna
- Acral lentiginous
- Nodular

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• **Superficial spreading**

Malignant melanoma involves the cells *(melanocytes)* that produce pigment *(melanin)*, which is responsible for skin and hair color. Melanoma can spread very rapidly and is the most deadly form of skin cancer. It is the leading cause of death from skin disease. Melanoma may appear on normal skin, or it may begin at a mole *(nevus)* or other area that has changed in appearance. Some moles present at birth may develop into melanomas. The development of melanoma is related to sun exposure, particularly to sunburns during childhood, and is most common among people with fair skin, blue or green eyes, and red or blonde hair.

There are **four major types** of melanoma:
Superficial spreading melanoma is the **most common** type of melanoma. It is usually flat and irregular in shape and color, with varying shades of black and brown. It may occur at any age or site and is most common in Caucasians.
Nodular melanoma usually starts as a raised area that is dark blackish-blue or bluish red, although some lack color. **Poorest prognosis.**
Lentigo maligna melanoma usually occurs in the elderly. It is most common in sun-damaged skin on the face, neck, and arms. The abnormal skin areas are usually large, flat, and tan with intermixed areas of brown. Develops from pre-existing lentigo maligna **(Hutchinson freckle).**
Acral lentiginous melanoma is the **least common** form of melanoma. It usually occurs on the palms, soles, or under the nails and is more common in African Americans.

Important: This tumor **exhibits** either initial **radial** *(do not metastasize)* or **vertical** *(metastasis may occur)* growth patterns within the skin. Radial growth is characteristic of spreading types, and vertical growth is characteristic of nodular melanoma.
Note: Skin cancer is a very common malignancy in the United States. Of the different types, basal cell carcinoma is **most common** followed by squamous cell carcinoma and the least common is malignant melanoma.

When using the **TNM** method in assessing the prognosis and therapy of malignant neoplasms, the **N** represents:

- The presence of Nikolsky's sign
- The presence of nodules
- The presence of regional lymph node involvement
- The presence of erythroplakia

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- **The presence of regional lymph node involvement**

Clinical staging of malignant neoplasms:

The **TNM** method of assessing the prognosis and therapy of malignant neoplasms is based upon **1)** the size of the primary tumor, **2)** the presence of regional lymph node involvement and **3)** the presence of distant metastases. This is represented as:

- **T** = size of the primary tumor
- **N** = presence of regional lymph node involvement
- **M** = presence of distant metastases

The most common site of squamous cell carcinoma of the **tongue** is the:

- Dorsum
- Posterior one-third
- Anterior two-thirds
- Lateral border and ventral surface

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- **Lateral border and ventral surface**

The oral cavity is defined as the area extending from the vermilion border of the lips to a plane between the junction of the hard and soft palate superiorly and the circumvallate papillae of the tongue inferiorly. This region includes the buccal mucosa, upper and lower alveolar ridges, floor of the mouth, retromolar trigone, hard palate, and anterior two thirds of the tongue. The lips are the **most common site** of malignancy in the oral cavity and account for 12% of all head and neck cancers. Squamous cell carcinoma *(SCC)* is the most common histologic type, with 98% involving the **lower lip.** This predilection to the lower lip has been attributed to sun exposure. Next most common sites in order of frequency are the tongue, floor of the mouth, mandibular gingiva, buccal mucosa, hard palate, and maxillary gingiva. **Important:** SCC is the **most common malignancy** in the oral cavity.

Location	Incidence	Etiology	Clinical Characteristics	Treatment
Lip	**Most** common site	Race, complexion, sunlight, pipe smoking	Men over 60, **vermillion of lower lip,** painless ulcer, keratotic plaque	Surgery or irradiation
Tongue *(lateral border and ventral surface)*	**2nd** most common intraoral site	Tobacco, alcohol, syphilis, Plummer-Vinson syndrome	Men over 60, posterior lateral border and middle third, painless ulcer, leukoplakia, erythroplakia	Surgery or irradiation
Floor of mouth	**3rd** most common intraoral site	Tobacco, alcohol	Men 40-60 years old, painless ulcer, leukoplakia, erthroplakia	Surgery or irradiation
Buccal mucosa	10% of all oral carcinomas	Tobacco, alcohol, denture irritation	Painless ulcer, exophytic mass, leukoplakia	Surgery or irradiation
Gingiva and alveolar mucosa	10-15% of all oral carcinomas	Tobacco, alcohol	Men over 60, mandibular mucosa, painless ulcer, plaque-like or exophytic mass	Surgery

Of the following types of squamous cell carcinomas, which is the **least common?**

- Squamous cell carcinoma of the palate
- Squamous cell carcinoma of the nasopharynx
- Squamous cell carcinoma of the oropharynx
- Squamous cell carcinoma of the maxillary sinus

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- **Squamous cell carcinoma of the nasopharynx**

Location	Incidence	Etiology	Clinical Characteristics	Treatment
Nasopharynx	Less than 2% of all cancers in the U.S.	Tobacco, alcohol	Men between 30 and 40, roof or lateral wall is most common site, cervical mass, earache sore throat, nasal obstruction	Surgery and irradiation
Palate	10% of all oral carcinomas	Tobacco, alcohol, denture irritation	Men over 60, soft palate is more common than hard palate, painful ulcer, leukoplakia, exophytic mass	Surgery and irradiation
Oropharynx	10% of all head and neck cancers	Tobacco, alcohol	Men over 50, sore throat, dysphagia, painful ulcer, cervical mass	Surgery and irradiation
Maxillary sinus	30% of all head and neck cancers	Unknown	Men over 40, chronic sinusitis, **bulging of palate**, loosening of teeth, paresthesia in cheek	Surgery and irradiation

Melanomas develop initially as a flat phase **without competence for metastasis** called the:

- "Horizontal" growth phase
- "Circular" growth phase
- "Vertical" growth phase
- "Radial" growth phase

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• **"Radial" growth phase**

***This refers to the initial growth of a melanoma in a **horizontal plane.** It is clinically macular or only slightly elevated. They then may evolve focally an elevated part, the **vertical growth phase,** with metastatic competence.

Generally, the **radial growth phase** is described by the A, B, C, D's of melanoma:
A = asymmetry
B = border irregularity
C = color variability *(brown, black, blue, gray, pink)*
D = diameter of *(often)* greater than 1/4 inch

Radial growth phase melanomas, although invasive, have a cure rate that approaches 100% with surgery alone. Radial growth phase melanomas include the superficial spreading, lentigo maligna, and acral lentiginous melanomas.

The **"vertical"** growth phase is the phase that begins when neoplastic cells populate the underlying dermis. It is characterized clinically by an increase in size, a change in color, nodularity and, at times, ulceration. **Metastasis** is possible once the melanoma reaches this phase. Overall, patients with vertical growth phase melanomas have a cure rate of 70%. **Note:** This phase predominates in nodular melanoma.

Remember: Malignant melanoma is an uncommon neoplasm of the oral mucosa. It exhibits a definite predilection for the palate and the maxillary gingiva / alveolar ridge. Unfortunately, oral mucosa melanomas have a dismal prognosis. The five-year survival rate for such tumors is approximately 7%.

The lips are the most common site of malignancy in the oral cavity, with 98% involving the:

- Upper lip
- Philtrum
- Lower lip
- Corners

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• **Lower lip**

Cancer of the lips → the lips are the most common site of malignancy in the oral cavity, with 98% involving the **lower lip.** 90 to 98% of cancers of the lower lip occur in males. Chronic exposure to the sun and **pipe smoking** have been implicated in the etiology.

Cancer of the **tongue** causes more deaths than do malignant lesions in other regions of the head and neck. This has been attributed to the fact that it is a highly mobile organ that is **richly endowed with lymphatics and blood vessels** which facilitate metastases. It very rarely gives rise to skeletal metastasis.

Notes:

1. The **dorsum** of the tongue is the area least frequently involved.
2. Squamous cell carcinoma of the tongue commonly **metastasizes** to the cervical lymph nodes.

Cancer of the floor of the mouth → occurs most commonly in the **anterior segment** on either side of the midline, near the orifices of the salivary glands. Premalignant lesions of squamous epithelium most often occur here. **Note:** Prognosis is **very poor** for lesions found here.

Cancer of the buccal mucosa → generally occurs along the **plane of occlusion,** midway anteroposteriorly.

Cancer of the gingiva → is more common in the mandible than in the maxilla, and posterior sites are seen more frequently than anterior.

Burkitt's lymphoma is associated with the:

- Herpes virus
- Epstein-Barr virus
- Cytomegalovirus
- Human papillomavirus

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• **Epstein-Barr virus**

Burkitt's lymphoma is a high-grade, **non-Hodgkin's lymphoma** that is endemic in Africa and occurs only sporadically in North America. It is manifested most often as a large osteolytic lesion in the jaw *(African form)* or as an abdominal mass *(Non-African form).*

Burkitt's lymphoma is the first human cancer with **strong evidence** of a viral etiology. The Epstein-Barr virus *(a herpes-type virus)* has been isolated from cultures of tumor cells and patients with Burkitt's lymphoma have high titers of antibodies against EBV. Also, an antibody against a surface antigen on the tumor cells has been demonstrated.

Note: The Epstein-Barr virus is also associated with **infectious mononucleosis** and **oral hairy leukoplakia.**

<u>Two forms of Burkitt's lymphoma:</u>
1. **African** → younger *(mean age 3)*, male predominance, typically involves the jaws.
2. **Non-African** → older *(mean age 11),* no sex predilection, presents most often as an **abdominal mass.**

***Both forms are **histologically identical.**

Note: The jaw lesions usually present as expanding intraoral masses on the palate and gingiva. Lesions appear as soft tissue nodular masses and many are hemorrhagic. **See picture #37 in booklet. Radiographically,** there is a **moth-eaten**, poorly marginated destruction of bone. **See picture #38 in booklet.**

Radiographically, the most characteristic appearance in the jaws of a patient with **Ewing's sarcoma** is:

- Multiple radiolucent / radiopaque lesions resembling "cotton ball" or "cotton wool" appearance
- Multiple "punched-out" radiolucencies
- "Moth-eaten" destructive radiolucencies of medulla and erosion of the cortex with expansion
- Lytic lesion that may be ill-defined or sharply defined

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- **"Moth-eaten" destructive radiolucencies of medulla and erosion of the cortex with expansion**

Ewing's sarcoma is an uncommon, highly lethal, malignant neoplasm of bone of uncertain origin. The most common sites for Ewing's sarcoma are the pelvis, the thigh, and the trunk of the body. The peak ages are between 10 and 20.

Pain, usually of an intermittent nature, and **swelling** of the involved bone are often the earliest clinical signs and symptoms of Ewing's sarcoma. Fever, leukocytosis, raised ESR, and anemia are also present. **Histologically,** it is often difficult to distinguish this tumor from a neuroblastoma or a reticulum cell sarcoma, however, the cells of Ewing's sarcoma contain **glycogen.**

Note: When the jaws are involved, there is predilection for the **ramus of the mandible**. There is usually pain followed by rapid swelling and loosening of the teeth. **See picture #39 in booklet.**

Radiographically, the most characteristic appearance is that of a **moth-eaten, destructive radiolucency** of medulla and erosion of the cortex with expansion. A variable periosteal **"onion-skin"** reaction may also be seen. **See picture #40 in booklet.**

Important: The most common osseous malignancies are **osteosarcomas,** followed by chondrosarcomas, fibrosarcomas and Ewing's sarcoma.

Radiographically, the **giant cell tumor** produces a radiolucent lesion similar in appearance to the:

- Osteochondroma
- Central giant cell granuloma
- Odontogenic myxoma
- Osteoid osteomas

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• **Central giant cell granuloma**

The **giant cell tumor** is a bone tumor of multinucleated **giant cells** that resembles osteoclasts scattered in a matrix of spindle cells. It arises most frequently in the long bones, especially in the area of the knee joint. Most investigators regard the giant cell tumor as a distinct entity from the central giant cell granuloma, acknowledging the very rare occurrence of the giant cell tumor within the jaws. Neoplasms of this kind may be benign or malignant and may cause pain, functional disability, and in some cases, pathologic fracture. **Note:** Giant cell tumors have been noted in association with preexisting Paget's disease in both the jaws and long bones.

The **central giant cell granuloma** is an intraosseous destructive lesion of the anterior mandible and maxilla in which larger lesions expand the cortical plates, cause movement of teeth, and produce root resorption. It is composed of multinucleated giant cells.

Clinical features: 1) Common in children and young adults, **2)** More often in females than males, **3)** Mandible more often than maxilla, **4)** Slow growing will be asymptomatic swelling, **5)** Rapid growing will be painful and dentition will be loose.

Radiographic Features: 1) Unilocular, multilocular radiolucencies, **2)** Well defined or irregular borders, **3)** Expansile, **4)** Root resorption. **See picture #41 in booklet.**

Remember:

- An **osteochondroma** is a benign tumor of bone and cartilage.
- The **odontogenic myxoma** is a rare tumor of the jaw. It is most often seen in the mandible. The patients are usually under 35 years of age. It is slow growing and usually asymptomatic, but eventually leads to localized expansion of the jaw. The treatment is curettage.

Osteosarcomas involving the jaws present most commonly with:

- No symptoms
- Painless swelling and expansion
- Swelling and localized pain
- Bilateral, symmetrical enlargement of alveolar ridge

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• **Swelling and localized pain**

Osteosarcomas *(also called osteogenic sarcomas)* are the most common of the malignant neoplasms derived from bone cells that in the jaws exhibit radiographic widening of periodontal membrane of teeth and histologically exhibit a wide spectrum of findings, all of which contain atypical osteoblasts and abnormal bone or osteoid formation.

Clinical features:

• Between ages of 30 and 40, predilection for males *(62%)*
• Equal involvement of maxilla and mandible
• Firm, rapidly growing swelling and localized pain
• Some cases, there may be loosening and displacement of teeth as well as paresthesia

The histologic classification of an osteosarcoma is based on the **dominant type of tissue cell:**

- **Osteoblastic** → produces osteoid tissue
- **Chondroblastic** → produces cartilage tissue
- **Fibroblastic** → produces fibrous tissue

***A very important early radiographic feature of an **osteosarcoma of the jaw** is a **symmetrically widened PDL space** around one or more teeth.

Other **radiographic** features, depending on the degree of calcification:

- Sclerotic → excessive production of bone, **"sun-ray appearance". See picture #42 in booklet.**
- Lytic → irregular radiolucency
- Mixed → most osteosarcomas **have this appearance**

Important → for **osteosarcoma of the jaw,** the differential diagnosis should include:

- Chrondrosarcoma
- Metastatic carcinoma and Pindborg tumor
- Ossifying subperiosteal hemangioma
- Peripheral odontogenic fibroma
- Scleroderma
- Chronic osteomyelitis

The most common neoplasm seen in **AIDS** patients is:

- Non-Hodgkin's lymphoma
- Squamous cell carcinoma
- Basal cell carcinoma
- Kaposi's sarcoma

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• **Kaposi's sarcoma**

Kaposi's sarcoma is a unique form of angiosarcoma and is the most common cancer associated with AIDS patients and has a predilection for the palate.

Four clinical presentations:
- Classic
- Endemic *(African)*
- Immunosuppression associated
- AIDS-related

Kaposi's sarcoma is a malignant neoplasm originating in the skin. It is characterized by **abnormal vascular proliferation** *(it is a cancer of the lining of blood vessels)*. It occurs on multiple sites, especially the lower extremities. Initial lesions are small, red papules, which enlarge and fuse to form purple-to-brown, spongy nodules. It spreads to lymph nodes and internal organs.

Note: Intraorally, the **hard palate** is the most common location, followed by the gingiva and buccal mucosa.

Remember: AIDS is caused by the RNA retrovirus, **HIV** *(also called HTLV-III)*. The HIV infection is acquired with IV drug use as well as by sexual contact *(homosexual and heterosexual)* and contaminated blood products.

A **poorly differentiated** squamous cell carcinoma involving lymphoid tissue in the region of the tonsils and nasopharynx is called:

- A lipoma
- A lymphoepithelioma
- A lymphoma
- A nasopharynioma

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• A lymphoepithelioma

It has a high frequency among young adults of East Asian extraction. The primary lesion is usually very small, often completely hidden. **Swelling of the lymph node** is the most common presenting symptom, followed by a sore throat, nasal obstruction, bloody nose and headache.

The **lymphoepithelioma** is composed of cells *(squamous or undifferentiated),* with a slight to moderate amount of fibrous stroma that contains numerous lymphocytes. **Most importantly,** this neoplasm shows **metastasis at an early stage** to the cervical lymph nodes.

The treatment of choice is x-ray radiation, **however**, the complicating factor lies in the relative inability to treat the widespread metastases in the various organs. The prognosis is poor, with a 30% five-year survival rate.

All of the following statements concerning **metastatic tumors of the jaws** are true **except:**

- They may be completely asymptomatic
- The patient is usually aware of slight discomfort or pain
- The maxilla is affected far more frequently than the mandible
- The molar region is predominantly involved

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• **The maxilla is affected far more frequently than the mandible**

***This is **false;** the mandible is affected far more frequently than the maxilla.

The most common malignancy affecting skeletal bones is **metastatic carcinoma.** However, metastatic disease to the mandible and maxilla is unusual *(only about 1%).* **Most importantly,** a tumor of the jaws may be the first evidence of dissemination of a known tumor from its primary site.

Note: Metastases to the jaws **most commonly** originate from primary carcinomas of the **breast,** kidney, lung, colon, prostate and thyroid. They are **least likely** to have originated in the brain.

Clinical features of metastatic jaw lesions:

- They may be completely asymptomatic
- Usually, however, there is paresthesia or anesthesia of the lip or chin due to involvement of the mandibular nerve
- Teeth in the area are loose or extruded
- There can be swelling or expansion of the jaw
- Appears as an asymptomatic radiolucency

Explain what is meant by a carcinoma of the oral cavity having the following **TNM** designation:

T1, N2, M1

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Clinical Staging of Carcinoma of the Oral Cavity

- **T = Size** of the primary tumor
  - TX: Primary tumor can not be assessed
  - TO: No evidence of tumor
  - Tis: Carcinoma in situ
  - **T1: less than 2 cm in greatest diameter**
  - T2: 4 cm in greatest diameter
  - T3: greater than 4 cm in greatest diameter
- **N =** Regional **lymph node** involvement
  - NX: Regional lymph nodes can not be assessed
  - NO: No clinically palpable lymph nodes, or lymph nodes palpable but metastases not suspected
  - N1: Palpable homolateral lymph node(s), not fixed but metastases suspected
  - **N2: Palpable centralateral/bilateral lymph node(s), not fixed but metastases suspected**
  - N3: Palpable lymph node(s), fixed metastases suspected
- **M = Distant** metastasis
  - MX: Presence of distant metastasis can not be assessed
  - MO: No distant metastasis
  - **M1: Clinical and / or radiographic evidence of metastasis other than regional lymph nodes**

Verrucous Carcinoma *(Snuff Dipper's Cancer)* is a distinct, diffuse, papillary, superficial, non-metastasizing form of well-differentiated:

- Basal cell carcinoma
- Squamous cell carcinoma
- Malignant melanoma
- Osteogenic sarcoma

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- **Squamous cell carcinoma**

A **verrucous carcinoma** is a well-differentiated squamous cell neoplasm of soft tissue of the oral or laryngeal cavity. The lesion may invade or infiltrate the borders of adjacent structures **but it rarely metastasizes.** Verrucous carcinoma may transform into an invasive form of carcinoma or coexist with other squamous cell carcinomas. It is often misdiagnosed histologically as a benign lesion.

Clinical Features:

- Male predilection
- Broad-based, exophytic, indurated lesion
- Diffuse, whitish, cauliflower or coral papillary mass
- Mandibular mucobuccal fold, alveolar mucosa and palate
- Slow-growing, continuous enlargement
- Painless

See picture #43 in booklet.

Squamous cell carcinoma is the most common type of oral cancer, accounting for more than:

- 45% of all malignant neoplasms of the oral cavity
- 65% of all malignant neoplasms of the oral cavity
- 75% of all malignant neoplasms of the oral cavity
- 90% of all malignant neoplasms of the oral cavity

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- **90% of all malignant neoplasms of the oral cavity**

Squamous cell carcinoma is a malignant neoplasm of stratified squamous epithelium that is capable of locally destructive growth and distant metastasis. It is the **most common type of oral cancer,** accounting for over 90% of all malignant neoplasms of the oral cavity. It is two times more prevalent in males *(40-65 years of age)*. **See pictures #44 and #45 in booklet.**

Possible sites:
- Lower lip *(most common site)*
- Tongue *(lateral border and ventral surface most common location, dorsum least common location)*
- Floor of the mouth *(least favorable prognosis)*
- Soft palate *(uncommon)*
- Gingiva / alveolar ridge
- Buccal mucosa

Clinical features:
- Early presentation of leukoplakias and erythroplakias
- Painless ulcer, tumorous mass, or verrucous *(papillary growth)*
- Occasional loosening or loss of teeth
- Possible paresthesia of the teeth and lower lip

Risk factors identified include **smoking**, alcohol consumption, painful and ill-fitting dentures, chronic inflammation and the use of **smokeless tobacco. Important:** Tobacco use is the **primary** risk factor.

Remember: The most reliable histologic criterion for a diagnosis of oral squamous cell carcinoma is **invasion.**

The most common cause of facial weakness which comes on suddenly is referred to as:

- Polio
- Bell's palsy
- Tic douloureux
- Multiple sclerosis

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• **Bell's Palsy**

Bell's palsy is a form of facial paralysis resulting from damage to the facial nerve. It can strike at any age; **however**, it disproportionately attacks pregnant women and people who have diabetes, influenza, a cold, or some other upper respiratory ailment.

Clinical signs include a unilateral paralysis of all facial muscles with loss of eyebrow and forehead wrinkles, drooping of the eyebrows, flattening of the nasiolabial furrow, sagging of the corner of the mouth and the inability to frown or raise the eyebrows. The upper and lower lips may also be paralyzed on the side affected.

After its sudden onset the paralysis begins to subside within two or three weeks, and gradual, complete recovery occurs in over 85% of patients.

Triggering events related to Bell's palsy are acute otitis media, atmospheric pressure change, exposure to cold, ischemia of the facial nerve near the stylomastoid foramen, Melkersson-Rosenthal syndrome, and multiple sclerosis. **Note:** Melkersson-Rosenthal syndrome is the term used when cheilitis occurs with facial palsy and plicated tongue.

Remember: While attempting to give an inferior alveolar nerve block, if you inject the anesthetic solution into the capsule of the parotid gland, you may cause a Bell's palsy like feeling for the patient by anesthetizing the facial nerve.

The **prime factor** that initiates the myofascial pain-dysfunction syndrome *(MPDS)* is:

- Trauma
- Muscle spasm
- Periodontal disease
- Tumor

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• **Muscle spasm**

Such spasm may arise in one of three ways: muscular overextension, muscular overcontraction or muscle fatigue. The most frequent cause of the spasm seems to be **muscle fatigue.** This syndrome is seen predominantly in women, usually in the 20 to 40 age range, and generally occurs unilaterally.

There are four cardinal signs and symptoms of the syndrome:
1. Pain
2. Muscle tenderness
3. A clicking or popping noise in the TMJ
4. Limitation in jaw motion *(especially in the morning)*

The pain itself is usually **unilateral** and is described as a dull ache in the ear or preauricular area, which may radiate to the angle of the mandible, temporal area or lateral cervical area. The muscle most apt to exhibit tenderness is the **lateral pterygoid** muscle.

Note: There are **no radiographic** findings associated with MPD.

Treatment: Most cases are self-limiting. Soft diet, limited talking, no gum chewing, moist heat, NSAID's and Diazepam help relieve symptoms.

Which of the following is described as sharp, jabbing, electric, or shock-like pain located deep in the throat on one side?

- Postherpetic neuralgia
- Orolingual paresthesia
- Frey's syndrome
- Glossopharyngeal neuralgia

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• **Glossopharyngeal neuralgia**

Glossopharyngeal neuralgia refers to pain similar to that of trigeminal neuralgia, which arises from the glossopharyngeal nerve *(CN IX)*. It is not **as common** as trigeminal neuralgia, but the pain may be as severe when it does occur. It occurs in both sexes, in middle-aged or older persons and is described as sharp, jabbing, electric, or shock-like pain located deep in the throat on one side. It is generally located near the tonsil although the pain may extend deep into the ear. It is usually triggered by swallowing or chewing. It is almost always **unilateral.**

Frey's syndrome *(also called the auriculotemporal syndrome)* is an unusual phenomenon, which arises as a result of damage to the auriculotemporal nerve and subsequent reinnervation of the sweat glands by parasympathetic salivary fibers.The syndrome follows some surgical operation such as removal of a parotid tumor or ramus of the mandible, or an infection of the parotid that has damaged the auriculotemporal nerve *(a branch of V-3)*.
Important: Gustatory sweating is the chief symptom of this syndrome. The patient typically exhibits flushing and sweating of the involved side of the face during eating. This syndrome is not a common condition.

Postherpetic neuralgia is a persistent burning, aching, itching and hyperesthesia along distribution of a cutaneous nerve following an attack of **herpes zoster.** It may last for a few weeks or many months. Involvement of the **facial nerve and geniculate ganglion** produces the **Ramsey Hunt Syndrome**, which is characterized by facial paralysis and otalgia *(earache)*.

Which of the following is a relatively rare autoimmune disorder of peripheral nerves in which antibodies form against acetylcholine *(ACh)* nicotinic postsynaptic receptors at the myoneural junction?

- Myasthenia gravis
- Myelofibrosis
- Multiple sclerosis
- Graves' disease

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• **Myasthenia gravis**

Myasthenia gravis is an autoimmune disorder in which antibodies form against acetylcholine *(ACh)* nicotinic postsynaptic receptors at the myoneural junction. The muscles are quickly fatigued with repetitive use. It is typical for a myasthenic patient to have a flattened smile and droopy eyes, with slow papillary light responses. **Xerostomia and rampant caries** may accompany myasthenia gravis. The acetylcholine that is necessary for the proper transmission of nerve impulse is destroyed, with the result that salivary glands **do not** receive adequate stimulation. **Note:** Head and neck manifestations include inability to focus eyes, drooping eyelids, double vision, difficulty in chewing and swallowing, and slurring of words.

Multiple sclerosis is a chronic, often disabling disease that randomly attacks the CNS *(brain and spinal cord)*. It is believed to be due to an autoimmune response in which the immune system attacks a person's own tissue. Twice as many women as men have MS, with the onset of symptoms occurring most often between the ages of 20 and 40. Symptoms may range from tingling and numbness to paralysis and blindness. Patients with multiple sclerosis sometimes have facial and jaw weakness. In addition, both **Bell's palsy and trigeminal neuralgia** may develop more frequently in patients with MS.

Trigeminal neuralgia, also known as tic douloureux, is a pain syndrome recognizable by patient history alone. The condition is characterized by pain often accompanied by a brief facial spasm or "tic". Pain distribution is:

- Bilateral and follows the sensory distribution of Cranial Nerve VII
- Unilateral and follows the sensory distribution of Cranial Nerve VII
- Unilateral and follows the sensory distribution of Cranial Nerve V
- Bilateral and follows the sensory distribution of Cranial Nerve VII

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• **Unilateral and follows the sensory distribution of Cranial Nerve V**

Trigeminal neuralgia is an excruciating painful illness in which the afflicted feels **sudden stab-like pains** in the face. The pains usually last only moments, but are among the most severe pains humans can feel. The pain is provoked by touching a **"trigger zone",** typically near the nose or mouth. It is caused by degeneration of the **trigeminal nerve** or by pressure being applied to it. Pain distribution is unilateral and follows the sensory distribution of cranial nerve V, typically radiating to the maxillary *(V-2)* or mandibular *(V-3)* area. At times, both distributions are affected. The momentary bursts of pain recur in clusters, lasting many seconds. Paroxysmal episodes of the pains may last for hours.

Necessary criteria for diagnosis of trigeminal neuralgia:

- Onset of pain initiated by a trigger point
- Pain extreme, paroxysmal, lancinating
- Duration is less than 2 minutes
- A refractory period experienced for several minutes after attack
- Pain limited to known distribution of one or more branches of trigeminal nerve with no motor deficit in that area
- Pain diminished with use of carbamazepine
- Spontaneous remissions occur lasting more than six months during the early phase of the disease

The drug of choice for treating trigeminal neuralgia is **carbamazepine** *(Tegretol)*. It is an analgesic and anticonvulsant. It is also prescribed in the treatment of certain seizure disorders. When used for the treatment of trigeminal neuralgia, it usually relieves the pain within 48 hours after treatment is started.

Which of the following are typically within **soft tissue** and may mimic inflammatory lesions of odontogenic origin?

- Median alveolar cyst
- Globulomaxillary cyst
- Nasiolabial cyst
- Nasopalatine cyst

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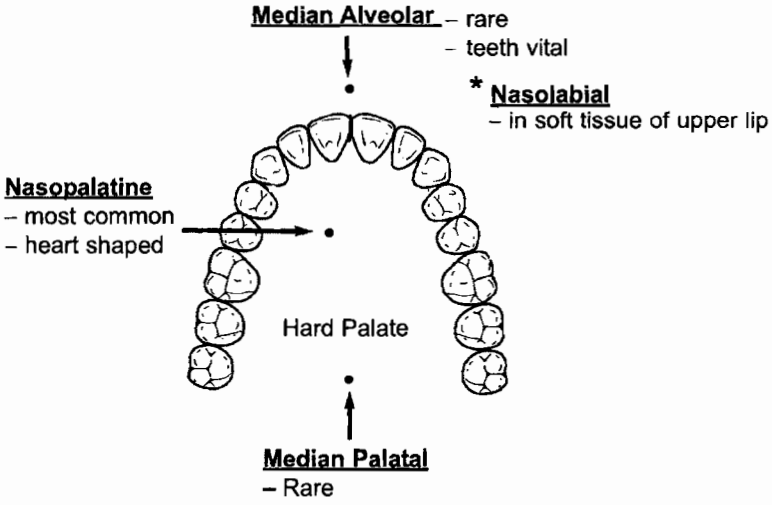
• **Nasolabial cyst** → also called nasoalveolar cyst

Because this cyst is extraosseous, it is **not likely to be seen on a radiograph.**

Quick reference for developmental cysts:



All of the following cysts are **congenital except:**

- Thyroglossal duct cyst
- Branchiogenic cyst
- Globulomaxillary cyst
- Dermoid cyst

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- **Globulomaxillary cyst**

Congenital Cysts:

- The **thyroglossal duct** cyst, which may arise from any portion of the thyroglossal duct. This cyst is therefore found in a **midline position** and is usually dark in color. It may be vascular as to resemble a hemangioma. One frequent important symptom is **hemorrhage into the mouth,** resulting from the rupture of the overlying veins. Complete excision of the tract to the base of the tongue, frequently including a portion of the hyoid bone, is necessary for a cure.

- The **branchiogenic cyst,** which arises from the persistence of the second branchial arch cleft. This cyst is characteristically located along the **anterior border of the sternocleidomastoid muscle** at any level in the neck. This type of cyst is lined with ciliated and stratified squamous epithelium and contains a milky or mucoid fluid. The treatment consists of complete surgical excision.

- The **dermoid cyst** is relatively uncommon in the oral cavity. This cyst frequently contains hair, sebaceous and sweat glands, as well as tooth structures. The most common site is the **floor of the mouth.** The treatment is the surgical removal of the entire tumor.

Important: Globulomaxillary cysts are thought to develop from epithelial remnants remaining following joining of the globular portion of median nasal process with the maxillary process. They are characterized by a large **"pear-shaped"** radiolucency between the maxillary lateral incisor and cuspid. They are asymptomatic, **all regional teeth are vital.** They tend to cause divergence of the roots. **However,** there is considerable controversy as to whether this cyst actually exists. Many of these are, in reality apical cysts associated with **non-vital lateral incisors.** Often these apical cysts have a tendency to extend between the two teeth to simulate a globulomaxillary cyst.

All of the following cysts are **developmental** *(or fissural)* **except:**

- Nasopalatine cyst
- Nasolabial cyst
- Branchiogenic cyst
- Median palatal cyst
- Median alveolar cyst

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- **Branchiogenic cyst**

Developmental Cysts:

- **Nasopalatine** → "heart-shaped" radiolucency in midline, **most frequent** type of non odontogenic cyst. Usually asymptomatic or may produce an elevation in the anterior part of the palate. **Teeth are vital.** Treatment is enucleation.

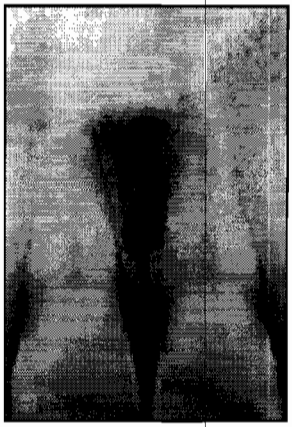
- **Nasolabial** *(nasoalveolar)* → is superficially located in soft tissues of the upper lip. This is an **extraosseous** cyst. Treatment is surgical excision.

- **Median palatal** → rare, may occur anywhere along median palatal raphe. May produce swelling on palate. Treatment is enucleation.

- **Median alveolar** → rare, occurs in bony alveolus between central incisors. Distinguished from periapical cyst by the fact that **adjacent teeth are vital.** Treatment is enucleation.

Remember: The **globulomaxillary** cyst was originally believed to be a developmental *(fissural)* cyst. The developmental origin has been disputed. Today, the literature almost uniformly agrees that the entity of globulomaxillary cyst should only be used as a clinical descriptive name. According to the literature, the majority of the lesions *(over 80%)* presenting with the radiographic features of a globulomaxillary cyst are of periapical origin. The evidence in the literature is in favor of this lesion being predominantly of tooth origin.

The **circular radiolucent** area seen in this radiograph is clinically seen as a **marked swelling** in the region of the palatine papilla. It is situated distal to the roots of the central incisors. The pulps of the anterior teeth in this patient tested **vital**. These findings would be compatible with what **diagnosis?**



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- **Nasopalatine duct cyst** → also known as an incisive canal cyst

Of the cysts of the jaw, those that arise from epithelial remnants in the incisive canal are the **most common type** of maxillary developmental cyst. Histologically, this cyst is lined with vessels, nerves and mucous glands in the wall. They most often remain limited as to size and are **asymptomatic**. Some of them, however, become infected or show a tendency to grow extensively. When this occurs, surgical intervention is indicated.

Note: The **soft tissue** *(and far less common)* **variant** of the nasopalatine canal cyst is the cyst of the **palatine papilla**.

When making a diagnosis of this cyst, the following **two cysts** should be **ruled out:**

- The **globulomaxillary cyst** → usually appears between the roots of the lateral incisor and those of the canine. It is **"pear-shaped"** and often causes the roots of involved teeth to diverge. **See picture #47 in booklet.**

- The **median palatal cyst** → usually situated in the midline of the hard palate, posterior to the premaxilla. Clinically, this lesion presents as a firm swelling, which is usually painless. **Note:** Some investigators now believe that this cyst represents a **more posterior** presentation of a nasopalatine duct cyst, rather than a separate cystic degeneration of epithelial rests at the line of fusion of the palatine shelves. **See picture #48 in booklet.**

The **most common** developmental *(or fissural)* cyst is:

- The nasopalatine duct cyst
- The median palatal cyst
- The median alveolar cyst
- The nasiolabial cyst

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- **The nasopalatine duct cyst**

Fissural Cyst	Histogenesis	Clinical Characteristics	Radiographic Characteristics	Treatment and Prognosis
Nasopalatine duct	Remnants of nasopalatine ducts *(within bone)*	Most common fissural cyst: usually asymptomatic. May complain of tender swelling of palate	Well-demarcated round, oval or heart shaped radiolucency between and above maxillary central incisors; rarely just lateral to midline; lesion crosses midline; teeth **vital**	Surgical excision, prognosis excellent, don't confuse with enlarged palatine foramen
Median palatal	Epithelial remnants in line of fusion between palatine processes	Soft fluctuant or crepitant swelling in **midline of** hard palate	Well-demarcated radiolucency	Enucleation; prognosis excellent
Nasolabial *(nasoalveolar)*	Epithelial remnants from the inferior and anterior portion of the nasolacrimal duct	Swelling just below or inside nostril. Soft tissue cyst	Not visible, but may produce "cupping" of underlying bone. Not within bone	Enucleation; prognosis excellent

A **neurilemoma** *(schwannoma)* is a well-demarcated, benign lesion consisting of a fibroblastic proliferation of the nerve sheath cell *(Schwann cell)*. It is most frequently located on the:

- Palate
- Alveolar ridge
- Tongue
- Buccal mucosa

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• Tongue

Tumor	Etiology	Clinical Character	Treatment and Prognosis
Traumatic Neuroma	Trauma to a peripheral nerve	Most common site over mental foramen in edentulous mouths; nodule or swelling, which may be painful to digital pressure	Excision with small proximal portion of involved nerve; recurrence uncommon
Neurilemoma *(Schwannoma)*	It is derived from proliferation of **Schwann cells** of the neurolemma that surrounds peripheral nerves	Encapsulated mass that presents as an asymptomatic lump. The **tongue** is the most common location. Bony lesions may cause pain or paresthesia.	Conservative excision; recurrence rare
Neurofibroma	Some investigators say it is derived from the Schwann cell;	**Two forms:** 1. **Solitary neurofibroma** — asymptomatic nodule, occurs on tongue, buccal mucosa and vestibule 2. **Multiple** lesions as part of the syndrome neurofibromatosis	1. **Solitary:** surgical excision 2. **Neurofibromatosis:** removal is impractical. Watch for high rate of malignant transformation
Palisaded Encapsulated Neuroma	Unknown	Sessile, smooth– surfaced nodule of less than 1 cm in diameter. Predilection for the face, with nose and cheek most common	Local surgical excision: recurrence rare

Albright's syndrome is characterized by all of the following **except:**

- Heart disease
- Polyostotic fibrous dysplasia
- Café-au-lait spots
- Endocrine dysfunction

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• **Heart disease**

Albright's syndrome *(also called* **McCune-Albright** *syndrome)* is the most severe form of polyostotic fibrous dysplasia. It affects young people *(males and females equally)*. It is characterized by Café-au-lait spot on the skin, and endocrine abnormalities *(the most common of which is precocious sexual development in females)*. The extent to which each of these problems exist in those with the syndrome is quite variable. The hallmark of Albright's syndrome is **premature puberty in the female.** Early sexual development in the male is less common than the female.

Clinical features:

- Early childhood
- Multiple, slow-growing, painless expansile bone lesions confined to the craniofacial area or throughout the skeleton
- Endrocrine manifestations; in females often **sexual precocity**
- Irregular shaped Café-au-lait spots on the torso and sometimes intraorally
- Disfiguring
- Increased level of serum alkaline phosphatase

***Pathologic fractures** are frequently associated with this syndrome

There is no specific treatment for this syndrome. Drugs that inhibit estrogen production, such as testolactone, have been tried with some success.

Important: An additional complication is the **malignant transformation potential** of both the polyostotic *(mainly)* and monostotic fibrous dysplasia into **osteosarcomas.**

An 8-year-old child has complained of pain on the left side of his head for 5 weeks. There are no abnormal findings on physical examination. A skull radiograph reveals a solitary destructive bony lesion of the left mastoid region. A biopsy is performed and microscopic examination shows that this lesion is composed histologically of histiocytes, eosinophils, plasma cells, and lymphocytes. Which of the following is the **most likely** diagnosis?

- Osteoid osteoma
- Letterer-Siwe syndrome
- Eosinophilic granuloma
- Osteitis fibrosa cystica

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• **Eosinophilic granuloma**

Histiocytosis X is a generic name for a group of disorders *(Eosinophilic Granuloma, Hand-Schuller-Christian disease and Letterer-Siwe disease)* characterized by an abnormal increase in the number of certain immune cells called **histiocyte cells.** These include **monocytes, macrophages, and dendritic cells.**

Clinical Features:

- Acute disseminated ***(Letterer-Siwe)*** disease
  - aggressive
  - infants or young children
  - widespread disease: skin and mucosal lesions, visceral, and bone involvement
- Chronic disseminated ***(Hand-Schuller-Christian)*** disease
  - exophthalmos, diabetes insipidus, lytic skull lesions → **characteristic triad**
  - hepatosplenomegaly, skin and mucosal lesions *Oral signs include bad breath, sore mouth, and loose teeth
- Focal chronic ***(Eosinophilic Granuloma)*** disease
  - solitary localized bone destruction
  - swelling and pain
  - looks like a **periodontal or apical lesion**

Eosinophilic granuloma is the most **benign** form of Histiocytosis X. It may be totally asymptomatic, however, there may be local pain or swelling particularly if fracture has occurred. In the mouth, the **mandible** is most likely affected, with teeth on the affected side being loose with signs of gingivitis. When the lungs are affected, the symptoms can include coughing, shortness of breath, fever, and weight loss. Pneumothorax is a common complication.

Radiographically, the lesions appear as irregular radiolucent areas usually involving superficial alveolar bone. Lesions in the jaw usually appear as single or multiple radiolucencies, which may be so well-circumscribed as to resemble cysts or periapical granulomas.

The **central giant cell granuloma** is a non-neoplastic process that can occasionally behave in a very aggressive and expansile manner, destroying bone and displacing teeth. Over 70% of cases occur:

- In the maxilla anterior to the first molar tooth
- In the maxilla posterior to the first molar tooth
- In the mandible anterior to the first molar tooth
- In the mandible posterior to the first molar tooth

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• **In the mandible anterior to the first molar tooth**

The **central giant cell granuloma** is a benign process that occurs almost exclusively **within the jawbones.** It may be caused by trauma from a fall or blow, or even a tooth extraction. Over 60% of central giant cell granuloma cases occur in patients younger than 30 years of age, with twice as many occurrences in females as in males. Central giant cell granuloma is classified into **aggressive** and **non-aggressive** types; the aggressive type tends to occur in younger patients and is known to cause disfiguration, especially after surgery.

Either jaw may be involved, but the **mandible** is affected more often. Over 70% of cases occur in the mandible anterior to the first molar tooth. Pain is not a prominent feature of this lesion. Slight to moderate bulging of the jaw due to expansion of the cortical plates occurs in the involved area, depending upon the extent of bone involvement. The radiographic feature of this granuloma consists of a multilocular radiolucency of bone. The margins are well-defined.
Note: It appears similar to an **ameloblastoma** and an **odontogenic keratocyst.**

Histologically, the central giant cell granuloma is made up of loose fibrillar connective tissue. **Multinucleated giant cells** are prominent throughout the connective tissue.

The usual treatment is surgery, however, treatment alternatives to surgery have emerged with successful results ranging from steroid injections to calcitonin injections or nasal spray to interferon alpha-2a injections, which are administered 2-3 times per week for several months.

See picture #41 in booklet.

Condylar hyperplasia is an idiopathic disease characterized by a:

- Progressive, unilateral overgrowth of the mandible. The chin is deviated towards the unaffected side
- Progressive, bilateral overgrowth of the mandible. The chin is deviated towards the unaffected side
- Progressive, unilateral overgrowth of the mandible. The chin is deviated towards the affected side
- Progressive, bilateral overgrowth of the mandible. The chin is deviated towards the affected side

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- **Progressive, unilateral overgrowth of the mandible. The chin is deviated towards the unaffected side**

Congenital and developmental anomalies of the temporomandibular joint, although relatively rare, are important to identify early to re-establish normal midface growth centers. The more common entities include condylar agenesis, condylar hypoplasia, and condylar hyperplasia.

Condylar agenesis is the absence of all or portions of the coronoid process, condylar process, ramus and mandibular body. Other first and second arch abnormalities are commonly seen. Early treatment is indicated to limit the degree of deformity, with the primary objective being to re-establish the condylar growth center. This is best done with a costochondral graft with or without orthodontic surgery and facial plastic augmentation.

Condylar hypoplasia may be congenital, but is usually the result of trauma or infection. The most common facial deformity is shortness of the mandible with deviation of the chin **towards the affected side.** Treatment of the child involves the placement of a costochondral graft. In the adult, treatment involves either shortening of the normal side or lengthening of the involved side. Both result in an acceptable cosmetic and functional result. Orthodontic therapy is necessary in all cases to establish proper occlusion.

Condylar hyperplasia is an idiopathic disease characterized by a progressive, unilateral overgrowth of the mandible. The chin is deviated **towards the unaffected side.** Presentation is common in the second decade. Radiographic findings are usually a normal condyle but an elongated neck. Treatment depends on whether the condyle is still growing. Its growth is occurring, condylectomy is the treatment. If growth has ceased, orthognathic surgery is performed.

The bones affected by **fibrous dysplasia** usually have a characteristic appearance on X-ray. Which of the following is true.

- Radiographically, the lesions are usually radiolucent, well-circumscribed, and may have a "cotton wool" appearance
- Radiographically, the lesions are usually a saucer-shaped radiolucency
- Radiographically, the lesions are usually radiopaque, not well-circumscribed, and may have a "ground-glass" appearance
- Radiographically, the lesions are usually well-demarcated unilocular or multilocular radiolucencies

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- **Radiographically, the lesions are usually radiopaque, not well-circumscribed, and may have a "ground-glass" appearance**

Fibrous dysplasia is an asymptomatic regional alteration of bone in which the normal architecture is replaced by fibrous tissue and nonfunctional trabeculae-like osseous structures; lesions may be monostotic or polyostotic, with or without associated endocrine disturbances. **See picture #49 in booklet**.

Clinical features:
- Most often juveniles and young adults
- Singular, slow-growing, painless swelling of either jaw
- Overgrowth of tissue that occurs centrally in the jaws
- May involve impacted or unerupted teeth
- Increased level of serum alkaline phosphatase

Classification:
- **Monostotic:** Most common form *(80%)*. Only one bone affected
- **Polyostotic:** numerous bones affected
- **Craniofacial form:** occurs in 10-25% of patients with the monostotic form and in 50% with the polyostotic form. Sites of involvement most commonly include the frontal, sphenoid, maxillary, and ethmoidal bones. The occipital and temporal bones are less commonly affected.
- **Albright's syndrome:** The combination of polyostotic fibrous dysplasia, hormonal disturbances, and skin pigmentation *(café-au-lait spots)*.
- **Jaffe syndrome:** A syndrome characterized by polyostotic fibrous dysplasia and café-au-lait spots.

Mandibular tori most often appear:

- In the retromolar pad region
- On the lingual surface of the mandible, most often in the premolar region
- On the lingual surface of the mandible, inferior to the mylohyoid ridge
- Along the midline of the hard palate

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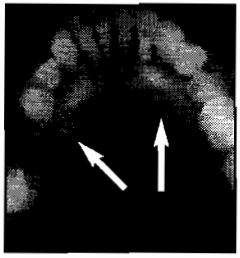
• **On the lingual surface of the mandible, most often in the premolar region**

Mandibular tori *(also called torus mandbularis)* are bony, exophytic growths that occur along the lingual surface of the mandible superior to the mylohyoid ridge.

Mandibular tori may occur singly, however, there is a marked tendency toward **bilateral occurrence,** and the lesion is not necessarily confined to the premolar region. Unlike palatal tori, the mandibular tori are more readily demonstrated radiographically.

Tori *(maxillary and mandibular)* are of no pathological significance and rarely are they of clinical significance while the normal teeth are still present. If, **however**, a complete denture needs to be made, they should be carefully removed.

Palatal tori



Mandibular tori

All of the following are clinical features of the **central ossifying fibroma** except:

- Slow growing expansile lesion
- More often in maxilla
- Asymptomatic
- Common in young adults around 35 years of age

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- **More often in maxilla**

***This is **false;** it occurs more often in the mandible.

The **central ossifying fibroma** is a benign neoplasm of bone origin which presents as a **well-demarcated** complete radiolucency to a mixed radiolucent / radiopaque mass with a peripheral radiolucent rim. It is a slow-growing, expansile lesion with characteristic downward expansion of the inferior border of the mandible. It is common in young adults around 35 years of age and is five times more likely to occur in females than males. It affects the **posterior mandible** in about 90% of cases.

This neoplasm presents an extremely variable radiographic appearance depending upon its stage of development. However, despite the stage of development, the lesion is **always well-circumscribed** and demarcated from surrounding bone **(See picture #85 in booklet),** in contrast to fibrous dysplasia. In its early stage it appears as a radiolucent area. As the lesion matures, it eventually becomes a relatively uniform radiopaque mass. It most commonly involves only **one bone** *(as opposed to* **Paget's disease***)*. **Note:** Treatment should be directed toward complete removal of the mass, using enucleation or surgical resection with bone grafting. The prognosis is known to be fair, and recurrence after surgical removal seems to be unusual. Radiotherapy is contraindicated because of its radioresistance and post-radiation complications.

There is a remarkable similarity in clinical features between this lesion and the **central cementifying fibroma**, a tumor accepted by most investigators as being odontogenic in origin. It has been suggested that these are two separate benign tumors, identical in nature **except** for the cell undergoing proliferation; the osteoblast with bone formation in one case, or the cementoblast with cementum formation in the other case.

Gardner's syndrome is a rare autosomal dominant disease characterized by all of the following **except:**

- GI polyps
- Café-au-lait spots on the skin
- Multiple osteomas
- Soft tissue tumors

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• **Café-au-lait spots on the skin**

Gardner's syndrome is a polyposis syndrome *(as are Familial multiple polyposis, Peutz-Jeghers syndrome and Turcot's syndrome).* The most serious complication of Gardner's syndrome is the multiple polyps that affect the large intestine. The inevitable outcome of this disease is **colon cancer.**

Clinical features:
- Onset early puberty
- Polyps of the colon ultimately change into **adenocarcinoma** by the fourth decade of life
- Abnormality of the retina of the eye
- Development of multiple epidermal cysts usually on face, scalp, and extremities

The oral findings of Gardner's syndrome include:
- Multiple impacted and supernumerary teeth
- Multiple jaw osteomas which give a **"cotton-wool"** appearance to the jaws. These osteomas appear as dense, well-circumscribed radiopacities. Osteomas most often develop first within the angle of the mandible.
- Multiple odontomas

***When Gardner's syndrome is suspected **based on oral findings,** the patient should be referred to a gastroenterologist for consultation. **Note:** Multiple desmoid tumors *(fibromatosis)* and epidermoid cysts of the skin are also characteristic of the disease.

Remember: Multiple impacted and supernumerary teeth are also seen in **Cleidocranial dysplasia.**

The most common form of **fibrous dysplasia** is:

- Monostotic
- Polyostotic
- Albright's syndrome
- Jaffe syndrome

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• **Monostotic**

Fibrous dysplasia is an asymptomatic regional alteration of bone in which the normal architecture is replaced by fibrous tissue and nonfunctional trabeculae-like osseous structures; lesions may be monostotic or polyostotic, with or without associated endocrine disturbances.

Monostotic fibrous dysplasia → is the most common form *(80%)*. Affects children and young adults *(affects both sexes equally)*. This form of the disease affects **one bone** *(the ribs and the femur are common sites)*. The jaws are also affected frequently, mainly the **maxilla** *(it presents as a painless swelling or bulge)*. A panorex will show a radiopaque mass with irregular borders that has a **"ground glass"** appearance.

Polyostotic fibrous dysplasia → usually displays a segmental distribution of the involved bones *(multiple)*. Occurs during childhood *(mainly females)*. Affects long bones, face, clavicles and pelvic bones. The initial signs may be a limp, a pain or a fracture of the affected side. Females who are affected often reach premature puberty.

Important points concerning monostotic fibrous dysplasia:
- It is the most common form and affects both sexes equally
- Jaw involvement is common *(especially maxilla)*
- **Radiographically:** the lesion has a **"ground glass"** appearance with poorly defined margins
- The differential diagnosis of fibrous dysplasia of the jaws includes the ossifying fibroma, however, radiographically the ossifying fibroma has a well-circumscribed appearance

Important: There has been documentation to support the possibility that **malignant transformation** can occur in patients who have been **treated by radiation therapy.**

The **nevoid basal cell carcinoma** syndrome is characterized by:

- Café-au-lait spots of the skin
- Multiple osteomas
- Odontogenic keratocysts
- Polyps of the colon which ultimately change into adenocarcinoma by the fourth decade of life

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• **Odontogenic keratocysts**

The **nevoid basal cell carcinoma syndrome** is an autosomal dominant disorder characterized by oral, systemic and skeletal anomalies. It is an inherited group of defects which involve abnormalities of the skin, eyes, nervous system, endocrine, glands and bones. The condition is characterized by an unusual facial appearance and a predisposition for skin cancer. It is also known as the basal cell nevus-bifed rib syndrome, the basal cell nevus syndrome and the Gorlin and Goltz syndrome.

Possible abnormalities include:

- **Cutaneous anomalies** → including **multiple basal cell carcinomas,** other benign dermal cysts and tumors, palmer pitting, palmer and plantar keratosis and dermal calcinosis.
- **Dental and osseous anomalies** → including odontogenic keratocysts *(often* **multiple***)* mild mandibular prognathism, rib anomalies *(often* **bifed***),* and vertebral anomalies.
- **Ophthalmologic abnormalities** → including hypertelorism with wide nasal bridge an congenital blindness.
- **Neurologic anomalies** → including mental retardation, dural calcification, agenesis of corpus callosum and congenital hydrocephalus.
- **Sexual abnormalities** → including hypogonadism in males and ovarian tumors in females.

Radiographic features:

- Calcification of the falx cerebri
- Presence of odontogenic keratocysts

In most cases, what is the proper treatment for an **eruption cyst?**

- Incise and drain
- Prescribe antibiotics
- Extract the tooth associated with the eruption cyst
- No treatment is necessary as the cyst often ruptures spontaneously

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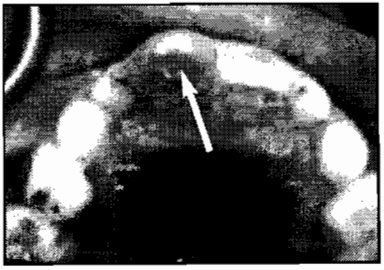
• **No treatment is necessary as the cyst often ruptures spontaneously**

In a few rare cases, incision or even the removal of the overlying tissue may be necessitated by pain or tenderness associated with the lesion.

An eruption cyst is essentially a soft tissue variant of the dentigerous cyst. It is invariably associated with an erupting tooth *(usually* **primary** *but occasionally a permanent tooth).* The effects are mostly limited to the overlying gingival tissues rather than bone.

Clinically, the lesion usually appears as a **smooth-surfaced,** reddish – pink or bluish – black, fluctuant, localized swelling on the **alveolar ridge** over the crown of an erupting primary or permanent molar tooth. The intense bluish color, which is often characteristic, is due to an accumulation of blood. Due to this appearance, it may be mistaken for a hemangioma or hematoma.



Eruption Cyst

ORAL PATHOLOGY

Odont Cyst

Upon viewing a panorex of a 14-year-old patient, you see a large radiolucent area on the left side of the mandible apical to the premolars and first molar. No clinical symptoms are present. Teeth are not carious and respond normally to vitality tests. Medical history is unremarkable. Upon opening into the area, no fluid or tissue is evident. What is the **most probable diagnosis?**

- Dentigerous cyst
- Traumatic bone cyst
- Primordial cyst
- Residual cyst

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- **Traumatic bone cyst** → also called a Simple bone cyst, Hemorrhagic bone cyst, Solitary bone cyst, Idiopathic bone cavity, Progressive bone cyst or an Extravasation cyst

Remember: This cyst may be completely devoid of solid or liquid material. It occurs most frequently in younger persons with no sex predilection. The usual location is in the **mandible** between the cuspid and ramus. The regional teeth are **vital**.

Clinical features:
- Children and adolescents, usually before the age of 20
- Usually asymptomatic
- Primarily seen in mandible
- Slight if any cortical bone expansion
- Associated with vital teeth, no displaced teeth

The **dentigerous** cyst contains a crown of an **unerupted tooth** or dental anomaly such as an odontoma. Enlarged dentigerous cysts can cause marked displacement of teeth. Pressure of accumulated fluid usually displaces the tooth in an apical direction. **See picture #50 in booklet.**

The **primordial** cyst *(also called a* **follicular** *cyst)* differs from the periodontal and dentigerous cysts in that it contains no calcified structures. These cysts are lined by stratified squamous epithelium and may be either unilocular, multilocular or multiple.

The **residual** cyst refers to a situation in which a tooth associated with a **radicular** cyst is extracted but the cyst is left undisturbed, it persists within the jaw and this lesion is called a **residual** cyst. **Note:** You must curette the socket of a tooth with a radicular cyst after extraction.

A **dentigerous cyst** is an odontogenic cyst that surrounds the crown of an impacted tooth. It arises from proliferation of remnants of:

- Hertwig's epithelial root sheath
- The reduced enamel epithelium
- Epithelial rests of the dental lamina
- A pre-existing cyst

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• **The reduced enamel epithelium**

Clinical features:

- It usually contains a crown of an **unerupted** tooth
- Usually not clinically visible without radiographs
- Asymptomatic, occasionally pain or swelling
- Usually involve unerupted mandibular third molars, other frequent sites include maxillary canines, maxillary third molars and mandibular second premolars **See picture #50 in booklet.**

Radiographic feature: Well-circumscribed, unilocular radiolucency around crown of tooth

Remember:

1. The **lateral periodontal** cyst is inflammatory in origin and forms along the lateral surface of the tooth. If at the apex, they are termed **radicular** cysts.
2. The **fissural** cysts *(which are also called* **developmental** *cysts)* are non-dental in origin, they include nasoalveolar, median palatal, and nasopalatine cysts.
3. The **primordial cyst** *(also called* **follicular** *cyst)* contains **no** calcified structures.
4. The **traumatic bone** cyst may contain blood, fluid, debris or be **completely empty.** Commonly found in young persons, in the mandible between the cuspid and ramus.

The **residual** cyst is often found in edentulous areas. This cyst refers to a situation in which a tooth with a radicular cyst associated with it was extracted, and the socket wasn't curetted. The radicular cyst persists in the jaw as a **residual** cyst.

The **gingival** cyst is a rare, circumscribed swelling of the gingiva, which is usually seen in the canine and bicuspid areas of the mandible. Usually limited to the gingiva but larger ones may erode the bone. They are easily excised.

The **odontogenic keratocyst** *(OKC)* is derived from:

- Hertwig's epithelial root sheath
- The reduced enamel epithelium
- Remnants of the dental lamina
- A pre-existing osteoma

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• **Remnants of the dental lamina**

Keratocysts differ from other odontogenic cysts in their microscopic appearance and clinical behavior. They may resemble periodontal, primordial or follicular cysts. Usually they **cannot** be distinguished radiographically.

Clinical features:
- Wide age range, peak occurrence in 2nd and 3rd decades
- More common in males than females
- 70-80% involve the mandible
- Typically asymptomatic
- The cysts are multiple in 10% of cases → in cases with multiple odontogenic keratocysts, the patient should be evaluated for other manifestations of nevoid basal cell carcinoma syndrome *(gorlin syndrome)*

Radiographic features:
- Well-demarcated area of radiolucency with a scalloped, radiopaque margin
- Unilocular or multilocular

Keratocysts increase in size principally by a process of epithelial cell multiplication. The treatment of choice is the excision of overlying mucosa in the area where the cyst wall is adhered.

Important: The most remarkable feature of keratocysts is their **great tendency toward recurrence.**

Which cyst is found in **place of a tooth** rather than directly associated with one?

- Residual cyst
- Traumatic bone cyst
- Primordial cyst
- Periodontal cyst

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- **Primordial cyst**

Cyst	Clinical Characteristics	Radiographic Characteristics
Primordial Cyst	• Found in place of tooth • Arises from epithelium of the enamel organ • Affects males and females under 25 equally • Located in **mandibular third molar** space	• Well-defined oval lesion
Residual Cyst	• Any age, male and females equally • Usually asymptomatic • Located in edentulous space • History of **prior extraction** area	• Well-defined RL not associated with any other tooth • Usually solitary

Which of the following radiographically appears as a well-defined, round or **tear-dropped shaped radiolucency** with an opaque margin?

- Lateral periodontal cyst
- Dentigerous cyst
- Odontogenic keratocyst

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- **Lateral periodontal cyst**

Cyst	Clinical Characteristics	Radiographic Characteristics
Dentigerous *(follicular cyst)*	• Children and teenagers • Mandibular third molar and maxillary canine area *(70% in mandible)* • Associated with impacted or unerupted teeth • Second most common odontogenic cyst	• Well-defined usually unilocular RL associated with the crown of an unerupted tooth
Odontogenic keratocyst	• Usually occurs between the ages of 10-30 • Often associated with an **impacted tooth** • 50% mandibular third molar area • **Over 30% recurrence rate**	• Well-circumscribed RL with smooth margins and thin radiopaque borders
Lateral Periodontal cyst	• 95% mandibular cuspid-bicuspid area • Apposition with root of vital tooth • Usually symptomless	• Well-defined, round or teardrop shaped RL with an opaque margin along lateral surface of tooth

A dental granuloma and a radicular cyst can be **differentiated:**

- Based on symptoms
- Radiographically
- Histologically
- By an electric pulp tester

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• **Histologically** → and only histologically

Lesion	Usual Location	Clinical and Radiographic Characteristics	Treatment
Dental granuloma *(It is one of the **most common** of all sequelae of pulpitis)*	Apex of tooth	• Asymptomatic • Circumscribed radiolucency at apex of tooth • Tooth is **non-vital** • May be sensitive to percussion	Root canal treatment or extraction of involved tooth
Radicular cyst *(Also called **apical periodontal & periapical** cyst).* It is the **most common** odontogenic cyst	Apex of tooth	• Asymptomatic • Circumscribed radiolucency at apex of tooth • Tooth is **non-vital** • May be sensitive to percussion	Root canal treatment with apicoectomy or extraction with curettage of socket

The **radicular** or **periapical** cyst develops within a **pre-existing** periapical granuloma *(also called a dental granuloma).*

Note: Increased osmotic pressure in the cyst lumen is important in the pathogenesis of a radicular cyst.

The **odontogenic myxoma:**

- Is composed of large polyhedral, neoplastic, epithelial cells
- Is composed of neoplastic epithelium and mesenchyme
- Arises from follicular connective tissue resembling pulp tissue
- Is composed of spindle-shaped mesenchymal cells and aggregates of multinucleated giant cells

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- **Arises from follicular connective tissue resembling pulp tissue**

Tumor	Histogenesis	Clinical Characteristics	Radiographic Characteristics	Treatment and Prognosis
Odontogenic myxoma	Dental papilla, dental sac, or periodontal ligament	Mean age at detection is 25 to 35; mandible most common site; painless swelling	Poorly defined multilocular radiolucency may be associated with unerupted and/or displaced teeth; it is an aggressive tumor	Thorough curettage with cautery; recurrence common if inadequately treated
Odontogenic fibroma	Dental papilla, dental sac, or periodontal ligament	Children and young adults; mandible; painless swelling	Multilocular radiolucency; may be associated with unerupted and/or displaced teeth	Enucleation; recurrence rare

Notes:

1. The **calcifying epithelial odontogenic** *(pindborg)* tumor is composed of large polyhedral, neoplastic, epithelial cells.
2. The **ameloblastic fibro-odontoma** is composed of neoplastic epithelium and mesenchyme.
3. The **central giant cell granuloma** is composed of spindle-shaped mesenchymal cells and aggregates of multinucleated giant cells.

The **benign cementoblastoma** usually occurs:

- In the maxilla, in the first premolar to molar region
- In the mandible, in the first premolar to molar region
- In the maxilla, in the incisor to canine region
- In the mandible, in the incisor to canine region

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- **In the mandible, in the first premolar to molar region**

Tumor	Histogenesis	Clinical Characteristics	Radiographic Characteristics	Treatment and Prognosis
Benign cementoblastoma *(true cementoma)*	Periodontal ligament	**Males, under 25**, mandibular first premolar to molar region; usually solitary; may cause expansion of cortical plates; **tooth vital**	Well-demarcated, mottled or densely radiopaque mass with radiolucent periphery; attached to the tooth root; root resorbed	Extraction of involved tooth
Gigantiform cementoma *(familial multiple cementomas)*	Periodontal ligament	Middle-aged black women; multiple, often symmetrical; may cause expansion of jaw	Large, dense, often lobulated radiopaque masses	Conservative excision

Periapical cemental dysplasia *(cementoma)* has:

- A predilection for persons younger than 20 years old
- A predilection for middle-aged caucasian men
- A predilection for middle-aged black women
- No age, racial or sex predilection

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- **A predilection for middle-aged black women**

Tumor	Histogenesis	Clinical Characteristics	Radiographic Characteristics	Treatment and Prognosis
Cementifying fibroma	Periodontal ligament	Adult, mandible, painless swelling	Well-defined radiolucency with scattered radiopaque foci	Thorough curettage, recurrence is rare, no evidence of malignant change
Cementoma *(periapical cemental dysplasia)*	Periodontal ligament	A predilection for middle-aged black women, **mandibular incisor region**, frequently multiple, teeth vital	Small sharply circumscribed radiopacity attached to or adjacent to apices of teeth, early lesions radiolucent, then central opacity, finally densely radiopaque	None, simply recognize the condition and periodically observe

The most common location for a **pindborg tumor** *(calcifying epithelial odontogenic tumor)* is the:

- Anterior jaws, with an equal distribution between the jaws
- Mandible, anterior to the first molars and may cross the midline
- Area between the roots of the maxillary central incisors
- Mandibular posterior *(molar)* area

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- **Mandibular posterior *(molar)* area**

Tumor	Histogenesis	Clinical Characteristics	Radiograpgic Characteristics	Treatment and Prognosis
Adenomatoid odontogenic tumor *(Adenoameloblastoma)*	Enamel organ, lining of dentigerous cyst, reduced enamel epithelium, Rests of Malassez	**Second** decade *(teens)*, anterior maxilla most common site, asymptomatic or painless swelling	Unilocular radiolucency associated with crown of unerupted tooth, may be tiny radiopaque foci	Enucleation; recurrence extremely rare
Calcifying epithelial odontogenic tumor *(Pindborg tumor)*	Reduced enamel epithelium	**Fourth** decade *(30%)* not likely in children and adolescents, mandibular molar area, painless swelling, rarely extraosseous	Radiolucent-radiopaque area associated with unerupted tooth, associated with amyloid production	Resection of affected area; recurrence common if inadequately treated
Squamous odontogenic tumor	Rests of Malassez	May be asymptomatic or painless swelling, associated with mobile teeth	Triangular or circumscribed radiolucency associated with unerupted or erupted tooth	Conservative excision and close follow-up; recurrence rae

Remember: These tumors all are of **ectodermal** origin *(purely epithelial)*.

The **ameloblastic fibroma** is a well-defined radiolucency predominantly located:

- Over unerupted mandibular molars in young patients
- Over unerupted mandibular incisors in young patients
- Over unerupted mandibular canines in young patients
- Over unerupted maxillary premolars in young patients

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- **Over unerupted mandibular molars in young patients**

Odontogenic Tumors with a Mixed Origin			
Tumor	**Clinical Characteristics**	**Radiographic Characteristics**	**Treatment and Prognosis**
Ameloblastic fibroma ***often mistaken for ameloblastoma	Below age 20, **mandibular molar** area, painless swelling **Compared to ameloblastoma:** • Younger age • Slower growth • Does not infiltrate	Well-defined radiolucency frequently associated with unerupted tooth	Conservative excision, recurrence rare
Ameloblastic fibro-odontoma	Below age 20, similar to ameloblastic fibroma, cut occurs with equal frequency in **maxilla and mandible**	Similar to ameloblastic fibroma, but may show foci of calcification	Similar to ameloblastic fibroma
Ameloblastic odontoma	Below age 20, **maxillary or mandibular** bicuspid-molar area, painless swelling	Well-defined radiolucency with foci of calcificatioin, may or may not be associated with erupted tooth	Resection of affected area, **may recur** if incompletely excised

How would you refer to the group of small **radiopacities** between the mandibular canine and first premolar on the periapical x-ray below?

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- **Compound odontoma**

Tumor	Histogenesis	Clinical Characteristics	Radiographic Characteristics	Microscopic Characteristics	Treatment and Prognosis
Complex	Ectodermal and mesenchymal components of tooth germ	Second and third decades **mandibular bicuspid-molar** area, **asymptomatic**, may cause delayed eruption of permanent teeth	Well-defined radiopaque mass surrounded by narrow radiolucent zone; may or may not be associated with erupted tooth	Conglomerate mass of dental tissues *(dentin, enamel, and cementum)*	Enucleation; does not recur
Compound	Ectodermal and mesenchymal components of tooth germ	Second and third decades; **maxillary incisor-cuspid** area; **asymptomatic**, may also appear in the mandible near canine-cuspid area; may cause delayed eruption of permanent teeth	Multiple small **tooth-like structures**	Multiple small, **malformed teeth;** composed of dentin, enamel and cementum	Enucleation does not recur

Remember: A compound odontoma **looks like a tooth,** and a complex odontoma **does not** *(it is a disorganized arrangement of tubular dentin, enamel, and thin layers of cementum).*
See picture #52 in booklet for picture of complex odontoma.

Cementomas *(periapical cemental dysplasia)* occur most frequently in the:

- Maxillary incisor region
- Mandibular incisor region
- Mandibular molar region
- Maxillary molar region

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• **Mandibular incisor region**

Periapical cemental dysplasia *(cementoma)* represents a reactive rather than a neoplastic process. The term "cementoma" is a misnomer as the opacities are not cementum **but bone.** While they appear to arise from the teeth, the lesions apparently arise within the bone instead. It appears to be an unusual response of the periapical bone to some local factor *(for example,* **traumatic occlusion** *or* **infection***).* **See picture #53 in booklet.**

Clinical features:

- Occurs at the apex of **vital** teeth
- A predilection for **middle-aged black women**
- Asymptomatic, usually multiple, small periapical areas of radiolucency in the mandibular incisor area.

Note: Depending on stage, it may appear mixed radiolucent and radiopaque or totally radiopaque.

Important: Age, gender, location, radiographic appearance, and tooth vitality considered together are **diagnostic of this condition.**

Three stages:

1. **Osteolytic** stage → **radiolucency** appears on radiograph.
2. **Cementoblastic** stage → beginning of calcification in the radiolucent area *(***mixed radiocent** *and* **radiopaque** *appearance).*
3. **Mature** stage→ **radiopacity** appears on radiograph with a thin radiolucent line around area.
   Note: No treatment is required for cementomas. Once this stage is reached, the lesion stabilizes and causes no complications.

Peutz-Jeghers syndrome is an autosomal dominant condition associated with:

- Antinuclear antibodies and anti-DNA antibodies that participate in immunologically mediated tissue injury
- Multiple melanotic macules and gastrointestinal polyposis
- Multiple telangiectasias
- Ipsilateral macular hemangiomas of the face

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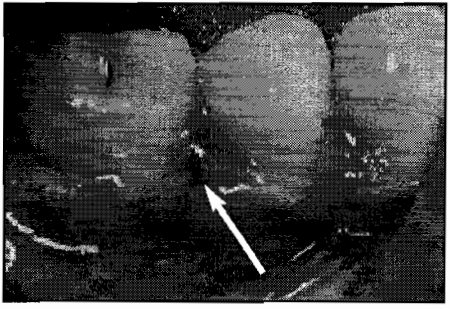
• **Multiple melanotic macules and gastrointestinal polyposis**

Peutz-Jeghers syndrome *(also called hereditary intestinal polyposis syndrome)* is an unusual condition which is of interest to the dentist because of the oral findings. The pigmentations usually appear at an early age, often during the first decade of life and at this time are restricted to the oral region. **Intraorally,** these pigmentations may be located anywhere on the mucosa, but are most frequently seen on the **buccal mucosa,** gingiva and hard palate. The mucosal surface of the **lower lip** is almost invariably involved. These spots or macules, while of variable intensity, may range through shades of brown, blue and black. During the succeeding decades of the patient's life, pigmentations may arise elsewhere on the skin, especially on the extremities. **It should be noted** that the pigmentations of Peutz-Jeghers syndrome may occur without demonstrable evidence of polyps and, also that multiple polyps may be encountered without pigmentations. **See picture #35 in booklet.**

Important: It is significant to note that although the oral pigmentations per se are harmless, their presence is important in that they indicate a necessity for investigating the possible presence of multiple polyposis which may prove harmful. There is a strong tendency for these **multiple polyps of the colon to undergo malignant change.**

Note: It appears to be caused by a mutation of a gene known as LKB1 of chromosome 19 that encodes a multifunctional serine-threonine kinase.

What **most likely** caused the discoloration of the gingiva as seen in the picture below?



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• **Amalgam**

An **amalgam tattoo** is a common finding in dental practice today. The tattoo has been mistaken for a melanin-pigmented lesion. The most common locations for amalgam tattoos are the gingiva, buccal mucosa and alveolar mucosa.

Other lesions in the mouth that are due to **chemical injuries** include dilantin hyperplasia, an aspirin burn, and the ingestion of the heavy metal bismuth.

- **Dilantin** is an anticonvulsant drug used extensively in the control of epileptic seizures. An unfortunate side effect of its use is fibrous hyperplasia of the gingiva which is called dilantin hyperplasia.
- **An aspirin burn** occurs when patients place the tablet against an aching tooth, allowing the cheek or lip to hold it in position while it dissolves slowly. Within a few minutes a burning sensation of the mucosa will be noted and the surface becomes blanched or whitened in appearance. The caustic action of the **drug causes necrosis** of the oral mucosa, with subsequent sloughing of the necrotic epithelium. **Note:** Attempt to locate amalgam on x-ray if tattoo is suspected; if you can not locate any amalgam, a biopsy may be needed to rule out a melanocytic neoplasm. **See picture #54 in booklet.**
- The use of the heavy metal bismuth is still common in treating certain dermatologic disorders as well as various other diseases. Bismuth pigmentation appears as a **"bismuth line,"** a thin, blue-black line in the marginal gingiva which is sometimes confined to the gingival papilla.

The most common location for an intraoral **congenital nevi** *(birthmark)* is the:

- Buccal mucosa
- Tongue
- Hard palate
- Alveolar mucosa

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• **Hard palate**

Moles *(nevi)* are small, usually dark, skin growths that develop from pigment-producing cells in the skin *(called melanocytes).* While **nevi** are fairly common on the skin, intraorally they are **quite uncommon.** When present, they are usually on the hard palate, but can also be seen on the gingiva and lips. **Congenital nevi** *(commonly known as* **birthmarks***)* are usually large *(greater than 10 cm)* and with the passage of time, may change from flat, pale tan macules to elevated, verrucous, hairy lesions. Approximately 15% occur on the skin of the head and neck. Congenital nevi have a **higher incidence of malignant transformation** *(as opposed to acquired nevi).*

Acquired nevi are microscopically classified into five subtypes:

1. Intramucosal nevus → most common in oral cavity
2. Blue nevus
3. Compound nevus → rare in oral cavity
4. Junctional nevus → rare in oral cavity
5. Intradermal nevus → is the **most common lesion of skin,** known as the common mole

Note: Acquired nevi are much more common than congenital nevi both **intraorally and extraorally.**

Important: The **B-K mole syndrome** and the **dysplastic nevus syndrome** are both characterized by having numerous large, pigmented atypical nevi which have a high risk for developing **malignant melanoma.**

All of the following conditions demonstrate **pigmentation** of the intraoral mucous membranes **except:**

- Addison's disease
- Albright's syndrome
- Cushing's syndrome
- Peutz-Jeghers syndrome

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• **Cushing's syndrome**

Addison's disease *(also called chronic adrenocortical insufficiency)* results from hypofunction of the adrenal cortex. It is characterized by bronzing of the entire skin. **Oral signs** consist of diffuse pigmentation of the gingiva, tongue, hard palate and buccal mucosa. Although cutaneous pigmentation will most likely disappear following therapy, **pigmentation of the oral tissues tends to persist.**

Albright's syndrome *(also called McCune-Albright syndrome)* is a severe form of **polyostotic fibrous dysplasia,** involving nearly all bones in the skeleton. In addition to the bone lesions there are brown patches of cutaneous pigmentation *(called* **café-au-lait spots***)* and endocrine dysfunction, especially precocious puberty in girls. **Important:** There is an increased incidence of **osteosarcoma** seen with polyostotic fibrous dysplasia.

Peutz-Jeghers syndrome *(also known as Hereditary Intestinal Polyposis Syndrome)* is an inherited disorder that is characterized by having **multiple intestinal polyps** and intraoral **melanin pigmentations.** These pigmentations usually appear during the first decade of life and at this time are restricted to the oral region. They may be located anywhere on the mucosa, but are most frequently seen on the **buccal mucosa,** gingiva and hard palate. The mucosal surface of the **lower lip** is almost invariably involved.

Note: Cushing's syndrome is a hormonal disorder caused by prolonged exposure of the body's tissues to high levels of the hormone cortisol. It is relatively rare and most commonly affects adults aged 20-50. The symptoms vary, but most people have upper body obesity, rounded face, increased fat around the neck *("buffalo hump"),* and thinning arms and legs.

Addison´s Disease is a rare endocrine or hormonal disorder that occurs when the:

- Pituitary gland does not produce enough growth hormone
- Adrenal glands do not produce enough cortisol
- Thyroid gland does not produce enough of the hormone thyroxin
- Pituitary gland does not produce enough antidiuretic hormone

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• **Adrenal glands do not produce enough cortisol**

Addison's disease occurs when the adrenal glands do not produce enough cortisol *(a glucocorticoid)*. Cortisol's most important function is to help the body respond to stress. The failure to produce adequate levels of cortisol can occur for different reasons. The problem may be due to a disorder of the adrenal glands themselves *(primary adrenal insufficiency)* or to inadequate secretion of ACTH by the pituitary gland *(secondary adrenal insufficiency)*. The symptoms of Addison's disease usually begin **gradually**. **These include:**

- Muscle weakness
- Loss of appetite
- Weight loss
- Skin changes with areas of hyperpigmentation covering exposed and nonexposed parts of the body. This darkening of the skin is most visible on scars, skin folds, pressure points such as elbows, knees, knuckles, and toes as well as the **oral mucous membranes** → there is diffuse pigmentation of the gingiva, tongue, hard palate, and buccal mucosa.
- Nausea, vomiting and diarrhea
- Low blood pressure

***Clinical features do **not** begin to appear until **at least 90%** of glandular tissue has been destroyed.

Laboratory tests show:

- Low blood concentrations of sodium and glucose
- Increased serum potassium
- Decreased urinary output of certain steroids

Important: The main concern when performing dental procedures on a patient with Addison's disease is that the adrenal cortex has **no capacity** to put out extra cortisol as a response to stress.

Focal melanosis is a common circumstance in which brownish areas of pigmentation occur in the oral cavity; once properly diagnosed:

- Surgical excision is required
- Radiation is required
- No treatment is necessary
- Antibiotics are required

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• **No treatment is necessary**

Focal melanosis is a disorder of increased melanin pigmentation that develops without preceding inflammatory disease. It is a condition characterized by abnormal deposits of melanin *(especially in the skin)*.

Etiology: Developmental
Location(s): Any mucosal site; **gingiva** a common location *(called an oral melanotic macule - **See picture #55 in booklet**)* and the lips, most frequently on the **lower lip** *(called a labial melanotic macule)*
Clinical Features: Brownish coloration to the oral mucous membrane
Radiographic Features: None
Microscopic Features: Melanin pigment within melanocytes
Complications: None
Treatment: None
Prognosis: Excellent
Pathogenesis: Increased numbers of melanocytes locally

Important: Melanosis may be observed in adrenal insufficiency *(Addison's disease)*, ACTH-producing tumors, malignant tumors metastatic to the pituitary gland, or metastatic malignant melanoma.

The **intraoral** nevus is usually a (an):

- Intradermal nevus
- Compound nevus
- Junctional nevus
- Blue nevus
- Intramucosal nevus

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• **Intramucosal nevus**

Most pigmented skin tumors are composed of nevus cells and are a result of a developmental anomaly of melanocytes; **they are rare in the oral cavity.** The initial, flat, raised lesion can become nodular, with an increase in consistency. Spontaneous involution may occur and malignant transformation is a rare complication. When found intraorally they most frequently occur on the **hard palate (See picture #58 in booklet)** but can also be seen on the gingiva and lips. Intramucosal nevi are the most common variety found in the oral cavity followed by **blue nevi.** Compound and junctional nevi are **very rare.**

Subtypes of Acquired Nevi:

- **Intradermal nevus** → most common lesion of **skin**. Known as the **common mole**. Nevus cells lie exclusively within the dermis. **See picture #57 in booklet.**
- **Junctional nevus** → nevus cells are located at the interface between the epithelium and lamina propria. They are flat and not detected by palpation. Some regard as premalignant, may undergo transformation into **malignant melanoma. See picture #59 in booklet.**
- **Compound nevus** → nevus cells are located at the epithelium/lamina propria interface and also deep in the dermis. They are raised and solid. **See picture #60 in booklet.**
- **Blue nevus** → congenital, painless; color based on the deep cutaneous or subcutaneous / submucosal deposits of melanin. **See picture #56 in booklet.**
- **Intramucosal nevus** → nevus cells are located in the connective tissue or lamina propria of the oral mucosa. Under palpation these nevi appear solid and are slightly raised over the surface of the mucosa.

Important: If a pigmented lesion shows ulceration, an increase in size, darkening in color, etc., a **biopsy** should be performed → this may indicate transformation into a **malignant melanoma.**

Radiographically, **traumatic bone** cysts present as a:

- Well-defined unilocular or multilocular radiolucency with scalloping around the roots
- Not well-circumscribed radiopaque lesion which may have a "ground-glass" appearance
- Saucer-shaped radiopaque lesion
- Not well-defined multilocular radiolucency with a “pear-shaped” appearance between the maxillary central incisors

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• **Well-defined unilocular or multilocular radiolucency with scalloping around the roots**

The traumatic bone cyst is an asymptomatic intraosseous empty cavity of young patients located primarily within the mandible, lined by a thin loose connective tissue membrane.

Traumatic bone cysts are non-cysts *(referred to as* **pseudocysts***)* and have many names, which include the **simple** bone cyst, **hemorrhagic** bone cyst, **unicameral** bone cyst, **extravasation** bone cyst, **idiopathic** bone cyst and **solitary** bone cyst.

Clinical features:

- Children and adolescents, usually before the age of 20
- More common in males
- Usually asymptomatic, may produce enlargement of the jaw
- Commonly found in posterior mandible ***See picture #61 in booklet.**
- Regional teeth are vital teeth

The treatment of traumatic cysts is relatively easy. It consists of opening the lesion, curettage and closure. The resultant blood clot soon undergoes organization and the bone defect is soon repaired.

Note: This cyst is a closed compartment that has a connective tissue lining of varied thickness. It may contain blood, serosanguineous fluid, debris composed chiefly of a blood clot, or it may be completely devoid of solid material.

The following are also **not true cysts** → they are called "pseudocysts":

- Latent bone cyst
- Lingual mandibular concavity
- Aneurysmal bone cyst

All of the following statements are true concerning the **aneurysmal bone cyst except:**

- It is an uncommon expansile osteolytic lesion of bone consisting of a proliferation of vascular tissue that forms a lining around blood filled cystic lesions
- Most aneurysmal bone cysts occur in patients under 20 years of age, and it is uncommon after the age of 30
- It commonly involves the jaws
- The lesions are usually tender or painful, particularly upon motion of the bone affected
- Upon entering the lesion surgically, excessive bleeding is encountered

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• **It commonly involves the jaws**

***This is **false;** it commonly involves the proximal humerus, femur, tibia and pelvis. It is not as common in the jaws, but it does appear here as well.

The aneurysmal bone cyst *(ABC)* is an uncommon lesion *(not a true cystic lesion)* located primarily in the posterior mandible and maxilla with clinical features similar to the **central giant cell granuloma.**

Histologically, the aneurysmal bone cyst has no epithelial lining *(thus it is called a* **"pseudocyst"***)*. It consists of a fibrous connective tissue stroma containing many cavernous or sinusoidal blood-filled spaces. **Fibroblasts and macrophages** *(histiocytes)* line the sinusoids. **Multinucleated giant cells,** similar to those of a giant cell granuloma, are dispersed throughout.

The **radiographic** picture of the lesion is often distinctive. The bone is expanded and appears cystic with a **"honeycomb"** or **"soap bubble"** appearance. Teeth are often moved and the roots resorbed. **See picture #62 in booklet.**

Surgical curettage or excision is the treatment of choice, with little chance of recurrence.

Notes:

1. Males and females are **equally** affected.
2. When the jaws are affected, the **mandible** is the most frequent site.

A patient presents with an asymptomatic, elongated, erythematous patch of atrophic mucosa of the mid-dorsal surface of the tongue due to a chronic C. albicans infection. The **most likely diagnosis** is:

- Thyroglossal duct cyst
- Lymphangioma
- Hemangioma
- Median rhomboid glossitis

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• **Median rhomboid glossitis**

This entity was once thought to be a congenital abnormality related to the persistence of the tuberculum impar, **however,** it is now believed that this condition is a permanent end result of a chronic **Candida albicans infection.** Diabetics, immunosuppressed patients and patients on long-term antibiotic therapy are more susceptible to this condition.

Median rhomboid glossitis usually presents as a smooth, denuded, beefy, red lesion devoid of filiform papillae. The most common location is the midline of the dorsum of the tongue, just anterior to the circumvallate papillae. It is generally asymptomatic. Generally no treatment is necessary, however, topical or systemic antifungal drugs to manage the predisposing factors may be helpful.

Most patients who develop **burning mouth syndrome** are:

- Children
- Teenagers
- Post-menopausal women
- Middle-aged males

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• **Post-menopausal women**

Burning mouth syndrome is a relatively common condition that is characterized by a complaint of an abnormal sensation of the lining of the mouth that most patients describe as feeling like their mouth has been scalded. Usually this sensation develops in the front part of the mouth, typically affecting the inner surfaces of the lips, the roof of the mouth and the sides and tip of the tongue. In some patients, only the tongue will be affected, however, any combination of these sites may be seen. Some patients may have a decreased taste ability or altered taste sensation *(bitter or salty)*. Other patients may feel that their mouths are dry or sticky. In all cases, however, the **lining of the mouth clinically appears normal.**

The etiology is unknown. There are a few common diseases that should be tested for, such as anemia, diabetes and oral yeast infections. Burning mouth syndrome is diagnosed by doing blood tests and cultures to make certain that one of the other problems mentioned previously is not present. If those tests are all negative, and if the lining of the mouth appears normal, then the diagnosis of burning mouth syndrome is made.

Unfortunately, no one has developed a medically proven treatment for burning mouth syndrome. For about half of the affected patients, the condition will resolve after a period of time, but no one can predict how long that will be for a particular individual. For the most part, this problem is a nuisance, and it is a frustrating situation for both patients and doctors.

Osler-Weber-Rendu syndrome, also known as hereditary hemorrhagic telangiectasia, is an autosomal dominant disorder identified typically by the triad of:

- Progressive lymphocytic infiltration, dysfunction of exocrine glands and xerostomia
- Telangiectasia, recurrent epistaxis, and a positive family history for the disorder
- Neurofibromas of the skin, truncal obesity, and telangiectasia
- Conjunctivitis, genital lesions, and stomatitis

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- **Telangiectasia, recurrent epistaxis, and a positive family history for the disorder**

Osler-Weber-Rendu syndrome, also known as hereditary hemorrhagic telangiectasia, is a congenital hereditary form of hemangioma. It is characterized by numerous **spider-like telangiectases** on the face, neck, chest, lips, gingiva, buccal mucosa and tongue. One of the earliest signs of the disease is **epistaxis** *(nosebleeds).* Arteriovenous fistula, especially of the lungs and liver, are a variable component. Bleeding from the telangiectases may be recurrent, life-threatening and increase in severity with aging. The onset often is in childhood. Both sexes are affected equally.

Remember:

- **Encephalotrigeminal angiomatosis** *(Sturge-Weber disease)* is an uncommon congenital syndrome of unknown etiology *(sometimes classified as a variant form of hemangioma).* It consists of a facial lesion, known as the **port-wine stain,** which is distributed over the trigeminal nerve accompanied by a similar vascular disorder of the underlying meninges and cerebral cortex. It usually occurs **unilaterally**.
- **Juvenile nasopharyngeal angiofibroma** is a rare, benign neoplasm that nearly always affects adolescent males. It characteristically produces a mass in the nasopharynx that leads to obstruction or epistaxis. Treatment is surgery. Recurrences are common.

A clinical term defined as a **red patch that cannot** be clinically or pathologically diagnosed as any other condition is called:

- Leukoedema
- Psoriasis
- Erythroplakia
- White sponge nevus

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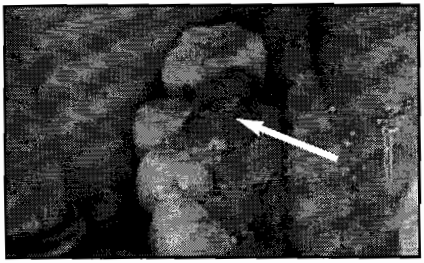
• **Erythroplakia**

The term **"erythroplakia"** like the term "leukoplakia" has **no histologic connotation.** Almost all true erythroplakias exhibit a microscopic picture of epithelial dysplasia, carcinoma in situ, or invasive squamous cell carcinoma. **Remember: Carcinoma in situ** exhibits all of the histologic characteristics of malignancy *(pleomorphism, hyperchromatism, abnormal mitoses, anaplasia, etc.)*, but does **not show** invasiveness or extension into adjacent structures.

Erythroplakias may be located anywhere in the mouth, but are **most likely** to be found in the mandibular mucobuccal fold, oropharynx and floor of the mouth. There is no sex predilection and patients over 60 years old are most commonly affected.

The arrow in the picture below is pointing to a fast-growing reactive proliferation of endothelial cells which is commonly found on the gingiva and usually forms in response to chronic irritation. The **most likely diagnosis** is:



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• **Pyogenic granuloma**

Pyogenic granulomas may also occur on the lower lip, tongue and the buccal mucosa. They rarely occur on other areas of the oral mucous membrane. It is generally believed that the pyogenic granuloma arises as a result of some **minor trauma** to the tissues *(cementation of a crown, calculus, etc.)*, which provides a pathway for the invasion of nonspecific types of microorganisms.

Pyogenic granulomas present as **soft pedunculated** broad-based growths that have a smooth red surface. This red appearance is due to the presence of **hyperplastic granulation tissue,** which contains many capillaries. They are often ulcerated, bleed easily and may have a raspberry-like appearance. Treatment consists of excision. They are benign, but may occasionally recur.

Note: Pregnant patients are prone to these lesions *(sometimes called* **"pregnancy tumor"***).*

The **peripheral giant cell granuloma** always occurs on:

- The buccal mucosa
- The alveolar mucosa or the gingiva
- The lower lip
- The palate

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• **The alveolar mucosa or the gingiva**

The **peripheral giant cell granuloma** has an unknown etiology, with some dispute as to whether this lesion represents a reactive or neoplastic process. However, most authorities believe the peripheral giant cell granuloma is a **reactive** lesion. Local irritation due to dental plaque or calculus, periodontal disease, poor dental restorations, ill-fitting dental appliances, or dental extractions has been suggested to contribute to the development of the lesion. 70% are found in the anterior segments of the jaws, such as in the premolar, canine, and incisor regions. A slight predilection for the mandible is observed in most reported series. **See picture #63 in booklet.**

These granulomas are **pedunculated** broad-based growths, which usually have a smooth surface. They are reddish-blue in color and sometimes lobulated. They usually bleed easily. The vast majority of patients are over 20 years of age. (**Remember:** *The central giant cell granuloma occurs predominantly* **before the age of 20**). Females are affected almost twice as frequently as males.

Periapical radiographs typically demonstrate a cupping out or saucerization of the alveolar bone that underlies a peripheral giant cell granuloma. **Histologic** sections are **diagnostic** and are identical to those of a central giant cell granuloma. It consists of a non-encapsulated mass of tissue composed of a delicate reticular and fibrillar connective tissue stroma with **multinucleated giant cells.** Conservative excision is typically curative, although the lesion must be completely removed to prevent recurrence

Note: It may resemble a **fibroma** or a **pyogenic granuloma** clinically.

The picture below shows a benign, soft, moderately well-circumscribed, painless mass which is deep red or blueish red in coloration. The **most likely diagnosis** is:

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- **Hemangioma**

Hemangioma			
Histogenesis ***(Etiology)***	**Clinical Characteristics**	**Microscopic Characteristics**	**Treatment and Prognosis**
Endothelial cells, Connective tissue origin.	Common tumor characterized by **proliferation of blood vessels**. Females 2:1; soft, smooth, blue, red, purple or purplish-red mass; the tongue, buccal mucosa, lips and palate are common sites; present at birth or arise in early life; enlarge as child grows.	**Three types:** 1. Capillary 2. Cavernous 3. Hemangioendothelioma: Stratified squamous epithelium covering loose, fibrous connective tissue that contains many thin-walled, engorged vascular spaces.	Laser therapy or surgery; may regress spontaneously. **Incisional biopsy is contraindicated.**

Tumors of the salivary glands are:

- Uncommon and represent 2–4% of head and neck neoplasms
- Common and represent 75–80% of head and neck neoplasms
- Uncommon and represent 25–30% of head and neck neoplasms
- Common and represent 95–98% of head and neck neoplasms

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- **Uncommon and represent 2–4% of head and neck neoplasms**

Salivary gland tumors may be broadly categorized into benign neoplasms, tumor-like conditions, and malignant neoplasms. The glands are divided into major and minor salivary gland categories. The major salivary glands are the parotid, the submandibular, and the sublingual glands. The minor glands are dispersed throughout the upper oral-digestive submucosa *(i.e., palate, lip, pharynx, nasopharynx, larynx, parapharyngeal space)*.

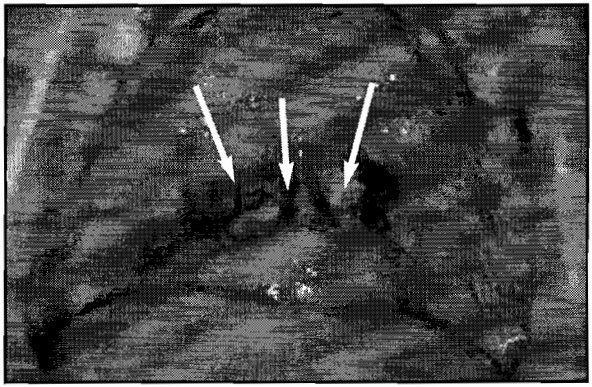
Benign Salivary Gland Tumors:

- **Pleomorphic adenoma** *(benign mixed tumor)* is the **most common** benign salivary tumor. Pathologically, it is characterized by slow growth and few symptoms.
- **Monomorphic adenomas** are a group of benign lesions with a variety of growth patterns. These lesions usually are found in the parotid glands.
- **Warthin's tumor** is a slow-growing, cystic tumor that almost always occurs in older men.
- **Benign lymphoepithelial lesions** include a wide range of cystic changes that share the common denominator in atypical lymphoid hyperplasia. Hyperplasia refers to a proliferation of cells that is non-neoplastic. These changes are found often in patients infected with HIV.

Notes:

1. Clinical features of **benign** salivary gland tumors: mucosa is normal, painless, nodular, movable, firm, and slow-growing.
2. The most common site of intraoral **minor** salivary gland neoplasms is the **palate.**
3. The most common site of intraoral **major** salivary gland neoplasms is the **parotid.**

The picture below is an uncommon, locally destructive inflammatory condition of the salivary glands. It most frequently is associated with the minor salivary glands of the palate. It is usually observed in adult males and presents as an asymptomatic, necrotic, ulcerated area involving the palatal mucosa. The **most likely diagnosis** is:



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• **Necrotizing sialometaplasia**

Necrotizing sialometaplasia is a recognized lesion of the minor salivary glands characterized by necrosis of the glandular parenchyma with associated squamous metaplasia and hyperplasia of the ductal epithelium. The etiology is unknown, but it is believed to be related to vascular insufficiency and infarction of the glands.

It is usually observed in adult males and presents as an asymptomatic, necrotic, ulcerated area involving the palatal mucosa. The **hard palate** appears to be the most common site. Histologically, there is lobular necrosis of the glandular parenchyma, with squamous metaplasia and hyperplasia of the ductal epithelium. Both clinically and histologically, the lesion may simulate a malignancy and in the past, the condition has been **misdiagnosed** as a squamous cell or mucoepidermoid carcinoma.

Following biopsy and the establishment of the diagnosis, further treatment generally is **not recommended** since healing usually occurs within 6–12 weeks.

The diagnosis of **mumps** is usually suspected based on clinical manifestations, in particular the presence of:

- Sialolithiasis
- Thyroiditis
- Candidiasis
- Parotitis

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• **Parotitis**

Mumps is an acute viral illness. The mumps virus is a **paramyxovirus** in the same group as the parainfluenza virus. The virus is acquired by respiratory droplets. It replicates in the nasopharynx and regional lymph nodes.

Clinical:

- **90%** of the cases occur **before 14 years of age**
- A major sign is **sudden salivary gland swelling** without purulent discharge from the duct.
  ***Parotid** → 90% involvement and bilateral in two-thirds of the cases.
- Mild fever, malaise and anorexia
- Most cases are self-limiting

Complications:

- **Orchitis** *(inflammation of the testis)* and epididymitis can occur in post-pubertal males.
  ***Important:** May cause sterility
- CNS system → meningitis and encephalitis
- Deafness, myocarditis, pancreatitis, oophoritis and pyelonephritis

The **serum amylase** may be elevated during the acute phase. Prevention with a **live attenuated vaccine** is 95% effective for at least five years. However, in non-inoculated individuals, it is still a cause of acute **non-suppurative salivary adenitis.**

All of the following statements concerning **developmental salivary gland defects** are true **except:**

- They are very common
- They are asymptomatic
- They are next to impossible to palpate manually
- They are discovered only during radiographic examination of the area

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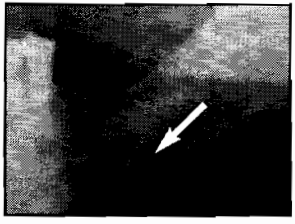
• **They are very common**

***This is **false;** they are **relatively rare.**

The image of the developmental salivary gland defect is a **round or ovoid radiolucency** that ranges in diameter from 1 to 3 cm. It generally develops below the mandibular canal and above the inferior border of the mandible, just anterior to the angle of the jaw, and below and just behind the third molar.



Salivary gland depression beneath the mandibular canal

Mikulicz's disease is due to an autoimmune process and is looked upon as a clinical variant of:

- Pierre-Robin syndrome
- Sjögren's syndrome
- Goltz-Gorlin syndrome
- Apert syndrome

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• **Sjögren's syndrome**

The term **"benign lymphoepithelial lesion"** *(also called* **Mikulicz's disease***)* is manifested essentially as a progressive, asymptomatic enlargement of the parotid and submandibular glands. It is initially unilateral, but over time, it becomes bilateral. The etiology is unknown, however, there is increasing evidence that both Mikulicz's disease and Sjögren's syndrome are both actually autoimmune diseases in which the patient's own salivary gland tissue becomes antigenic.

The benign lymphoepithelial lesion is rare. It occurs most frequently in **middle-aged women.** Histologically, there is replacement of the gland parenchyma by lymphocytic infiltrate within which there are scattered **epimyoepithelial islands**. This is the histologic cornerstone for the diagnosis of the benign lymphoepithelial lesion.

Important: Most of these lesions remain benign, however, **malignant transformation of the epimyoepithelial islands** has been demonstrated.

95% of **mucoceles** are found on the:

- Upper lip
- Palate
- Labial mucosa of the lower lip
- Gingiva

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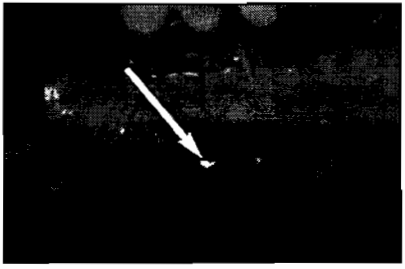
• **Labial mucosa of the lower lip**
***They are seldom found on the upper lip

The **mucous retention cyst** *(also called a mucocele)* is generally conceded to be of traumatic origin. It involves the **minor salivary glands and their ducts** *(for example, trauma to the salivary duct by lip biting or pinching)*. **Note:** It is **not a true** cyst *(not lined by epithelium)*. Microscopically, shows spilled mucin surrounded by granulation tissue, with many foamy histiocytes.

A mucocele is a common lesion that may also appear on the palate, cheek, tongue and floor of the mouth. Clinically, the lesion may lie fairly deep in the tissue, or be exceptionally superficial, and depending upon the location, will present a variable clinical appearance. The **superficial lesion** appears as a raised, circumscribed vesicle, several millimeters to a centimeter in diameter, **with a bluish, translucent cast.** The **deeper lesion** appears as a fluctuant swelling also, but the tissue appears **normal in color.** Treatment is excision.



What is the **most probable diagnosis** for a lesion that presents as a translucent, bluish, well-rounded, smooth-surfaced bulge that protrudes from one side of the floor of the mouth?

- A mucocele
- Squamous cell carcinoma
- A ranula
- A lymphangioma

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• **A ranula**

***It is **fluctuant and painless.**

The **ranula,** a true retention cyst, characteristically occurs in the floor of the mouth and is unilaterally located. It arises in association with the secretory ducts of the submandibular or sublingual glands, and is usually caused by an obstruction produced by either a salivary stone or soft organic substance.

Of diagnostic significance is a history of **increased size just before or during a meal,** and a decrease in size between meals. The treatment is surgical, either through complete excision or by removing the roof of the cyst. If it persists, excision of the gland may be needed.

Most **sialoliths** *(stones)* are found in the:

- Submandibular duct *(Wharton's)* and gland
- Parotid duct *(Stensen's)* and gland
- Sublingual gland and ducts

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• **Submandibular duct *(Wharton's)* and gland**

A **sialolith** is a stone *(salivary calculus)* within a salivary gland or duct. The formation of a sialolith is called **sialolithiasis** and occurs as a result of precipitation of calcium and phosphate salts around a nidus of mucous or bacterial debris. Sialoliths occur as single or multiple stones and can cause swelling and pain. The pain is experienced during salivary stimulation and is **intensified at mealtimes.**

The best radiographic projection for visualizing sialoliths in the submandibular duct and gland is the standard **mandibular occlusal view.** Occasionally, sialoliths are seen incidentally on periapical radiographs, in which case they may be misdiagnosed as osteosclerosis.

The rate of occurrence in **submandibular gland** and duct is much higher than in the parotid or sublingual areas. This is thought to be due to the tenacity of the submandibular saliva and the long and irregular shape of the duct. **See picture #64 in booklet.**

The treatment of choice is almost invariably **surgical extirpation of the sialolith.** Stones located in the glandular parenchyma usually require removal of the gland as well.

Of the neoplasms affecting the major or minor glands, the _____is the most common.

- Basal cell adenoma
- Sebaceous adenoma
- Pleomorphic adenoma
- Intraductal papilloma

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• **Pleomorphic adenoma**

***It is also called **"benign mixed tumor".**

The term **"mixed tumor"** was used by investigators who believed the neoplasm was of both ectodermal and mesenchymal origin.

Clinical features: The pleomorphic adenoma is the most common salivary gland neoplasm. Women are affected more frequently than men, and the majority of patients are between 40 and 60 years old. Approximately 93% arise in the major salivary glands and these are almost exclusively parotid neoplasms *(84%)*. **They present as painless lumps below and anterior to the ear.** Approximately 7% arise in the oral cavity with the palate by far the most common intraoral site. Here they appear as firm, painless swellings, and in the vast majority of cases, do not cause ulceration of the overlying mucosa. **See pictures #65 and #66 in booklet.**

Histologic features: The epithelial component of a pleomorphic adenoma consists of round, polyhedral, elongated or stellate cells, which are relatively small and stain uniformly. The mesenchymal component varies from areas of myxomatoid tissue to areas of dense, hyalinized connective tissue, pseudocartilage or bone.

Treatment and prognosis: Pleomorphic adenomas are invariably encapsulated or well demarcated, and **surgical excision** with a generous margin of normal tissue is the treatment of choice. Inadequate initial removal of the mixed tumor in major glands may result in recurrence. Approximately 25% of benign mixed tumors will undergo malignant transformation if lesions are untreated for an extended length of time.

All of the following metabolic conditions are associated with chronic salivary gland enlargement **except:**

- Diabetes mellitus
- Chronic alcoholism
- Malnutrition *(including anorexia and bulimia)*
- Hypothyroidism

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- **Hypothyroidism**

Other metabolic conditions that are associated with salivary gland enlargement include obesity, hypertension and hyperlipidemia.

Important: The **parotid gland** is most frequently enlarged. This can be unilateral or bilateral.

Other conditions associated with parotid gland enlargement:

- Sjögren's syndrome
- Sarcoidosis
- Warthin's tumor → also called papillary cystadenoma lymphomatosum
- Infections → for example, mumps, actinomycosis, tuberculosis
- Benign lymphoepithelial lesion → also called Mikulicz's disease
- Acute epidemic parotitis
- Malnutrition

The **acinic cell carcinoma** is derived from serous acinar cells and is found almost exclusively in the:

- Submandibular gland
- Parotid gland
- Minor glands of the palate
- Sublingual gland

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• **Parotid gland**

Acinic cell carcinoma: Are rare, accounting for less than 10% of all salivary gland tumors. They tend to arise in the parotid and rarely in the submandibular glands. They are considered low-grade malignancies.

Adenocarcinoma, NOS *(not otherwise specified):* are most frequently found in the minor salivary glands of the nose and paranasal sinuses. They do, however, account for 15% of malignancies of the parotid and 10% of malignancies of the submandibular glands.

The **adenoid cystic carcinoma** is the most common malignant tumor of the minor salivary glands and comprises 23% of all salivary gland tumors. These tumors occur with a median age of 43 years. They commonly present as a slowly growing tumor with severe pain and occasionally with _____ as the tumor infiltrates into this nerve.

- Trigeminal nerve paralysis
- Hypoglossal nerve paralysis
- Vagus nerve paralysis
- Facial nerve paralysis

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• **Facial nerve paralysis**

Adenoid cystic carcinoma *(ACC)* accounts for approximately 25% of all salivary gland carcinomas. Approximately 50 to 70% occur in the minor salivary glands of the head and neck. In the major salivary glands, the parotid gland is most often affected. ACC has a characteristic appearance under the microscope. Abnormal "nests" or cords of certain cells *(epithelial cells)* can be seen to surround or infiltrate ducts and glandular structures. These structures are typically filled with a mucous-like material or contain abnormal fibrous membranes *(hyaline membranes)*. **See picture #68 in booklet.**

ACC is an unusual tumor. It is **slow-growing but relentless.** It tends to be locally invasive and infiltrate the "sheaths" or coatings surrounding nerve fibers *(perineural spaces)*. ACC often recurs years later at the site where the tumor first arose or it may metastasize. Unlike most carcinomas, it seldom metastasizes to nearby lymph nodes but rather to distant sites. The lung is the most common site of metastasis, with the liver second. Bone metastases indicate a poor prognosis.

Mucoepidermoid carcinoma is the most common **malignant** salivary gland neoplasm. It affects chiefly the parotid, but also other major and minor salivary glands. These tumors grow slowly and present as painless masses in most cases. They are unencapsulated or poorly encapsulated and infiltrate surrounding tissue freely. "High-grade" and "low grade" forms exist each with a different prognosis *(good for low-grade; poor for high-grade)*. Surgical excision is the treatment. **See picture #67 in booklet.**

Sjögren's syndrome is a chronic inflammatory disorder of probable autoimmune nature characterized by infiltration of the exocrine glands, particularly the salivary and lacrimal glands, by lymphocytes and plasma cells. The classic signs of the Sjögren's syndrome, include all of the following **except:**

- Rheumatoid arthritis
- Nephrosclerosis
- Xerostomia *(dry mouth)*
- Keratoconjuctiva sicca *(dryness of the eyes)*

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- **Nephrosclerosis**

Sjögren's syndrome is a disorder of unknown cause, however, recent evidence suggests that it is autoimmune in nature. It is marked chiefly by chronic inflammation of the salivary glands and lacrimal glands. This usually progresses to fibrosis and atrophy of these glands. **All three symptoms rarely occur in one patient.** A definite diagnosis can be made only when at least two of the symptoms are present. The patients **most commonly affected** are post-menopausal women who present with dry eyes, dry mouth and, in about 50% of the cases, enlargement of the parotid and submandibular glands bilaterally.

Important: The histological features of the salivary gland lesions in both **Sjögren's syndrome** and the **"benign lymphoepithelial lesion"** *(also called Mikulicz's disease)* are identical.

The treatment of Sjögren's syndrome is mainly **symptomatic**. The keratoconjunctivitis is treated with ocular lubricants and the xerostomia is treated by saliva substitutes.

Note: Biopsy of the labial or palatal salivary glands may be helpful in establishing the diagnosis, along with sialograms, salivary flow rate tests and blood work.

It is important to remember that **malignant lymphomas** and **"pseudolymphomas"** *(also called atypical benign lymphoid hyperplasia)* develop in some patients who have been diagnosed with Sjögren's syndrome. **This mandates close follow-up of the patients.**

Important: The decrease in salivation may cause **rampant caries** reminiscent of radiation caries.

The parotids are the salivary glands most often affected by tumors. Most of the tumors that grow in the parotid glands are:

- Malignant
- Mixed *(benign and malignant)*
- Carcinoma in situ
- Benign

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• **Benign**

Approximately 8 out of 10 salivary tumors diagnosed are in a parotid gland. One in 10 diagnosed is in a submandibular gland. The remaining 10% are diagnosed in other salivary glands. **In general,** glands more likely to show tumor growth are also glands least likely to show malignant tumor growth. Thus, although tumors of the **sublingual glands** are rare, almost all of them are **malignant.** In contrast, about one in four tumors of the parotid glands are malignant.

Clinical features of malignant salivary gland tumors:
– Mucosa is ulcerated – Firm – Painful – Nodulas – Fixed – Rapid growth

Malignant Salivary Gland Tumors:

- **Adenocarcinoma,** NOS *(not otherwise specified):* are most frequently found in the minor salivary glands of the nose and paranasal sinuses. They do, however, account for 15% of malignancies of the parotid and 10% of malignancies of the submandibular glands. Squamous cell cancers are uncommon in salivary gland tissue. In many cases they originate not in the parotid itself, but stem from metastases originating elsewhere in the head and neck.
- **Adenoid cystic carcinoma:** account for 25% of malignant salivary gland tumors and 15% of all parotid gland tumors. However, they seem to occur most often in the minor, rather than major, salivary glands. The disease is unique in that its tumors grow slowly, but metastasize readily.
- **Mucoepidermoid carcinoma:** is unique in that the tumors it produces can vary in aggressiveness from low-grade and slow growing to high-grade and rapidly growing. It occurs more frequently than any other malignancy of the major salivary glands.
- **Acinic cell carcinoma:** Are rare, accounting for less than 10% of all salivary gland tumors. They tend to arise in the parotid and submandibular glands, and are considered low-grade malignancies.
- **Malignant mixed tumors:** make up approximately 15% and 12% of parotid and submandibular neoplasms respectively. The disease typically is characterized by slow, protracted growth.

Oncocytomas are benign salivary gland tumors accounting for:

- 1% of all salivary gland tumors
- 35% of all salivary gland tumors
- 65% of all salivary gland tumors
- 90% of all salivary gland tumors

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- **1% of all salivary gland tumors**

The majority *(84%)* occur in the parotid gland and are found in even distribution between men and women usually in their 6th decade.

An **oncocytoma** *(also called an oxyphilic adenoma or acidophilic adenoma)* is a small benign, glandular tumor composed of large cells with cytoplasm that is granular and eosinophilic due to the presence of abundant mitochondria. Its development is believed to be related to the aging process.

Histologic features: The tumor is an encapsulated mass composed of relatively large cells with bright pink cytoplasm and small round nuclei. The cells may be arranged in sheets or cords, or form tubular or acinar structures.

Treatment and prognosis: The treatment of choice is surgical excision and recurrence is rare.

Note: Sialoscintigraphy is a simple and non-invasive procedure that can usually separate benign entities like Warthin's tumor and oncocytoma of the salivary glands from malignant tumors, and significantly affect the course of treatment.

Warthin's tumor is a slow-growing, cystic tumor that almost always occurs:

- In children
- In adolescent females
- In older men
- In older women

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• **In older men**

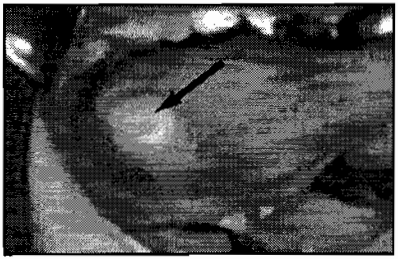
Papillary cystadenoma lymphomatosum *(a fancy name for* **Warthin's tumor***)* is almost exclusively a **parotid** neoplasm. It is believed to arise from heterotopic ductal epithelium within lymph nodes or near the parotid gland.

Clinical features: The vast majority of patients are over 50 years of age, with a 5:1 male predominance. Approximately 5% of the tumors are bilateral. The tumor most often arises in the lower pole of the parotid and presents clinically as a **non-tender,** slowly enlarging, firm to fluctuant nodule over the **angle or ramus** of the mandible.

Histologic features: The tumor is encapsulated and composed of cystic spaces containing an eosinophilic coagulum into which extend papillary projections of the lining epithelium. The epithelium consists of a double row of cells with eosinophilic, granular cytoplasm, a luminal layer of tall columnar cells and a basal layer of round, cuboidal or polygonal cells. Interspersed among the cystic spaces are aggregates of lymphoid tissue, some with germinal centers.

Treatment and prognosis: Surgery is the treatment of choice and recurrence is uncommon. Malignant variants of the tumor have been reported but are rare.

A 40 year-old female presented with a subcutaneous nodule on the right lateral surface of her tongue *(arrow is pointing to lesion)*. The nodule was non-tender, soft, movable and had been slowly growing for about 2 years. The nodule was excised. Its cut surface was yellow and lobulated. **What is the clinical significance of the nodule being movable and slowly growing?**



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• **These are characteristics of a benign neoplasm**

General characteristics of **benign** neoplasms:
- Well-differentiated
- **Slow growth**
- Encapsulated / Well–circumscribed
- Localized
- **Movable**

General characteristics of **malignant** neoplasms:
- Invasion
- **Immovable**
- **Rapid growth**
- **Metastasis**
- Not well-differentiated *(or anaplastic)*

Important:
- **Paresthesia** is suggestive of **metastatic** disease.
- **Metastasis** is the most important characteristic that distinguishes malignancy from benign.

Radiographically, a **benign** neoplasm in bone may be differentiated from a **malignant** neoplasm in the following ways:
- In a **benign** lesion, the cortex tends to **remain intact** but may be thinned and the part involved expanded.
- In a **benign** lesion, the margins are usually **defined** and demarcated from surrounding bone.

***The nodule in the picture on the front of card is a **lipoma.**

A mass of histologically **normal** tissue present in an **abnormal** location is called a:

- Teratoma
- Choristoma
- Hamartoma

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- **Choristoma**

Terms used in oncology:

- **Oncology:** the study of neoplasms
- **Neoplasm:** an uncontrolled new growth of tissue
- **Tumor:** a localized swelling, may or may not be a true neoplasm
- **Hyperplasia:** an increase in the size of a tissue or organ due to an increase in the **number** of component cells
- **Hypertrophy:** an increase in the size of a tissue or organ due to an increase in the **size** of component cells
- **Cancer:** a general term for all **malignant** neoplasms
- **Carcinoma:** a malignant **epithelial** neoplasm
- **Sarcoma:** a malignant **mesenchymal** *(connective tissue)* neoplasm
- **Hamartoma:** a developmental defect characterized by an overgrowth of tissues **normal** to the organ in which it arises
- **Teratoma:** a **neoplasm** composed of multiple tissues **foreign** to the organ in which it arises; may be benign or malignant

Malignant neoplasms range from well-differentiated to undifferentiated. Malignant neoplasms composed of **undifferentiated cells** are said to be:

- Metaplastic
- Hyperplastic
- Anaplastic
- Dysplastic

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- **Anaplastic**

Lack of differentiation, or **anaplasia,** is considered a **hallmark of malignant transformation.** It is marked by a number of morphologic and functional changes. Both the cells and the nuclei characteristically display **pleomorphism** – variation in size and shape. Characteristically the nuclei contain an abundance of DNA and are extremely dark staining *(hyperchromatic).* The nuclei are disproportionately large for the cell, and the **nuclear-cytoplasmic ratio** may approach 1:1 instead of the normal 1:4 or 1:6. The nuclear shape is usually extremely variable, and the chromatin is often coarsely clumped and distributed along the nuclear membrane. Large nucleoli are usually present in these nuclei.

Histological grading of malignant neoplasms:

- An attempt to estimate the aggressiveness of degree of malignancy of a malignant neoplasm based upon the **degree of differentiation** of the component cells and the number of mitoses.
  - Grade 1. **Well**-differentiated
  - Grade 2. **Moderately well**-differentiated
  - Grade 3. **Poorly** undifferentiated
  - Grade 4. **Undifferentiated**

- Applicable mainly to **squamous cell carcinomas** and of limited clinical usefulness. Most pathologist use **three grades** and prefer to designate squamous cell carcinomas as well differentiated, moderately well-differentiated or poorly differentiated.

Dysplasia is encountered principally in the epithelia. It is a loss in the uniformity of the individual cells as well as a loss in their architectural orientation. **Dysplasia:**

- Always progresses to cancer
- Never progresses to cancer
- Does not necessarily progress to cancer
- Has nothing to do with cancer

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• **Does not necessarily progress to cancer**

***Mild to moderate changes that **do not involve** the entire thickness of epithelium **may be reversible,** and with removal of the putative inciting causes, the epithelium **may revert to normal.**

Dysplastic cells exhibit considerable **pleomorphism** *(variation in size and shape)* and often possess deeply stained ***(hyperchromatic)*** nuclei, which are abnormally large for the size of the cell. It is associated with chronic irritation of a tissue by a chemical agent, such as cigarette smoke or by chronic inflammatory irritation, such as chronic cervitis. The tissue appears somewhat structureless and **disorganized** and may consist of **atypical cells** without invasion. Epithelium exhibits **acanthosis** *(which is an abnormal thickening of prickle cell layer).* **Important:** When dysplastic changes are marked and involve the entire thickness of the epithelium, the lesion is considered a pre-invasive neoplasm and is referred to as **carcinoma in situ.**

Histologic features of malignancy:

• Anaplasia • Hyperchromatism • Pleomorphism • Abnormal mitosis

The host response to a **malignancy** is best reflected by lymphocytic infiltration at the edge of a tumor. The most characteristic feature of a malignancy as opposed to an inflammatory lesion is that a malignancy **will grow after removal of the causative agent.** The most important characteristic of malignant neoplasms, which distinguishes them from benign neoplasms, is their **ability to metastasize.**

Metaplasia is the process whereby one cell type changes to another cell type in **response to stress** and generally assists the host to adapt to the stress. The **most common type** of epithelial metaplasia involves replacement of columnar cells by stratified squamous epithelium.

All of the following are forms or classifications of recurrent **aphthous** ulcers **except:**

- Initial
- Minor
- Major
- Herpetiform

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• **Initial**

***Recurrent aphthous ulcers are commonly referred to as **"canker sores"** by lay persons. In the literature other terms include aphthous stomatitis, recurrent aphthous stomatitis, recurrent ulcerative stomatitis or ulcerative stomatitis.

These ulcers appear to be **associated** with stress. The **stress factors** may include:
- Bacterial infection
- Trauma *(i.e., self-inflicted, oral surgery procedures, routine dental procedures)*
- Endocrine conditions *(i.e., a females menstrual period)*
- Allergic factors *(i.e., certain foods or drugs)*
- Immunologic abnormalities
- Iron, Vitamin B or folic acid deficiencies

***The cause is unknown;** however, evidence supports they are related to the focal immune dysfunction where T-lymphocytes play a major role.

Three classifications:
1. Recurrent aphthous **minor:** 80% occur on non-keratinized movable mucosa
2. Recurrent aphthous **major:** heal often with scarring, more common in HIV patients, occur on the soft palate, tonsillar fauces, labial and buccal mucosa, and tongue
3. Recurrent **herpetiform:** occur in up to 100 at a time, occur on any mucosal surface, heal without scarring

***All three classifications present painful recurrent ulcers.

Remember: Vesicles **do not** precede the formation of the ulcers.

Stevens-Johnson syndrome is a severe form of:

- Lichen planus
- Erythema multiforme
- Pemphigus vulgaris
- Cushing's disease

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• **Erythema multiforme**

Stevens-Johnson syndrome *(SJS)* is an immune-complex–mediated hypersensitivity complex that is a severe expression of erythema multiforme. It is now known also as erythema multiforme major. In SJS, the systemic symptoms are **severe** and the lesions are **extensive,** involving multiple body areas *(especially the mucous membranes).*

Typical symptoms are as follows:

- Cough productive of a thick purulent sputum
- Headache
- Malaise
- Arthralgia

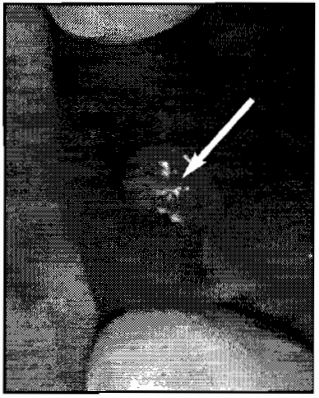
The typical **"bull's-eye-shaped "** target lesions are present. These lesions are considered pathognomonic.The classical triad of this SJS consists of **eye lesions, genital lesions and stomatitis. Important:** The lesions of SJS are **severe** and often **vesicular or bullous,** with hemorrhage after denudation. **Note:** Blindness can occur due to secondary infection.

Treatment of SJS is primarily supportive and symptomatic:

- Manage oral lesions with mouthwashes
- Topical anesthetics are useful in reducing pain and allowing the patient to take in fluids
- Areas of denuded skin must be covered with compresses of saline
- Underlying diseases and secondary infections must be identified and treated
- Offending drugs must be stopped

Drugs and **malignancies** are most often implicated as the etiology in **adults** and the **elderly. Pediatric cases** are related more often to **infections** than to malignancy or a reaction to a drug.

The lesion below is a small *(2mm-5mm in diameter)*, whitish sore with a red border. The patient states that it usually begins as a reddish area with a burning or tingling sensation. The **most likely diagnosis** is:



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- **Recurrent aphthous minor**

There are **three forms** or classifications of recurrent aphthous stomatitis *(which is also called recurrent aphthous ulcers, canker sores, etc.)*

1. **Recurrent aphthous minor** → is the **most common** form of the disease and the one referred to by the lay public as the **"canker sore."** The lesions occur somewhat more frequently in women than in men. Minor aphthae have a propensity for movable mucosa that is situated over minor salivary gland tissue. The lesions begin as a single or multiple superficial erosion covered by a gray membrane. The lesion is typically **very painful.** The lesions vary in size from 2-3 mm to over 10 mm in diameter. They generally persist for 7-10 days and heal gradually with **little or no** evidence of scarring.
2. **Recurrent aphthous major** → is characterized by the occurrence of **large lesions are large** *(5-20mm)* **painful ulcers,** usually one to ten in number. These ulcers occur at frequent intervals and many patients with this disease are seldom free from the presence of at least one ulcer. Unlike the minor aphthous ulcer, these lesions persist for up to six weeks and **leave a scar** upon healing.
3. **Recurrent herpetiform** → the prominent feature of the disease is the numerous, pinhead-sized, gray-white erosions that enlarge, coalesce, and become irregular ulcers. Ulcers occur in clusters of 10 to 100.

Remember: Vesicular lesions **do not precede** the formation of ulcers in all of the above. **This is a distinctive diagnostic feature.**

Note: In healing of an ulcer, the epithelium that eventually will cover the defect is derived from intact epithelium at the ulcer margin.

Erythema multiforme *(EM)* is an acute self-limited eruption characterized by a distinctive clinical eruption, the **hallmark of which** is the:

- Chronic desquamative gingivitis
- Petechial hemorrhage
- Iris or target lesion
- Mucocutaneous rash

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- **Iris or target lesion** → appears as a central lesion surrounded by concentric rings of pallor and redness over the dorsal aspect of the hands and forearms.

Erythema multiforme *(EM)* is a hypersensitivity reaction that occurs in mild and severe forms. It produces tissue reactions centered on the superficial vessels of the skin and mucous membranes. Precipitating factors include infections such as herpes simplex, mycoplasma pneumonia and histoplasmosis, drugs, radiation therapy, etc. It occurs primarily in children and young adults. The diagnosis of erythema multiforme is primarily based on the **classic skin lesion** appearance.

Clinical features → 3 forms:

- **EM minor** → primarily involves the skin with only 25% in oral mucosa. A low-grade fever, general malaise and headache usually precede the appearance of the lesions by 4 to 7 days. **Oral lesions** appear as red macules, papules or vesicles that may become eroded and painful. **Note:** These lesions are covered by a yellowish-white membrane after rupturing. Focal or diffuse areas of erythema follow and the skin "target" or "bulls eye" lesions appear. **See picture #69 in booklet.**
- **Chronic EM Minor** → mildest form and lesions are small in size and shorter in duration. Oral lesions vary from focal erosions similar to aphthous ulcers to more diffuse areas of erythema or erosions that are painful.
- **EM Major** *(Stevens-Johnson Syndrome)* → acute form that involves the skin and mucous membrane. Large bullae form on the mucous membranes and skin. A positive Nikolsky's sign is common. The bullae collapse which produces a whitish pseudomembrane on the mucosa and dark red crusted lesions on the dry skin surfaces *(lips and eyes very common)*.

Significant diagnostic findings in exudates from cases of human **actinomycosis** include:

- Black granules
- Small encapsulated gram-negative rods
- Strongly acid-fast slender rods
- Sulfur granules

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- **Sulfur granules**

Actinomycosis is a subacute-to-chronic bacterial infection caused by **Actinomyces israellii,** an anaerobic, gram-positive filamentous bacterium. The infection is not a contagious disease and can not be spread from person to person. Infections appear after trauma, surgery or previous infection.

Clinical features:

- Most infections occur in the thorax, abdomen and **head and neck** → **cervicofacial actinomycosis** *(infection typically occurs in patients with poor dental hygiene or following oral surgery)*
- Often presents as a swelling of the mandible and usually is painful
- Once in the tissue, Actinomyces forms an abscess, producing a hard, red-to-reddish-purple lump, often on the jaw → **"lumpy jaw" ***See picture #70 in booklet.**
- Exudate from the draining sinus tracts often contain small, clinically visible yellow-green calcified structures *(sulfur granules)* → which are actually colonies of infecting organisms

***Treatment for actinomycosis is **long-term penicillin.**

Histoplasmosis is a chronic **lung** infection caused by inhalation of spores of Histoplasma capsulatum. The **classic oral manifestation** of histoplasmosis infection is a **chronic non-healing ulcer. Note:** The lesions are usually covered by a non-specific gray membrane and are indurated.

Clinical features:

- Fever, malaise, cough, and dyspnea
- In **chronic** forms, **dissemination** to the skin and oral mucous membranes may be the first signs of infection
- Cervical lymphadenopathy is common

***Treatment for histoplasmosis is amphotericin B, itraconazole or ketoconazole for 6-12 months.

The most infectious stage of **syphilis** is the:

- Primary stage
- Secondary stage
- Tertiary stage

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• **Secondary stage**

Syphilis is an infectious, contagious venereal disease that is caused by the spirochete **treponema pallidum.** It is one of the less common sexually transmitted infections *(STI).* The incubation period varies somewhat between 10 and 90 days. The symptoms of syphilis may pass unrecognized, or may be misinterpreted and at times there are no initial symptoms at all. When present, the classical symptoms of syphilis manifest themselves in three stages as follows :

1. **Primary** → the first symptom is a non-painful **chancre** that generally appears 2-6 weeks after exposure. It usually is found on the part of the body exposed to the partner's ulcer, such as the penis, the vulva, or the vagina. It can also develop on the cervix, tongue, lips, or other parts of the body. The chancre disappears within a few weeks whether or not a person is treated. If not treated during the primary stage, about one-third of people will progress to chronic stages.
2. **Secondary** → is a highly **infectious** stage; it occurs 6 weeks after non-treatment of primary syphilis. Widely disseminated spirochetes cause mucous membranes to exhibit a **reddish brown maculopapular cutaneous rash** and ulcers that are covered with a mucoid exudate *(called mucous patches).* **Condylomata lata** *(which are elevated broad-based plaques)* are also seen on skin and mucosal surfaces. If left untreated, these symptoms will resolve on their own...but the infectious microbe remains behind. It is at this point that syphilis passes into its **latent phase.** This silent period may last for many years and permits the infection to evolve without any obvious external symptoms. At this point, the only method of detecting the presence of syphilis is via a blood test specific for syphilis.
3. **Tertiary** → occurs in infected persons many years after non-treatment of secondary syphilis. The **gumma** *(which is a focal nodular mass)* typifies this stage. It most commonly occurs on the palate and tongue. The bacteria damage the heart, eyes, brain, **nervous system**, bones, joints, or almost any other part of the body. **Note:** Headache, stiff neck, and fever are symptoms of **neurosyphilis.**

The screwdriver-shaped central incisors seen in picture below of a child with **congenital syphilis** are called:

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• **Hutchinson's incisors**

Congenital syphilis is caused by infection with the spirochete Treponema pallidum during the fetal period. Expectant mothers who have syphilis can transmit the disease through the placenta to the unborn infant. Nearly half of all infants infected with syphilis during gestation die shortly before or after birth.

The severity of congenital syphilis depends upon **1)** The time in which the organisms pass the placental barrier *(protected up to 16th week),* **2)** The mother's stage of syphilis and, **3)** The immunologic response of the fetus. If treated by the 4th or 5th month, 95% show no manifestations; if not treated, fetal sepsis may result in stillbirth or visceral and mucocutaneous manifestations.

1. **Symptoms in the newborn**

- Irritability
- Bloody discharge from the nose
- **Early rash:** small blisters or flat or bumpy rash on the face, palms and sole
- Failure to thrive
- Later rash: copper-colored, vesicles on the palms and soles
- Saddle nose, frontal bossing and short maxilla

2. **Symptoms in older infant and young child**

- Bone pain
- Joint swelling
- Abnormal teeth *(Hutchinson's incisors)*
- Gray, mucous-like patches on the anus and vulva *(condyloma lata)*
- Saber shins *(bone abnormality of the lower leg)*
- Visual loss, CN VIII nerve deafness and interstitial keratitis
- Scarring of the skin around earlier lesions of the mouth, genitalia, and anus *(called rhagades)*

Remember: Hutchinson's triad → the combination of Hutchinson's teeth, interstitial keratosis, and deafness due to lesions of the eight cranial nerve.

Mucormycosis is an aggressive, opportunistic infection with a high affinity for afflicting individuals with:

- Tuberculosis
- Diabetes mellitus
- Hypertension
- Ischemic heart disease

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• **Diabetes mellitus**

The genera most commonly responsible for mucormycosis usually are Mucor or Rhizopus. Orbito-rhino-cerebral mucormycosis, the most common type, generally occurs in conjunction with sinus or nasal involvement. Mucormycosis also may affect other parts of the body, including the lungs, GI tract, or skin.

Diabetic patients are predisposed to mucormycosis because of the decreased ability of their neutrophils to phagocytize and adhere to endothelial walls. Furthermore, the acidosis and hyperglycemia provide an excellent environment for the fungus to grow.

Other patients **at risk** include the following:

- Patients on chronic antibiotics, steroids, or cytotoxic therapy
- Patients with chronic renal failure or liver problems
- Patients with transplants
- Patients with cancer, HIV, malnutrition or acidosis

Common **symptoms** include the following:

- Orbital and facial pain
- Sinusitis
- Headache
- Fever
- Visual changes
- Nasal discharge or stuffiness

The use of systemic **amphotericin B** is important in treating mucormycosis; its use, along with increased awareness of the disease, has decreased the mortality.

Herpangina is a non-treatable mild infection caused by a group A coxsackievirus. It is localized to the:

- Gingiva
- Posterior soft palate and nasopharynx
- Alveolar mucosa
- Hard palate

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• **Posterior soft palate and nasopharynx**

Herpangina refers to a **stomatitis** *(inflammation of the mouth)* caused by a strain of the Coxsackievirus. It is differentiated in clinical practice from Type 1 Herpes infection *(the cold sore virus)* by the fact that the ulcerations of **herpangina** are localized to the posterior soft palate and nasopharynx. **Remember:** Type 1 Herpes lesions are found typically more forward in the mouth on the tongue, gingiva, buccal mucosa and appear as vesicles *(small, clear blisters that ulcerate and crust)* around the mouth and on the lips.

Clinical features:

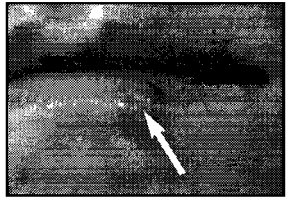
- Most commonly affects infants and young children
- Typically occurs during the summer
- Typically spreads via the fecal-oral route or via the respiratory droplets
- Symptoms are mild and short in duration *(no more than 1 week)*
- Sore throat and difficulty swallowing
- Mild fever
- Small vesicular or punctuate lesions with white base on posterior soft palate near uvula and anterior fauces of the tonsils ***See picture #71 in booklet.**

Important: The disease usually runs its course in less than a week. The treatment is palliative.

Note: Hand-foot- and mouth disease, is a highly contagious systemic infection caused coxsackievirus A of limited duration in which vesicular eruptions occur on the palms of hands, soles of feet and mucosa of the anterior part of the mouth. It is **uncommon** in the oropharyngeal area *(unlike herpangina)*.

Which type of **herpes virus** is associated with the lesion on the lower lip?

- HSV-1
- HSV-2
- HSV-3
- HSV-4



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- **HSV-1**

***Remember:** HSV-1 → the majority of oral herpes cases are caused by HSV-1 and the majority of genital herpes cases are caused by HSV-2.

Oral and para-oral presentations of Herpes Simplex Type 1 include:

- **Herpes labialis** *(also called fever blisters or cold sores)* is an extremely common disease caused by the herpes simplex virus Type 1, characterized by an eruption of small and usually painful blisters on the skin of the lips, mouth, gingiva, or the skin around the mouth. **Note:** The reason most patients suffering from recurrent herpes labialis rarely give a history of having had acute herpetic gingivostomatitis is that the primary infection was subclinical.
- **Acute herpetic gingivostomatitis** *(also known as* **primary** *herpetic gingivostomatitis)* generally affects children under the age of three and young adults. There are **prodromal symptoms** *(fever, malaise, irritability, headache, dysphagia, vomiting, lymphadenopathy)* 1-2 days prior to local lesions. Then small, yellowish **vesicles** form, which rupture quickly, resulting in shallow, round, discrete ulcers with an erythematous halo. It affects both the **free** and **attached mucosa.** A generalized **marginal gingivitis** may precede the ulcers. Treatment includes fluid intake, good oral hygiene and gentle debridement of the mouth. In healthy individuals the lesions heal spontaneously in 7-14 days without a scar.
- **Recurrent** *(secondary)* **herpetic stomatitis** generally occurs in adult patients and is triggered by trauma, fatigue, URI, stress, allergy or UV exposure, which causes the release *(or reactivation)* of the latent HSV-1 virus. This reactivation causes a recurrent infection *(i.e., cold sores)* on the **lips** *(that is bound to periosteum),* hard palate, attached gingiva and alveolar ridge. **Site-specificity is a characteristic manifestation.**

Primary herpes is most common in:

- Middle age adults
- Elderly people
- Children and young adults
- Infants

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• **Children and young adults**

Primary herpes is most common in children and young adults. Patients develop fever, irritability, regional lymphadenopathy and headache. Within days, the **gingiva** becomes intensely inflamed. Any part of the **oral mucosa and lips** may become involved. Vesicles then form and rupture a short time later to leave shallow ulcers covered with a gray membrane and surrounded by a red halo. These ulcers are **very painful.** These ulcers will heal on their own within 7 to 14 days.

After recovery from primary HSV infection, the virus **is not** cleared from the body, but, rather, it **lies dormant** in a non-replicating state, in the sensory nervous system *(specifically, the* **trigeminal ganglion***)*. Periodically, latency reactivates, allowing the virus to return to the skin or mucous membranes, where it causes a recurrent infection. **Cold sores** are a manifestation of **recurrent herpes simplex virus** infection around the mouth. The most common site is on the **lips.** Some factors that are often associated with a recurrent outbreak are: sunburn, fatigue, emotional upset, trauma, upper respiratory tract infection or menstruation. Often a day before the formation of vesicles there will be a tingling or itching of the skin or mucosa. Vesicles ulcerate and resolve the same as in the primary infection.

Histologically, the cytopathic effect *(CPE)* take the form of ballooning degeneration of the epithelial cells with loss of cohesion to adjacent cells. The nuclei are often multiple with margination of the chromatin around the intra-nuclear inclusions called **Lipschutz bodies.** These changes can be seen in scrapings taken from an unroofed vesicle *(these scrapings are called a* **Tzanck smear***)*.

The **Tzanck smear** is a rapid test done to diagnose infections caused by:

- Epstein-Barr virus
- Hepatitis viruses
- Tumor viruses
- Herpesviruses

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- **Herpesviruses**

The **T**zanck smear is done by smearing cells taken from a fresh blister or ulcer onto a microscope slide. The cells are stained with a special stain, such as Wright's stain, and then examined under a microscope for characteristic changes caused by a herpesvirus. Herpes causes giant cells with multiple nuclei. The shape of each nucleus appears molded to fit together with those adjacent. The background of the cell looks like ground glass and contains small dark spots called inclusion bodies *(***Lipschutz bodies***)*.

Other tests that can be used to diagnose herpetic lesions include:

- **Fluorescent staining** → cells show positive fluorescence when stained with fluorescent labeled HSV immune serum and globulin. This procedure is used to distinguish between herpes zoster and herpes simplex.
- **Isolation in tissue culture**
- **Antibody titers** *(anti HSV antibody titers)* → is a test for complement fixing or neutralizing antibody in acute and convalescent sera as well as on tissue sections *(this begins in one week and peaks in three weeks).*
- **Biopsied material** will show an intraepithelial cleft covered by an exudates of fibrin and polymorphonuclear leukocytes. The epithelium will exhibit **degenerative cells,** which include bizarre giant cells and cells with displaced chromatin with perinuclear halos and inclusions.

The treatment for herpes is primarily supportive:

- Analgesics
- Topical anesthetics prior to eating
- Maintain electrolyte balance
- Antiviral agents

The **varicella-zoster virus** *(VZV)* is the cause of:

- Chickenpox and herpes zoster *(also called shingles)*
- Herpangina and hand-foot- and mouth disease
- Mumps and infectious mononucleosis
- Hepatitis A and Hepatitis B

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• **Chickenpox and herpes zoster *(also called shingles)***

The varicella zoster virus *(VZV)* is a member of the **herpes virus** group. It causes the disease **chickenpox** *(varicella)* and **shingles** *(herpes zoster).* The virus is very contagious and may be spread by direct contact or droplets.

Chickenpox is primarily a **disease of childhood,** which peaks at school-age in the winter and spring. It is characterized by the appearance on skin and mucous membranes of successive crops of typical **pruritic vesicular lesions** that are easily broken and become scabbed. It is generally accompanied by mild constitutional symptoms *(fever, malaise).* It is **most contagious** one day before the onset of the rash and until all the vesicles have crusted. It is relatively benign in children, but adult infection may be complicated by pneumonia and encephalitis. **Note: ZIG** *(Zoster Immune Globulin)* reaches morbidity in high-risk children.

Shingles *(herpes zoster)* is the result of **reactivation of a latent varicella-zoster** virus that may have remained within the body from a childhood case of chickenpox. The virus reaches the sensory ganglia of the spinal and cranial nerves, producing an inflammatory response. It is characterized by **painful vesicles** that occur on the skin or mucosal surfaces along the distribution of a sensory nerve in a **distinctive unilateral pattern.**

Note: The histology for both chickenpox and shingles shows the **same cytopathic effect** as seen in herpes simplex.

Over 90% of **primary herpes simplex** viral infections are:

- Manifested as ANUG
- Associated with HIV
- Subclinical
- Characterized by severe lymphadenopathy and acute dermatitis

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• **Subclinical**

Herpes simplex is one of the most common viral diseases effecting man. The primary infection, which is known as **primary herpetic gingivostomatitis,** is most common in young children *(under five years old).* It usually occurs in a child who has had no contact with the Type 1 herpes simplex virus, and who therefore has no neutralizing antibodies. It may also affect young adults *(15-25).* Nearly all primary infections *(90%)* are of the **subclinical type** *(they may only have* **flu-like symptoms***)* and one or two mild sores in the mouth which go unnoticed by the parents.

In other children, the primary infection may be manifested by acute symptoms, which is known as **acute herpetic gingivostomatitis.** These symptoms include fever, irritability, cervical lymphadenopathy, fiery red gingival tissues and small yellowish vesicles that rupture and result in painful ulcers on the free and attached mucosa. The most serious potential problem in a child with this infection is dehydration due to the child not wanting to eat or drink because of the pain. **See picture #72 in booklet.**

The treatment is **supportive** and aimed toward the relief of the acute symptoms so that fluid and nutritional intake can be maintained. Primary herpetic gingivostomatitis usually runs a course of 12-20 days, and the ulcers heal without scarring.

Note: Corticosteriods are contraindicated in patients with herpes simplex infections.

After the initial primary attack during the early childhood period, the **herpes simplex** virus remains inactive **most commonly** in the:

- Geniculate ganglion
- Ciliary ganglion
- Trigeminal ganglion
- Pterygopalatine ganglion

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• **Trigeminal ganglion**

The inactive herpes simplex virus resides in **sensory nerve ganglia** *(most commonly, the trigeminal ganglion)*, but will often reappear later as the familiar **"cold sore"**, usually on the outside of the lips. This disease is referred to as **"recurrent herpes labialis".** Emotional stress, trauma and excessive exposure to sunlight have been implicated as factors for the appearance of the recurrent herpetic lesions on the lip. **Acyclovir 5%** ointment *(Zovirax)* as well as valacyclovir, and famciclovir have been successful in reducing the duration and severity of these sores.

Remember:

1. **Herpes Simplex Type I** *(primary herpetic gingivostomatitis, recurrent herpes labialis)* is transmitted by direct contact. It affects the lips, face, skin and oral mucosa.
2. **Herpes Simplex Type II** *(herpes genitalis)* is spread by sexual contact. It affects the mucosa of the genital and anal regions.

Note: Genital herpes may have serious consequences in pregnant women because the virus can be transmitted to the infant during vaginal delivery. The virus can cause damage to the infant's central nervous system and / or eyes.

Remember: The **primary** infection of herpes simplex can range from **subclinical** *(asymptomatic, which is most common)* to severe systemic infections.

Nikolsky's sign is a characteristic feature of:

- Herpes zoster
- Lupus erythematosus
- Lichen Planus
- Pemphigus

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• **Pemphigus**

Pemphigus is a term used to describe blistering of the skin caused by binding of **antibodies** to the surface of the cells of the outer layer of the skin, the epidermis. In **pemphigus vulgaris,** the most common form of pemphigus, there are **IgG** antibodies that bind to the cell surfaces of epidermis of the skin as well as the epithelium lining mucosal surfaces such as the mouth. As a result, patients develop severe oral ulcerations, and may also have inflammation or erosions of the lining of the eye and eyelids *(conjunctiva)*, the nasal mucosa, or the genital mucosa. Half of the patients also develop blisters or erosions of the skin, often in the head and neck area. It seldom occurs before the age of 30 *(usually between 30 and 50)* and occurs more frequently in Jewish people.

There are four types of Pemphigus:

1. Pemphigus vulgaris → **most common**
2. Pemphigus vegetans
3. Pemphigus foliaceus
4. Pemphigus erythematosus

Oral lesions are often the first manifestation of the disease. Intact bullae are rarely seen in the oral cavity, instead, large areas of ulceration and erosions are often seen that are covered by a white or blood-tinged exudates. Sometimes, areas of epithelium will slide off simply by rubbing of an apparently unaffected area *(this is termed* **Nikolsky's sign***)*. This disease is often fatal without therapy, which includes high-dose systemic steroids or chemotherapy *(for example,* **methotrexate***)*.

Important histological features: The vesicles and bullae are formed entirely intraepithelially, just above the basal layer of cells *(called* **suprabasilar** *vesicles)*. There is intercellular edema and loss of intercellular bridges with loss of cohesiveness. This is called **acantholysis.** Clumps of cells are often found floating free in the vesicle space *(these cells are called* **Tzanck cells***)*.

The oral lesions of **benign mucous membrane pemphigoid** most commonly present as a:

- Candidiasis
- Hairy leukoplakia
- Desquamative gingivitis
- Hemorrhagic mass

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• **Desquamative gingivitis**

Benign mucous membrane pemphigoid *(BMMP)* is an autoimmune or "self-allergy" disease in which a patient's own circulating antibodies become altered so that they attack the fibrous attachment of the skin and membrane epithelium to the underlying connective tissues. Women are more commonly affected than men and the disease is usually diagnosed between the ages of 40-60 years. The typical lesion is a small or large, clear-fluid blister which breaks fairly rapidly in the mouth to leave a flat white, somewhat tender ulcer with a thin red line around it. The gingivae are especially likely to be involved, resulting in sloughing during eating or tooth brushing **("desquamative gingivitis").** Systemic steroid therapy has provided adequate management of BMMP. **Note:** Conjunctival involvement may lead to blindness.

Remember: Nikolsky's sign, which is an indication of **pemphigus vulgaris,** may also be found in BMMP. The sign occurs when apparently normal epithelium may be separated at the basal layer and rubbed off when pressed with a sliding motion.

While **similar** in its clinical presentation to **pemphigus vulgaris,** it is much less severe and the involved antibodies attack the attachment fibrils *(Type VII collagen)* of the basement membrane rather than the desmosomal attachments between epithelial cells, as occurs in pemphigus.

Important: Histologically, the major difference between BMMP and pemphigus vulgaris is that the vesicles in BMMP are **subepidermal** and there is no **evidence of acantholysis** → in pemphigus vulgaris there is **acantholysis and a suprabasilar vesicle.**

The common wart or **verruca vulgaris** is caused by the:

- Human papillomavirus *(HPV)*
- Adenovirus
- Epstein-Barr virus
- Parvovirus

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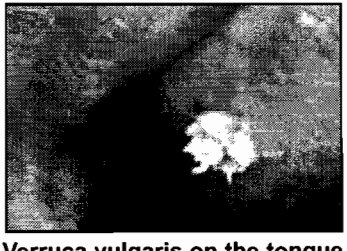
• **Human papillomavirus *(HPV)***

This is the same virus that causes genital warts which leads to dysplasia and cervical cancer. Some estimates of the prevalence of HPV infection in the population range as high as 79%. Warts may arise on any skin surface, but occur most commonly at acral sites. Although treatment may remove the wart, the virus remains latent within the skin cells. However, treatment may diminish spread of HPV in the skin of the infected patient and possibly to uninfected contacts.

Verruca vulgaris *(also called squamous papilloma)* has an incubation period from about six weeks to a year. Although it is primarily a lesion of the skin, it may occur in the oral cavity, particularly on the **lips** and **palate.** Clinically, it is a **sessile, soft, cauliflower-like** lesion. If excised, they usually do not recur, but **autoinoculation** is possible. **Note:** Intraorally, that is how most cases develop.



Verruca vulgaris on the tongue

The **verruciform xanthoma** is a:

- Herpes look-alike lesion
- Papilloma look-alike lesion
- Mucocele look-alike lesion
- Melanoma look-alike lesion

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• **Papilloma look-alike lesion**

This unique lesion occurs in middle-aged and older individuals, usually 40-70 years of age. There is a strong female predilection *(1:2 male/female ratio)* and the usual intraoral locations are the **gingiva and alveolar mucosa,** but any oral mucosal site may be involved. The lesion appears as a well-demarcated, soft, painless, sessile, slightly elevated mass with a white, yellow/white, or red color and a papillary or roughened surface *("verruciform" = with pointed projections, warty)*.

Benign Soft Tissue Tumors

Tumor	Histogenesis *(Etiology)*	Clinical Characteristics	Treatment and Prognosis
Papilloma	Squamous epithelium *(epithelial origin)*	Sessile or pedunculated, whitish, cauliflower-like mass; tongue, lips, soft palate common sites	Conservative excision; recurrence rare
Fibroma *(Irritation or traumatic fibroma)*	Fibrous connective tissue *(connective tissue origin)*	Smooth, soft to firm nodule; buccal mucosa, lips, tongue common sites	Conservative excision; recurrence rare
Lipoma	Adipose tissue *(connective tissue origin)*	Smooth or lobulated, sessile or pedunculated, soft yellowish mass; vessels visible over surface; floor of mouth, buccal mucosa, tongue common sites	Conservative excision; recurrenee rare
Verruciform xanthoma	Unknown; not associated with any systemic condition	Adults; 40-70 yrs. of age, female predilection; gingiva & alveolar mucosa are most common sites; well demarcated soft, painless, sessile mass	Simple excision; no recurrence

Inflammatory papillary hyperplasia occurs on the:

- Soft palate
- Buccal mucosa
- Hard palate
- Tongue

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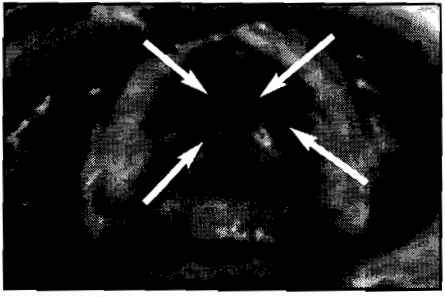
• Hard palate

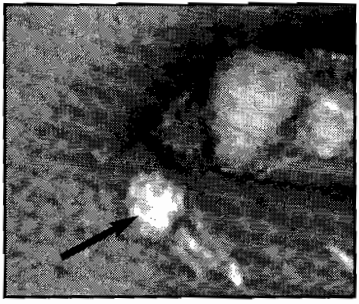
Entity	Histogenesis *(Etiology)*	Location	Clinical Characteristics	Treatment and Prognosis
Inflammatory fibrous hyperplasia: **"Epulis fissuration"**	Ill-fitting prosthesis *(dentures)*	Area of denture borders; maxillary arch is most common	Rolls of tissue in muco-labial fold, pink, elongated, firm ulceration, soft lesion	Surgical excision and re-evaluate prosthesis with possible remaking of dentures or relining dentures
Inflammatory papillary hyperplasia: **"Palatal papillomatosis"**	Poor oral hygiene & ill-fitting prosthesis *(dentures)*	Hard palate *(vault)*	Numerous red papillary projections, soft lesion	Surgical excision and correct prosthesis

"Epulis fissuratum"



"Palatal papillomatosis"

The picture below shows a **benign epithelial neoplasm** which appears as a pedunculated, whitish cauliflower-like mass on the lower lip. The **most likely** diagnosis is:



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• **Papilloma**

The papilloma is a benign exophytic papillary growth of stratified squamous epithelium. **Note:** The common wart, or **verruca vulgaris,** is a frequent tumor of skin analogous to the oral papilloma.

Clinical features:

- Adults
- Anywhere on the oral mucosa
- Sessile or pedunculated exophytic growth
- Papillary *(cauliflower-like)* appearance
- Long duration
- May show considerable keratin and in some instances appear white clinically. If it has little keratin on the surface it will appear pink.

Treatment and Prognosis: Conservative excision; recurrence is rare.

Remember: A **fibroma** is a benign neoplasm of **connective tissue** origin.

Leukoedema:

- Appears as a diffuse gray-brown pigmentation of the mucosa or skin
- Appears as a slowly enlarging swelling of the jaw
- Will disappear when applying the clinical "stretch test"
- Is a benign, exophytic, usually pigmented, congenital lesion of the skin

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- **Will disappear when applying the clinical "stretch test"**

The appearance varies from a filmy opalescence of the mucosa in the early stages to a more definite grayish-white cast with a coarsely wrinkled surface in the later stages. The lesions usually occur bilaterally and are most noticeable along the occlusal line in the bicuspid and molar region. **Diagnostically,** one can stretch the tissue and the white essentially disappears. **Important:** Leukoplakia would **not disappear** when stretched.

Important point: Leukoedema appears to be **simply a variant** of normal mucosa and no treatment is necessary; merely diagnosis. **See picture #73 in booklet.**

The differential diagnosis should include:
- Leukoplakia
- Smokeless tobacco keratosis
- Frictional keratosis
- White sponge nevus
- Hereditary benign intraepithelial dyskeratosis

Histologically, in leukoedema, the epithelium is parakeratotic and acanthotic, with marked intracellular edema of spinous cells.

Note: The white appearance of leukoedema is caused by water within the spinous cells causing the light to reflect back as whitish.

Actinic cheilitis is caused by:

- A virus
- Stress
- Chronic and excessive exposure to the ultraviolet radiation in sunlight
- Certain medications

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• **Chronic and excessive exposure to the ultraviolet radiation in sunlight**

The short-term effects of exposure to UV light are transient, but the cumulative long-term effects produce irreversible damage *(actinic cheilitis)*, usually to the lower lip of exposed individuals. Actinic cheilitis, a variant of oral leukoplakia, is considered to be the labial counterpart of solar *(actinic)* keratosis *(a precursor of SCC of the skin)*. The lips appear dry, mottled, and opalescent with slightly elevated white or gray plaques and that cannot be stripped off. Isolated areas of hyperkeratotic callus may also be evident as well as **loss of elasticity and definition of the vermilion border. See picture #74 in booklet.**

Other clinical signs include:

- Erythematous or hemorrhagic areas
- Parallel marked folds
- An unobtrusive "chapped lip" appearance

Malignant change is manifested clinically by areas of more diffuse cheilitis and ulcerations of relatively long duration. Although degenerative changes have been observed predominantly in men after the age 40, the condition now is increasingly recognized in younger men.

Important: This condition is considered **premalignant** and may lead to squamous cell carcinoma. It should be treated accordingly.

An **incisional biopsy** is indicated for which of the following lesions?

- A .5 cm papillary fibroma of the gingiva
- A 2 cm exostosis of the hard palate
- A 2 cm area of Fordyce's disease of the cheek
- A 3 cm hemangioma of the tongue
- A 3 cm area of leukoplakia of the soft palate

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• A 3 cm area of leukoplakia of the soft palate

Leukoplakia is a **premalignant** lesion. This means that if left untreated, some of the lesions progress to carcinoma. It is because of this chance of malignant transformation that **all leukoplakias should be biopsied.**

It is a slowly developing change in a mucous membrane characterized by thickened, white, firmly attached patches that are slightly raised and sharply circumscribed. It is most often caused by tobacco irritation *(especially pipe).* It is more common in older men. Lesions of the **floor of the mouth** and **base of the tongue** are **most aggressive.** Most display no dysplasia but can develop into malignancy. In all cases, leukoplakia **must be completely excised. See picture #75 in booklet.**

Explanation of lesions on front of card:

- **Papillary fibroma** → is a benign neoplasm of **connective tissue** origin.
- **Exostosis** of the hard palate → tori *(palatal or mandibular):* most common exophytic lesions, slow-growing benign knots of bone.
- **Fordyce's disease** *(or granules)* → ectopic sebaceous glands in the oral mucosa. They are present in over 75% of adults. They usually appear as yellow, sometimes yellow white submucosal clusters that are essentially normal. **See picture #76 in booklet.**

Note: When mechanical irritation produces a white lesion it is called **frictional keratosis.**

A **white patch** on the oral mucosa that cannot be rubbed off and cannot be ascribed to any other disease is **most probably:**

- Squamous cell carcinoma
- Lichen planus
- Erythroplakia
- Leukoplakia

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• **Leukoplakia**

The etiology of leukoplakia is thought to be a varied one. Possible etiologic factors include tobacco, alcohol, and oral sepsis. It is most often due to **tobacco use.** Some investigators believe that **pipe smoking** is most harmful. **See picture #77 in booklet.**

Leukoplakia is a clinical white patch or plaque on the oral mucosa which will not rub off and which cannot be characterized as any specific disease. Most reports indicate that leukoplakia is more common in elderly men. Although less common than leukoplakias, **erythroplakias,** have a **much greater potential** for becoming malignant.

***Important:** Any white or red lesion that does not resolve itself in two weeks should be reevaluated and considered for biopsy to obtain a definitive diagnosis.

The term **carcinoma in situ** is applied to mucosal lesions which resemble leukoplakia in all respects **except** that dysplasia is very pronounced and involves almost all epithelial layers. It shows **no tendency** to invade or metastasize to other tissues.

The **clinical differential diagnosis** of a white patch should include:

- Leukoplakia
- Lupus erythematosus
- White sponge nevus
- A chemical / thermal burn
- Candidiasis
- Lichen planus
- Migratory glossitis / stomatitis

Which of the following is a condition in which small accumulations of saliva gather in the skin folds at the commissural angles and are subsequently colonized by yeast organisms causing fissured, erythematous alterations of the skin?

- Squamous cell carcinoma
- Angular cheilitis
- Verruca vulgaris
- Stomatitis nicotina

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• **Angular cheilitis** → *also called* **Perleche**

It is also associated with the loss of vertical dimension.This situation is generally observed in elderly patients.The corners of the mouth become painful, irritated, red, cracked, and scaly. The fungus Candida albicans *(thrush)* may grow in the corners of the mouth, keeping them sore.

It occurs in individuals that habitually lick their lips and deposit small amounts of saliva in the commissural angles.

Note: Nystatin will invariably eliminate the fungal infection.

See picture #78 in booklet.

White sponge nevus produces mucosal lesions that are:

- Asymptomatic, white, folded and spongy
- Painful, white, creamy white, or yellow slightly raised papules
- Painful, with a raw, red, or bleeding surface
- Asymptomatic, and will disappear when applying the clinical "stretch test"

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• **Asymptomatic, white, folded and spongy**

White sponge nevus appears to follow a hereditary pattern as an autosomal dominant trait. It has no sex preference. This mucosal abnormality is congenital in many instances, however, it may occur in childhood or adolescence. The most common location is the **buccal mucosa, bilaterally,** followed by the labial mucosa, alveolar ridge and floor of the mouth. The gingival margin and dorsal tongue are almost never affected. The mucosa appears thickened and folded with a soft or spongy texture and peculiar pearly white hue. **See picture #79 in booklet.**

A feature that is **unique to white spongy nevus** is an eosinophilic condensation in the perinuclear region of the cells in the superficial layers of the epithelium.

Important: There is no treatment for white sponge nevus, however, since the condition is perfectly **benign**, the prognosis is excellent. There are no serious clinical complications. **Note:** It is often **mistaken for leukoplakia.**

Note: Hyperkeratosis is an abnormal increase in the thickness of the keratin layer *(stratum corneum)* of the epithelium. It is one of the **most common** white cheek lesions of the oral mucous membranes *(often in an area of chronic cheek biting)* and presents as being thick and scaly.

Hairy tongue is a condition characterized by hypertrophy of the:

- Filiform papillae
- Fungiform papillae
- Circumvallate papillae
- Foliate papillae

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• **Filiform papillae**

Hairy tongue is a **benign** condition of the tongue. The dorsum of the tongue appears furry due to the elongated papillae. The color varies from yellowish-white to brown or black. **See picture #80 in booklet.**

The four types of papillae present on the tongue:

1. **Filiform** → **most numerous,** small cones arranged in "V"– shaped rows paralleling the sulcus terminalis. They are characterized by the **absence of taste buds** and **increased keratinization.**
2. **Fungiform** → scattered among the filiform papillae, they are flattened, mushroom shaped and found mainly at the **tip and lateral margins.**
   Circumvallate → largest, have circular shape. Arranged in an inverted "V"– shaped row toward the back of the tongue. Associated with the ducts of **von Ebner's glands.** They are the **least numerous** of all papillae.
3. **Foliate** → found on **lateral margins** as 3-4 vertical folds.

Note: Taste buds are **present** on the fungiform, circumvallate and foliate papillae only.

Lace-like white striae present on the buccal mucosa bilaterally may represent:

- Lupus erythematosus
- Erythema multiforme
- Pemphigus vulgaris
- Lichen planus

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• **Lichen planus**

These lace-like white striae, the so-called **Wickham's striae,** are a classic presentation of lichen planus. They are often bilateral and symmetrical in distribution.

Lichen planus is a fairly common inflammatory disease that usually affects the skin, the mouth, or sometimes both. It affects women slightly more than men, and occurs most often in middle-aged adults. The cause of lichen planus is unknown *(possibly autoimmune).* Lichen planus of the mouth most commonly affects the buccal mucous membrane. It may also been seen on the tongue, lips, hard palate and gingiva. The lace-like striae are usually asymptomatic, but sometimes the patient may complain of a burning sensation. The intraoral lesions respond to topical steroid therapy. In addition to the usual form of lichen planus, there are two other forms, **bullous** and **erosive.** In the **bullous form,** fluid-filled vesicles project from the surface. In the **erosive form,** the lesions are intensely red or raw-appearing. When these lesions of erosive lichen planus involve the gingiva, they resemble desquamative gingivitis. **See picture #81 in booklet.**

The microscopic appearance of lichen planus is **characteristic and pathognomonic:**

- **Hyperparakeratosis** with thickening of the granular cell layer.
- Development of a **"saw tooth"** appearance of the rete pegs.
- **Degeneration** of the basal layer of cells.
- Infiltration of **inflammatory cells** into the subepithelial layer of connective tissue.

A white patch which, when scraped or removed from the oral mucosa, leaves a raw, bleeding surface **most likely** is:

- Leukoplakia
- White spongy nevus
- Candidiasis
- Lichen planus

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• **Candidiasis**

Oral candidiasis also known as **thrush,** causes white, curd-like patches in the mouth or throat. These patches typically appear on the tongue, inside of the cheeks, or on the palate. Oral candidiasis typically occurs in people with abnormal immune systems. These can include people undergoing chemotherapy for cancer, people taking immunosuppressive drugs to protect transplanted organs, or people with HIV infection.

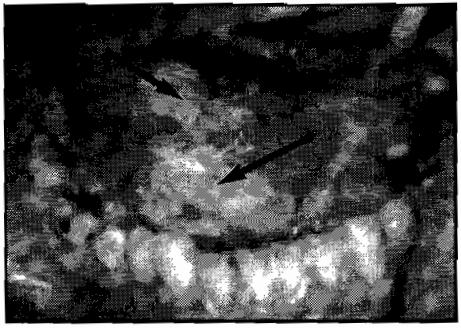
Candidiasis is caused by a yeast-like fungus, **C. albicans**, which causes an inflammatory, pruritic infection characterized by a thick, white discharge. It appears as diffuse, curly or velvety white mucosal plaques on the cheeks, palate and tongue that can be **wiped off,** leaving a red, raw or bleeding surface.

This yeast-like fungi is a normal inhabitant of the oral cavity and vaginal tract, however it is normally held in check by the indigenous bacteria of these areas. The treatment for oral candidiasis is topical through the use of lozenges *(also called trouches)* and mouth rinses, the most widely used is **nystatin.**

Notes:

1. **Acute pseudomembranous** candidiasis is the most common form of oral candidiasis and is usually found on the buccal mucosa, tongue and soft palate. **Oral cytology** smears are useful for a diagnosis *(it will reveal budding organisms with branching pseudohyphae).*
2. **Angular cheilitis** *(Perleche)* has also been linked to C. albicans.
3. Factors that may **stimulate** Candida growth include: the extended use of antibiotics, steroids, diabetes, pregnancy, or a deficiency in iron, folate, Vitamin B_{12} or zinc.

The **"white patch"** seen below has been present for nine months on a patient who is a ***heavy pipe smoker.*** What is the **treatment** of choice?



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• **Biopsy**

Clinically, this is **leukoplakia** and should always be biopsied due to the possibility of it being a pre-malignant lesion. **Remember: Pipe smoking** is thought to be one of the most important predisposing etiologic factors for the development of a leukoplakia.

Important: The floor of the mouth, tongue and lower lip are the regions at greatest risk for carcinoma occurring in leukoplakia.

Stomatitis nicotina is related to pipe smoking *(as well as cigar smoking)* and occurs exclusively on the palate. It affects males predominantly. The palate first appears red and inflamed. Soon it develops a diffuse, grayish-white, thickened, multinodular popular appearance with a small red **"spot"** in the center of each tiny nodule. This "spot" corresponds to the orifices of palatal salivary gland ducts. The treatment for this condition is the cessation of smoking. It is usually not considered to be a pre-malignant lesion. **See picture #84 in booklet.**

Note: In **India,** the use of smokeless tobacco in various forms is very popular. This habit, which usually involves the chewing of a **betel quid** *(combined areca nut, betel leaf, tobacco and slack lime)*, has led to the development, in a large proportion of users, of a unique generalized fibrosis of the oral soft tissues, called **oral submucous fibrosis.** It typically affects the buccal mucosa, lips, retromolar areas and the soft palate. **Early lesions** present as a blanching of the mucosa, imparting a mottled, marble-like appearance. **Later lesions** demonstrate palpable fibrous bands running vertically in the buccal mucosa and in a circular fashion around the mouth opening or lips. As the disease progresses the mucosa becomes stiff, causing difficulty in eating and considerably restricting the patient's ability to open the mouth *(trismus)*. If the tongue is involved, it becomes stiff and has a diminished size. There is a fibroelastic transformation of the juxta-epithelial connective tissues and an **increased risk of oral carcinoma** from the tobacco of the quid.

Which of the following is characterized by irregularly shaped red, map-like smooth swollen patches on the tongue which change daily in size and shape?

- Fissured tongue
- Macroglossia
- Geographic tongue
- Hairy tongue

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• **Geographic tongue**

***It is also called erythema migrans, benign migratory glossitis, wandering rash of the tongue, erythema areata migrans, and stomatitis areata migrans.

Geographic tongue is a harmless and very common condition in which there is desquamation of the **filiform papillae.** It is characterized by having one or more irregularly shaped patches on the tongue. The center area is redder than the rest of the tongue and the edges of the patch are whitish in color. These patches appear and remain for a short time, heal, then reappear at another site. The patches usually do not respond to treatment but disappear spontaneously. **Note:** The patient may complain of a slight burning of the tongue. **See picture #82 in booklet.**

Fissured tongue *(also called scrotal tongue)* is characterized by a deep median fissure with laterally radiating grooves. The lateral grooves vary in number but are usually symmetrical in arrangement. It is rare in children and increases in incidence with age. The fissuring occurs across the dorsum of the tongue and is usually asymptomatic, but may become painful if infected with Candida Albicans. **See picture #83 in booklet.**

Note: Fissured tongue is found in **Melkersson–Rosenthal Syndrome** *(along with cheilitis granulomatosum and facial nerve paralysis).*
Remember: Geographic tongue often occurs in **association** with fissured tongue.

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Legend

Major Topic	Abbreviation
Behavioral Sciences	BS
OSHA	OSHA
Public Health	PH
Infection Control	IC

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Arrange the following **seven steps** in the educational process in their correct order.

- Expressing needs
- Stimulating motivation
- Recognizing needs
- Evaluating results
- Reinforcing learning
- Setting goals
- Acting to achieve goals

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Educational Process

- **Step 1: recognizing needs** → the dentist recognizes educational needs as he checks for treatment needs. **Then the dentist helps the patient to recognize his own needs.**

- **Step 2: expressing needs** → the dentist records educational needs and **helps the patient to state his own needs.**

- **Step 3: stimulating motivation** → motivation arouses **and** maintains interest. The dentist may appeal to inner needs or use artificial stimuli.

- **Step 4: setting goals** → these may be short-range or long-range guides to activity. **They must be meaningful, attractive and attainable.**

- **Step 5: acting to achieve goals** → activity is **necessary** to learning. The activity should be directed toward specific goals.

- **Step 6: reinforcing learning** → review and **repetition aid in retention of learning.**

- **Step 7: evaluating results** → this aids in judging what the patient has learned and how **effective the dentist's teaching has been.** This can help clarify or redefine the goals.

Note: Keep in mind that each learning situation **won't follow** these steps in exact sequence, **but** most situations will include all of the steps in some form.

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Which of the following statements are **true** concerning **needs** and **learning**?

- **Needs** are driving forces that **prompt a person to act**
- The **process of learning is continual and multiple**
- **Learning occurs** as a person attempts to **satisfy his needs**
- Telling a person **what he needs** may convince him that a behavior change is desirable
- **Expressing needs** helps to pinpoint them for the **dentist and patient**
- **Recording educational needs** can be as important as **recording treatment needs**
- All of the above statements are true

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- **All of the above statements are true**

Remember → in terms of needs and learning:

- **Motivation stimulates** a person to **act on his needs.**
- **Motivation** is a **fundamental** part of **every learning situation.**
- **Motivation** may be **artificial or built-in.**
- **Needs** and **goals** may **provide motivation.**
- Patients rarely learn **without** some kind of motivation.
- **Motivation** arouses and **maintains interests.**
- **Short-range goals** are less remote and **more easily attained.**
- **Goals** should be **attractive** and **attainable** in order to be **meaningful.**
- **Goal-directed activity** is necessary for **learning.**

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Behavior is strongly affected by which of the following **psychosocial factors?**

- Attitudes
- Beliefs
- Values
- Job strain
- Family
- Society
- Culture
- Education

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- **Job strain**

Behavior can be defined as a **determined, purposeful unit of activity.**

Each term can be further examined:

- **Determined** → the assumption that **behavior is lawful and has determinants.**
- **Purposeful** → the assumption that **behavior is goal-oriented,** that it seeks to achieve positive and reduce negative need or motivated states.
- **Unit of activity** → what a person does that can be reported or described as discrete elements.

Example: Teeth **do not** behave, individuals do. Observing that a pulpal or periodontal problem exists is a **common behavior for the dentist.** Avoiding the dentist, even though an objective need exists and the patient requires treatment, is a **common behavior for patients.** Both meet the criterion of being determined, purposeful units of human activity.

Behavior management is the means by which the dental health team effectively and efficiently performs treatment for the patient and, at the same time, instills a positive attitude.

Note: Most researchers believe that changes in behavior are a **prerequisite** to changes in attitude.

The **principal nonverbal cue** that two or more people can use to regulate verbal communication is:

- Posture
- Facial expression
- Eye contact
- Body position *(physical proximity to another)*
- Gesticulations

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• **Eye contact**

Eyes should be directed toward the patient and engage the patient's eyes as frequently as is comfortable for the talker and the listener.

Communication is basic to all aspects of the dentist-patient relationship. Without proper communication, whether it be verbal or nonverbal, the dentist-patient relationships will fail.

Remember: "Acceptable" nonverbal behavior varies with age, sex, ethnic background, geographical region, culture and situation.

When presenting treatment plans always use **open-ended questions** *(those that cannot be answered with a simple "yes" or "no")*. These types of questions are the **most effective** in helping patients to express their understanding of the proposed treatment plan. When reviewing oral hygiene, have the patient repeat what you have gone over with him or her.

When communicating with children, try to reinforce **positive behavior** by telling the child **exactly** or **specifically** what he is doing well.

Which mechanism listed below by which **behavioral responses are learned** operates by the simple process of association of one stimulus with another?

- Operant conditioning
- Classical conditioning
- Observational learning

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• **Classical conditioning** *(also called pavlovian or respondent conditioning)*

A stimulus leads to a response. If individuals in white coats are the ones who give painful injections that cause crying, the sight of an individual in a white coat soon may provoke an outburst of crying. If this is **not reinforced,** the conditioned response will no longer occur. This is referred to as **extinction of the conditioned behavior.**

Operant conditioning has as the **basic principle that the consequence of a behavior is in itself a stimulus** that can affect future behavior. In other words, the consequence that follows a response will alter the probability of that response occurring again in a similar situation. There are **four basic types of operant conditioning,** distinguished by the nature of the consequence. They are: positive reinforcement, negative reinforcement, omission and punishment.

Observational learning *(modeling)* is acquired through initiation of behavior observed in a social context. There are two distinct stages in observational learning: **acquisition** of the behavior by observing it and the actual **performance** of the behavior. Having an open dental office design may aid the dentist in this method. Children can watch other cooperative children and this may rub off on them.

Remember: Relative maturity modifies expectations of a child's behavior in that a child **cannot** be expected to learn a mode of behavior **until** he has matured to a stage at which he is ready for such learning.

Which kind of aggression is an act of hostility unnecessary for self-protection or preservation that is **directed toward an external object or person?**

- Destructive aggression
- Inward aggression
- Constructive aggression

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• **Destructive aggression**

Constructive aggression is an act of **self-assertiveness** in response to a threatening action for purpose of **self-protection and preservation.**

Inward aggression is destructive behavior that is **directed against oneself.**

Note: An **aggressive personality** is a personality with behavior patterns characterized by irritability, tantrums, destructiveness or violence **in response** to frustration.

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Match the type of question on the left to an example of that particular question type on the right.

• **Direct** question	You are not afraid of needles, are you?
• **Probing** question	How are you doing with your brushing and flossing?
• **Laundry** list question	How are you?
• **Open-ended** question	Is it easier to hold the brush this way?
• **Leading** question	What else did you notice about your gums?
• **Facilitating** question	Is the pain throbbing, aching, dull or sharp?

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Direct question→ Is it easier to hold the brush this way?
***These types of questions ask the patient for a **specific** bit of information.

Probing question → What else did you notice about your gums?
***These types of questions ask for **more specific** information that the patient offers **spontaneously.**

Laundry list question → Is the pain throbbing, aching, dull or sharp?
***These types of questions ask the patient to respond **from among** a list of alternative adjectives or descriptions provided.

Open-ended question → How are you doing with your brushing and flossing?
***These types of questions request information in the patient's own words and **specify** a general content area.

Leading question → "You are not afraid of needles, are you?"
***These types of questions entice a patient to **answer a specific way.**

Facilitating question → "How are you?"
***These types of questions encourage the patient to say more, **without** specifying an area or topic.

Note: These are all **verbal communication behaviors.**

What type of patient is **usually** considered to be the most **"difficult"** patient?

- The **"know-it-all"** patient
- The **anxious** patient
- The **shy** patient
- The **talkative** patient
- The **stubborn** patient

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- **The anxious patient**

Most dentists say that they themselves become anxious with an anxious patient.

Most patients who are anxious have had a **traumatic experience** in a dental or medical setting.

Some things to do to **reduce** patient anxiety:
- **Explain** procedures **before** doing them.
- **Forewarn** about the **possibility of pain.**
- **Give patient some control** over the procedures and pain *(ex., raise your hand if you feel anything).*
- **Build trust** between you and the patient.

Note: Watching a patient's **eyes or eyebrows** will give a good indication whether a patient is feeling pain during dental treatment.

Which type of parents have **excessively demanding** attitudes?

- Neglectful
- Overprotective
- Manipulative
- Hostile

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• **Manipulative parents**

***These demanding attitudes usually **start** with appointment times and can extend to directing the course of diagnosis or treatment.

Overprotective parents → they insist on remaining with the child in the dental operatory, **regardless** of the situation or age of the child. **Note:** Pointing to the lack of apprehension of a young child and the importance of establishing **one-to-one relationship** between the child and dentist usually satisfies most overprotective parents.

Hostile parents → they question the necessity for treatment *(this is usually not due to curiosity, but distrust)*.

Neglectful parents → they fail to maintain appointments, miss recall visits or do not oversee oral hygiene of the child.

Notes:

1. **Overprotective parents** usually have children who are **shy, docile and manageable.**
2. **Children with defiant behavior** are usually said to be **stubborn or spoiled.**
3. For children who are **hostile or angry,** try to **identify the underlying source** of these emotions *(this holds true for adult patients as well)*.

Which of the following refers to a state or feeling of apprehension, uneasiness, agitation or uncertainty resulting from the anticipation of some threat or danger, usually of intrapsychic rather than external origin, whose source is generally **unknown or unrecognized?**

- Fear
- Anxiety
- Stress

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• **Anxiety**

Fear generally refers to the **anticipation of a threat** elicited by an external object that is generally agreed to be harmful. **Note:** In evaluating a patient's **dental fears,** take note of *what he / she* **says,** *how he / she* **behaves,** *and also how he / she* **appears** while in the dental office.

Note: Fear is distinguished from **anxiety** on the basis of the person's ability to locate the threatening agent "out there" and to recognize the clear presence of a behavior that will reduce perceived danger. **Patients who are fearful or anxious will do anything to put off making dental appointments.**

Stress → is a term used to describe a general disturbance in psycho-physiological adaptation. It implies a person who is being maladaptively influenced by more than one negative or aversive factor.

Note: Of the three terms, **stress is most associated with response aspects.**

Stress, anxiety and fear are simultaneously negative or aversive emotional states, full of symptoms that can motivate through a process of threat appraisal. The interaction of the intensity of an emotional response with threat appraisal determines the content of the behavior that will follow → whether or not to show up at the dentist's office, to submit to an injection, to accept the need for an extraction or filling, etc.

Four elements common to all fears:
1. Fear of the unknown
2. Fear of physical harm or bodily injury
3. Fear of loss of control
4. Fear of helplessness and dependency

Important: Understanding the above elements of fear allows effective planning for treatment of fearful and anxious patients.

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Ashley, a 32-year-old woman, comes in for her routine cleaning appointment. Stephanie, the hygienist, finds that Ashley has **not** been following the home care program that was recommended six months ago. Stephanie believes that Ashley's problem is a management deficiency and not a skills deficiency. Which of the following is the **best** course of action for Stephanie?

- Accept that Ashley might never change her habits
- Provide Ashley with a pamphlet on periodontal disease
- Meet with her supervising dentist to determine the future course of action
- Go over brushing and flossing techniques

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- **Meet with her supervising dentist to determine the future course of action**

Since this is **not a skills deficiency** problem, reviewing home care techniques is **not** going to solve the problem. Ashley knows **what** to do. Now Stephanie and Ashley's supervising dentist just need to find a way to motivate Ashley to find the **time** to brush and floss.

Notes:

1. It has been shown that the **most** effective way to teach oral hygiene skills is by having the patient **participate** in repeated, supervised training sessions.
2. The best time to determine a patient's **plaque index** *(in order to assess the effectiveness of a patient's home care)* is at the **beginning** of an appointment.
3. Rather than just asking a patient about his or her home care skills, have the patient **show** you.
4. Maintaining a 4-year-old child's healthy dentition starts with **educating the parent.**
5. Having your teeth cleaned and examined on a regular basis and keeping them clean on a daily basis at home is the **best way** to prevent periodontal disease.
6. The following patient information is necessary in order to plan dental hygiene care: health history, dental history, dietary analysis, and periodontal examination.

Behavior shaping is also called:

- Aversive conditioning
- Successive approximation
- Restraining
- Hypnodontics

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• **Successive approximation**

Behavior shaping is an operant conditioning technique used in behavior therapy in which new behavior is produced by **providing reinforcement** for progressively closer approximations of the final desired behavior. It is a common nonpharmacologic technique.

Proponents of this theory hold that most behavior is learned and that learning is the establishment of a connection between a stimulus and a response. For this reason, it is sometimes **called stimulus-response** *(S-R)* **theory.**

When **shaping behavior,** the dental assistant or dentist is teaching a child how to behave. Young children are led through these procedures step by step.

Behavior shaping is regarded as a **learning model.** A general rule about learning models is that the most efficient learning models are those that follow the learning theory model most closely.

Example: Attempting to change several aspects of a patient's oral hygiene regimen should be done one aspect at a time *(sequentially)*. Have the patient mimic the correct oral hygiene behavior. This will increase the chances of succeeding in changing this patient's behavior.

Aversion conditioning → a technique in which punishment, unpleasant or painful stimuli are used in the suppression of undesirable behavior. In dentistry, it is known as the **Hand-Over-Mouth** technique or **HOME.**

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Which of the following is a **conceptual framework** that describes a person's health behavior **as an expression of his / her health beliefs?**

- The Health Maintenance Model
- The Fluid Mosaic Model
- The Health Belief Model
- The Leading Circle Model

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• **The Health Belief Model**

The Health Belief Model *(HBM)* is a psychological model that attempts to explain and predict health behaviors. This is done by focusing on the attitudes and beliefs of individuals. The HBM was first developed in the 1950s by social psychologists Hochbaum, Rosenstock and Kegels working in the U.S. Public Health Services. The model was developed in response to the failure of a free tuberculosis *(TB)* health screening program. Since then, the HBM has been adapted to explore a variety of long- and short-term health behaviors, including sexual risk behaviors and the transmission of HIV / AIDS.

Components of the HBM

These **concepts** were proposed as accounting for people's **"readiness to act."**

Concept	Definition
Perceived Susceptibility	One's opinion of chances of getting a condition
Perceived Severity	One's opinion of how serious a condition and its consequences are
Perceived Benefits	One's belief in the efficacy of the advised action to reduce risk or seriousness of impact
Perceived Barriers	One's opinion of the tangible and psychological costs of the advised action
Cues to Action	Strategies to activate "readiness"
Self-Efficacy	Confidence in one's ability to take action

The Health Belief Model **suggests** that individuals will act to prevent disease **only when they believe that they are susceptible** to it. Also true is that a patient's compliance is affected by their perception of the severity of a disease as well as the length of a treatment regimen.

Behavior modification is also known as:

- Behavior evaluation
- Behavior therapy
- Behavior shaping
- Behavior training

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- **Behavior therapy**

Behavior modification *(behavior therapy)* is a **kind of psychotherapy** that attempts to modify observable, maladjusted patterns of behavior by the substitution of a new response or set of responses to a given stimulus.

Psychologists have developed many **techniques** for modifying patients' behaviors by using the principles of learning theory. **Examples of techniques:**

- **Classical conditioning** → a form of learning in which a previously neutral stimulus comes to elicit a given response through **associative training.**
- **Operant conditioning** → a form of learning in which the person undergoing therapy is **rewarded** for the correct response and **punished** for the incorrect response.
- **Aversion conditioning** → a technique in which punishment, unpleasant or painful stimuli are used in the suppression of undesirable behavior. In dentistry, it is known as the **Hand-Over-Mouth** technique or **HOME.**
- **Modeling** *(behavior shaping)* → a technique in which the person learns a desired response by **observing it being performed.**
- **Systemic desensitization** → a technique used for eliminating maladaptive anxiety **associated with phobias.** The procedure involves the construction by the person of a hierarchy of anxiety producing stimuli and the general presentation of these stimuli until they no longer elicit the initial response of fear.

Note: These methods are used mostly in **pediatric dentistry.**

Behavioral development is defined as any observable response which is mediated through the neuromotor system. What are the **four major fields** of behavior?

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• **Four major fields of behavior**

1. **Personal social** → this is usually a function of environment, work, play and society.

2. **Motor** → starting point to access maturity.

3. **Language** → vocalization, words, sentences, facial and manual movements.

4. **Adaptive** → use of motor capacity and solutions to practical behavior.

Note: To **understand** the development of **human behavior** you need to **understand** the basic concepts of **maturation** and **learning.**

All of the following are **specific ways** in communicating effectively with patients **except:**

- Describe
- Be specific
- Be evasive
- Be responsive
- Pay attention

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- **Be evasive**

Some Specific Do's and Dont's In Communicating Effectively with Patients	
Do	**Don't**
Describe	Evaluate
Be specific	Be general
Be responsive	Be evasive
Time appropriately	Be premature or too deep
Pat attention	Be inattentive or wander

Remember: The best way to show a patient that you care about what he / she is telling you is to use **eye contact.**

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All of the following are considered to be **aversive conditions** of interaction between the dentist and patient **except:**

- Psychophysiological reactions
- Stress, anxiety and fear
- Preventive oral health behavior
- Pain

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- **Preventive oral health behavior**

Aversive conditions:
- Psychophysiological reactions
- Stress, anxiety and fear
- Pain

Note: These conditions are perceived as **aversive** and the dentist-patient interaction seeks to **minimize** them.

Nonaversive conditions:
- Communication-gathering information, identifying problems, giving information *(as in case presentations)*
- Preventive oral health behavior
- Management of exceptional patients such as the physically or emotionally disabled
  → for these patients, gradually expose them to the dental office

Note: These conditions are perceived as **nonaversive** and the dentist-patient interactions seeks to **maximize** them.

Which of the following are the **most commonly used dental materials** that are deemed **hazardous by OSHA?**

- Mercury
- Nitrous oxide
- Chemicals used for film developing
- All of the above

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• **All of the above**

Mercury is extremely common in dentistry and is used most often in amalgam capsules. It is also available in bulk form and found in scrap amalgam. Associated hazards include nausea, loss of appetite, diarrhea, fine tremors, depression, fatigue, increased irritability, headache, insomnia, allergic manifestations, contact dermatitis, pneumonitis, nephritis, dark pigmentation of the marginal gingiva, and loosening of the teeth. When working with mercury:

1. Work in well-ventilated spaces and avoid direct skin contact.
2. Store mercury in unbreakable, tightly sealed containers away from all heat sources.
3. Salvage amalgam scrap and store in tightly sealed containers covered with sulfide solution.
4. Clean up spilled mercury using appropriate procedures.

Photographic and radiographic chemicals are used in developing and fixing radiographic film. If used carelessly, they can cause contact dermatitis and irritation of the eyes, nose, throat, and respiratory system from vapors and fine particles. Proper manipulation of these chemicals includes the following:

1. Use protective eyewear; wear heavy-duty rubber gloves to avoid skin contact.
2. Minimize exposure to dry powder during the mixing of solutions.
3. Work in well-ventilated areas.
4. Clean up spilled chemicals immediately.
5. Regularly launder clothing that comes in contact with photographic solutions.
6. Store photographic solutions and chemicals in tightly covered containers.

Nitrous oxide is used in conscious sedation. High exposure may cause adverse effects, especially neuropathies and spontaneous abortions. When using nitrous oxide / oxygen, use the minimal concentration necessary to achieve the desired level of sedation. Use a scavenging system and always maintain adequate ventilation. Periodically check nitrous oxide machines, lines, hoses, and masks for leakage.
Note: The **acceptable maximum exposure** level allowed by OSHA for **nitrous oxide** is 1000 ppm.

Which of the following prompted **OSHA** to adopt the **Bloodborne Pathogens Standard for dentistry?**

- Tuberculosis
- AIDS
- Hepatitis B
- Infectious mononucleosis

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• **AIDS**

OSHA also is concerned about **Hepatitis B** and other bloodborne diseases, but AIDS is the disease that prompted regulatory action. In 1986, unions representing health care workers petitioned OSHA for an emergency rule to protect their members from work place exposure to the human immunodeficiency virus *(HIV)* and the hepatitis B virus *(HBV)*. OSHA denied the petition but agreed to adopt a permanent rule on exposure to bloodborne pathogens through the regular rule making process. It took five years to develop the rule. It applies to hospitals, physicians' offices, nursing homes, other health care settings, emergency response personnel and funeral homes, **as well as dental offices.**

Notes:

1. **Bloodborne Pathogens** means pathogenic microorganisms that are present in human blood and can cause disease in humans. These pathogens include, but are not limited to, hepatitis B virus *(HBV)* and human immunodeficiency virus *(HIV)*.
2. Other **potential infectious materials** *(OPIMs)* means (1) The following human body fluids: semen, vaginal secretions, cerebrospinal fluid, synovial fluid, pleural fluid, pericardial fluid, peritoneal fluid, amniotic fluid, saliva in dental procedures (*only in dental procedures is saliva considered a potentially infectious material)*, any body fluid that is visibly contaminated with blood, and all body fluids in situations where it is difficult or impossible to differentiate between body fluids; (2) Any unfixed tissue or organ *(other than intact skin)* from a human *(living or dead)*; and (3) HIV-containing cell or tissue cultures, organ cultures, and HIV- or HBV-containing culture medium or other solutions; and blood, organs, or other tissues from experimental animals infected with HIV or HBV.

In 1996 the definition and recommendations for **Universal Precautions** was revised and given the new name of:

- Mandatory Precautions
- Regular Precautions
- Standard Precautions
- Optional Precautions

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• **Standard Precautions**

Today, Standard Precautions is the primary strategy to be used to reduce the risk of transmission of pathogens from moist body substances and applies to all patients regardless of their diagnosis or presumed infection status. Healthcare workers must avoid all contact with moist body substances by the use of Personal Protective Equipment *(PPE)*, work practices and engineering controls.

A thorough medical history **should be obtained** for all patients at the first visit and updated and reviewed at subsequent visits. **However,** since not all patients with infectious diseases can be identified by medical history, physical examination or readily available laboratory tests, the **CDC** has introduced the concept of standard *(formerly "Universal")* precautions.

The term Standard *(formerly Universal)* precautions means that all procedures are performed as though the patients were known to be infectious.

Dentists **must provide** at-risk employees with protection from:

- Tuberculosis
- Hepatitis A
- Hepatitis B
- Infectious mononucleosis

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• **Hepatitis B**

The federal standard for occupational exposure to bloodborne pathogens requires employers to provide the vaccination for hepatitis B. You must offer to provide vaccination *(and boosters should these be recommended in the future)* to all employees who have occupational exposure, at no cost to the employees. The employee may refuse to be vaccinated, but OSHA will require proof that an employee has refused.

You must offer vaccination to a new employee within 10 working days of initial assignment to a position involving exposure. OSHA requires the employee to be trained regarding hepatitis B and the vaccination prior to being offered vaccination.
Remember: OSHA considers part-time, temporary and probationary workers as employees.

Any FDA-approved hepatitis vaccine is acceptable. Currently, these are Recombivax HB and Engerix-B.

Note: It is important to stress to your employees that hepatitis B has been a long-standing occupational threat in dentistry. HBV is a hardy virus and it only takes a low concentration to transmit the virus and infect someone.

Which of the following sharps does **OSHA regulate?**

- Non-contaminated sharps
- Contaminated sharps
- All sharps
- OSHA does not regulate sharps

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• **Contaminated sharps**
The rule defines **"contaminated sharps"** as any contaminated object that can penetrate the skin, including, **but not limited to,** needles, scalpels, broken glass, broken capillary tubes and exposed ends of dental wires. There may be other objects used in your office that are sharps, and if they become contaminated **with blood or other potentially infectious materials,** including saliva, then they are regulated.

Use and care of sharp instruments and needles → sharp items *(needles, scalpel blades, and other sharp instruments)*:

1. Should be considered as potentially infective and must be handled with extraordinary care to prevent unintentional injuries.
2. Disposable syringes and needles, scalpel blades, and other sharp items must be placed into puncture resistant containers located as close as practical to the area in which they were used. To prevent needle stick injuries, disposable needles **should not** be recapped; purposefully bent or broken; removed from disposable syringes; or otherwise manipulated by hand after use.
3. Recapping of a needle increases the risk of unintentional needlestick injury. There is no evidence to suggest that reusable aspirating-type syringes used in dentistry should be handled differently from other syringes. Needles of these devices should not be recapped, bent, or broken before disposal.
4. Because certain dental procedures on an individual patient may require multiple injections of anesthetic or other medications from a single syringe, it would be more prudent to place the unsheathed needle into a "sterile field" between injections rather than to recap the needle between injections. A new *(sterile)* syringe and a fresh solution should be used for each patient.

Information about recapping: The **rule generally prohibits** bending, recapping, breaking, shearing, or removing sharps. **However,** recapping with a one-handed method *("scoop technique")* or using a mechanical device is permitted. Such techniques ensure that needles are never pointed at or moved toward the practicing health care worker or other workers, either on purpose or accidentally. Newer, self-sheathing anesthetic syringes and needle devices do not require any movements associated with recapping.

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH OSHA

Employers must ensure that _____with occupational exposure participate in a _____, at _____, during _____, with material appropriate to the education, literacy and language of the employee.

- All employees, training program, minimal cost, non-working hours
- Most employees, training seminar, minimal cost, non-working hours
- Most employees, training program, no cost, working hours
- All employees, training program, no cost, working hours

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- **All employees, training program, no cost, working hours**

Training is the **key** to **OSHA compliance.** The training must include:

- **A copy of the standard** and an explanation of its contents
- **A general explanation** of the epidemiology, symptoms and modes of transmission of **bloodborne diseases**
- An **explanation of the office's exposure control plan** and how the employee can obtain a copy of it
- Information about the **office's protocol for gloves, gowns, masks and eyewear** *(or faceshields)*, including the type of equipment available, where it is located, when it is to be used and how it is to be removed, handled, decontaminated and disposed of
- An **explanation of how to recognize tasks** that may involve occupational exposure and how to prevent or minimize such exposure *(e.g., how to use a sharps containers properly)*
- **Information on the hepatitis B vaccine,** including efficacy, safety, how administered, benefits of being vaccinated, and that it will be offered free of charge
- **Information on how to handle emergencies** involving occupational exposure *(what actions to take and whom to contact)*
- An explanation of the office protocol for handling exposure incidents, such as injuries from contaminated sharps *(how to report the incident, follow-up medical care, and evaluation)*
- **An explanation of the biohazard labels** used in the office
- An **opportunity for interactive questions and answers with the trainer**

Wearing gloves, protective eyewear and a face mask is **recommended:**

- Only when treating patients that are known to have AIDS
- Only when treating patients that have ANUG
- When treating all patients
- Only when you want to

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• **When treating all patients**

Masks and protective eyewear **are required** when splashes, spray, splatter or droplets of blood or other potentially infectious materials, including saliva, may be generated and eye, nose or mouth contamination can be reasonably anticipated. **Note:** A surgical mask must be worn under a faceshield **unless** the face shield has full peripheral protection at the sides and under the chin. The mask protects the dental health care worker from splashes and spatters to the nose and mouth.

The rule states that **if eyewear is required,** it must be goggles or glasses with solid, **not perforated**, side shields.

The rule **specifically requires** the employer to ensure that employees use the protective clothing and equipment.

Remember:

1. The Centers for Disease Control **suggest** a new mask for each patient.
2. Masks should have at least **95-99% filtering efficiency** for small particle aerosols *(1 to 3 μm)*.
3. An effective face mask will prevent passage of organisms, have minimal leakage, and filter particles. The shape, material, and degree of absorption of the mask will influence its efficiency.

What employee records **must** be maintained other than the infection control training records?

- Insurance records
- Attendance records
- Medical records if employee is involved in an occupational exposure
- All of the above

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• **Medical records if employee is involved in an occupational exposure**

The employer **must maintain a medical record** for each employee whose job involves occupational exposure to blood and other potentially infectious materials. The **record must include:**

- The employee's name and Social Security number
- A copy of the employee's hepatitis B vaccination status *(dates and medical records regarding the employee's ability to receive the vaccination)*
- Medical opinions and evaluations
- Test results
- Details about exposure incidents *(routes of exposure, how they occurred)*

Note: The medical records must be maintained for the duration of the employment plus 30 years. Also, the record must be kept strictly confidential.

Also:

- Each employee is entitled to review his / her own medical record.
- The OSHA-required records must be transferred to the new owner. If you simply go out of business and there is no new owner, you must notify the director of the National Institute for Occupational Safety and Health *(NIOSH)* at least three months before you intend to dispose of the records and offer to transmit the records to NIOSH.

All of the following are considered to be **"regulated waste"** by OSHA **except:**

- **Blood** or other **potentially infectious materials**, including **saliva** in dental procedures
- Items that **would release blood** and other **potentially infectious materials**, including **saliva**, if compressed
- Items that are **caked with dried blood** or other **potentially infectious materials** and are capable of releasing these materials during handling
- **Non-contaminated sharps**
- **Pathological** and **microbiological waste containing blood** or other potentially infectious materials, including **saliva**

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• **Non-contaminated sharps**

Key point: Regulated waste includes blood and items contaminated with blood or other potentially infectious materials *(OPIM)*.

It is the **employer's responsibility** to determine the existence of regulated waste. This determination should not based on actual volume of blood, but rather on the **potential** to release blood, *(e.g., when compacted in the waste container)*. If an OSHA inspector determines that sufficient evidence of regulated waste exists, either through observation, *(e.g., a pool of liquid in the bottom of a container, dried blood flaking off during handling)*, or based on employee interviews, citations may be issued.

OSHA has provided some additional guidance for the determination of regulated waste. OSHA stated that bandages which are not saturated to the point of releasing blood or OPIM if compressed would **not be** considered as regulated waste. Similarly, discarded feminine hygiene products **do not** normally meet the criteria for regulated waste as defined by the standard. Beyond these guidelines, it is the employer's responsibility to determine the existence of regulated waste.

Regulated waste **must be placed in containers** that are closable, constructed to contain all contents and prevent leakage, and **labeled appropriately.** The **container must be closed** prior to removal to prevent spillage or protrusion of contents. The rule also requires that if the outside of the container becomes contaminated, it must be placed in a second container that has the same characteristics. **The need for a second container is extremely unlikely in a dental office.**

According to OSHA, an occupational exposure incident is:

- All skin contact with blood or other potentially infectious fluids during the course of one's duties while at work
- Any reasonably anticipated skin, mucosal, eye or parenteral contact with blood or other potentially infectious fluids during the course of one's duties while at work
- Any reasonably anticipated skin, mucosal, eye or parenteral contact with blood or other potentially infectious fluids during the course of one's duties while at work or at home preparing for work
- All eye or mucosal contact with blood or other potentially infectious fluids during the course of one's duties while at work but only when patients are in the office

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- **Any reasonably anticipated skin, mucosal, eye or parenteral contact with blood or other potentially infectious fluids during the course of one's duties while at work**

OSHA includes **saliva** in dental procedures in the definition of **"other potentially infectious materials"** because saliva may be mixed with blood in some dental procedures. OSHA concluded, therefore, that saliva should be treated as potentially infectious even though scientists believe that bloodborne diseases are not transmitted via saliva.

The following should be **included** in the procedures for evaluating an exposure incident:

- **State the policies** that were in place at the office at the time of the incident.
- **State the engineering controls** *(i.e., needle recapping device, sharps container, rubber dam)* **and work practices** that were in place at the office at the time of the incident.
- **State the personal protective equipment** *(gloves, lab coats, etc.)* that were in use at the office at the time of the incident.

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What does **OSHA** stand for?

- Occasional Safety and Habits Administration
- Occupational Safety and Health Administration
- Occupational Services and Hygiene Administration
- Optional Standards and Health Administration

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• **Occupational Safety and Health Administration**

OSHA is a federal agency, created by Congress in 1971, to protect workers from hazards in the work place.

The Occupational Safety and Health Administration aims to ensure worker safety and health in the United States by working with employers and employees to create better working environments. Since its inception in 1971, OSHA has helped to cut workplace fatalities by more than 60 percent and occupational injury and illness rates by 40 percent. At the same time, U.S. employment has doubled from 58 million workers at 3.5 million worksites to more than 115 million workers at 7.2 million sites.

Remember:

1. **OSHA** is concerned with regulated waste **within** the dental office.
2. The EPA *(Environmental Protection Agency)* regulates the transportation of waste **from the** dental office.

The **Center for Disease Control and Prevention** recommends a minimum of _____of flushing of the water lines between patients, and several minutes of flushing of the water lines if the system has been idle for a period of time, such as over the weekend.

- 5 to 10 seconds
- 10 to 15 seconds
- 20 to 30 seconds
- 40 to 50 seconds

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- **20 to 30 seconds**

Remember → antiretraction valves:
- Are used on handpiece and air-water syringe hoses to prevent the retraction of fluid back into the tubing
- Prevent patient material *(fluids)* from getting into the water lines
- Reduce the risk of cross-contamination to another patient

Remember:
1. Handpieces should be **autoclaved between** patients.
2. Disposable saliva ejectors **cannot be reused.**
3. Reusable air-water syringe tips **must be autoclaved** *(disposable air-water syringe tips are available).*

Note: The **FDA** *(Food and Drug Administration)* is responsible for regulating handpieces and making recommendations for sterilization procedures for them.

Dental health care workers are at **greatest risk** for contracting:

- Hepatitis
- Tuberculosis
- AIDS
- Herpes

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• **Hepatitis**

Remember: As a dentist *(employer)* you must offer to provide vaccination to all employees who have occupational exposure. Any FDA-approved hepatitis vaccine is acceptable. Currently, these are **Recombivax HB** and **Engerix-B. Note:** The first-generation vaccines were plasma-derived, but the vaccines in current use are genetically engineered. HBV **antibody titer tests** are recommended 1–2 months after completion of the vaccine series to verify that the health care worker is protected. **Anti-HBs titers** decline in 30–50% of adults within 8–10 years after vaccination. However, it is believed that the immune memory remains intact for at least 20 years after immunization.

The Hepatitis B Virus is usually transmitted by one of the following methods:
1. Percutaneous inoculation
2. Sexual intercourse
3. Prenatal transfer

The center for disease control has **identified** the following groups as being high risk:
• **IV** drug users
• **Homosexual / bisexual** males
• **Persons receiving transfusions** or blood products
• **Health care workers** who may come into contact with body fluids

Note: Treat each patient and instrument as potentially infectious → standard *(formerly "universal")* infection control precautions.

Waste that is **capable of causing a poisonous effect** is called:

- Hazardous waste
- Toxic waste
- Infectious waste

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• **Toxic waste**

Hazardous waste is waste that **causes harm or injury** to the environment.

Infectious waste is waste that contains strong enough pathogens in sufficient quantity to **cause disease**.

Notes:

1. **All** infectious waste is contaminated, however, **not all** contaminated waste is infectious *(may not be able to cause disease)*.
2. **Not all** hazardous waste is toxic *(may not be poisonous)*.

Standard *(formerly "Universal")* infection control precautions for bloodborne pathogens requires **changing gloves:**

- Only if they have contacted blood
- Between every other patient
- Only when they become worn out
- Between all patients

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• **Between all patients**

Gloves and gowns **are required** when you reasonably anticipate skin contact with blood or other potentially infectious materials, including saliva. This means that if you reasonably anticipate the forearms will be spattered with saliva or blood, then forearms must be covered.

Any gown or clinic jacket that **prevents** blood or other potentially infectious materials, including saliva from reaching work clothes, street clothes or skin is considered adequate. Fluid-resistant gowns **are not required** unless it is anticipated that large amounts of blood, saliva or other body fluids will soak through the gown to the employee's clothing. OSHA appears to consider cotton or cotton / polyester clinic jackets or lab coats as satisfactory barriers for most routine dental procedures. When surgical procedures are performed involving large quantities of blood *(e.g., trauma surgery)*, additional personal protective equipment, such as long-sleeved gowns, are required. **According to OSHA, the selection is to be based on the quantity and type of exposure expected.**

Note: When handling chemical agents, contaminated sharps or cleaning a dental office, you should wear protective eye wear, a mask, and **heavy-duty utility** or nitrile gloves. These gloves should be worn for safe pick-up, transport, cleaning, and packing of contaminated instruments. They should not be worn when handling or contacting clean surfaces or items. **Important:** Exam gloves **are not appropriate** for instrument cleaning or any housekeeping procedures in the dental office.

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Documents that contain information concerning a **hazardous chemical** are called?

- ADBC's
- MSDS's
- HGPF's
- TRDP's

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- **MSDS's**

***MSDS stands for **Material Safety Data Sheets.**

Chemical manufacturers and importers are **required** to obtain a material safety data sheet for each hazardous chemical they produce or import. Distributors are responsible for ensuring that their customers are provided with a copy of these MSDS's. Employers must have an MSDS for each hazardous chemical which they use. Employers may rely on the information received from their suppliers.

Key points on MSDS:

- The role of MSDS's under the rule is to **provide detailed information** on each hazardous chemical, including its potential hazardous effects, its physical and chemical characteristics and recommendations for appropriate protective measures.
- MSDS's must be readily **accessible to employees** when they are in their work areas during their work shifts. This may be accomplished in many different ways. You must decide what is appropriate for your particular work place. Some employers keep the MSDS's in a binder in a central location. As long as employees can get the information when they need it, any approach may be used.
- The employees must have access to the MSDS's themselves.

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH OSHA

The **OSHA bloodborne pathogens standard** is:

- A **comprehensive rule** that sets forth the specific requirements OSHA believes will prevent the transmission of bloodborne diseases to **patients**
- A **comprehensive rule** that sets forth the specific requirements OSHA believes will prevent the transmission of bloodborne diseases to **employers**
- A **comprehensive rule** that sets forth the specific requirements OSHA believes will prevent the transmission of bloodborne diseases to **employees**
- All of the above

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- **A comprehensive rule that sets forth the specific requirements OSHA believes will prevent the transmission of bloodborne diseases to employees**

It imposes a number of requirements:

- Employers covered by the standard **must make exposure** determinations and develop an exposure control plan.
- They **must also use** engineering and work practice controls to prevent employee exposure and develop a system to evaluate exposure incidents.
- It **requires training** all employees who provide or assist in providing patient care, as well as those who clean operatories, instruments, and gowns.

Remember → through this standard, OSHA directs that:

1. **Uniform clothing worn in the dental office** should be laundered at the dental office or an outside service, **not** at an employee's home.
2. **Offices are** using barrier techniques, communicating hazards to employees *(training)*, performing proper cleaning of office and **offering hepatitis B vaccinations.**

OSHA's Bloodborne Pathogens Standard requires testing of the source patient's blood for HIV and HBV, and **disclosure of the results** to the exposed employee:

- Always
- Never
- Only where it is permitted and not in conflict with applicable laws or regulation
- Only if the exposed employee gets sick following the exposure

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- **Only where it is permitted and not in conflict with applicable laws or regulation**

The standard further requires that the **exposed employee** be informed of any laws or regulations concerning disclosure of the identity and infection status of the source patient. The standard **does not,** therefore, require dental employers to violate any applicable privacy laws.

An **exposure incident** is defined as a specific occupational incident involving eye, mouth, other mucous membrane, non-intact skin or parenteral contact with blood or other potentially infectious materials, including saliva. The most common example is an injury from a **contaminated sharp.**

Following a report of an **exposure incident,** the employer must make immediately available, at no cost to the employee, a confidential medical evaluation and follow-up that includes:

- **Documentation of route(s) of exposure** and circumstances in which the incident occurred
- **Identification and documentation of the source individual** *(i.e., the patient),* unless the employer can establish that identification is infeasible or prohibited by state or local law
- **Results of testing of the source individual's blood,** if they are available
- **Collection and testing of the employee's blood <u>after</u>** consent is obtained
- Medically indicated **prophylaxis**
- **Counseling**
- **Evaluation of reported illnesses** in the weeks **following** the incident

The employer **must provide** the following information to the health care professional who performs the evaluation:

- **A copy of the OSHA standard**
- **A description of the employee's duties** as they relate to the incident
- **Documentation of route(s) of exposure and circumstances** under which exposure occurred
- **Results of the source individual's blood testing** *(if available)*
- **All medical records relevant to treatment of the employee,** including vaccination status, which are the employer's responsibility to maintain

OSHA's Bloodborne Pathogens Standard requires that a written exposure control plan be reviewed:

- Biannually
- Monthly
- Every 3–5 years
- Annually

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• **Annually**

The plan must be reviewed and updated after any changes in knowledge, practice, personnel, guidelines, or regulations that may affect occupational exposure. The plan should document consideration and implementation of appropriate safer devices that are designed to eliminate or minimize occupational exposure. This documentation must include evidence that employees who use the devices have had input into the identification, evaluation, and selection of the devices.

Exposure control plan *(ECP)* → the standard requires that every employer have a **written exposure control plan** designed to **eliminate or minimize employee exposure to bloodborne diseases**. Basically, the plan must set forth your office policies and protocols to protect employees from these diseases.

OSHA requires that the plan contain the following elements:
How and on what schedule your office is implementing OSHA's requirements for: barrier techniques, hepatitis B vaccination, housekeeping, disinfection of contaminated work surfaces and equipment, handling regulated waste, post-exposure evaluation and follow-up, communication of bloodborne pathogen hazards to employees, and record keeping.

For example, the plan should outline your office protocol for **when gloves, gowns, masks and eyewear** *(or faceshields)* are to be used; **when and how** you will provide training for employees; **your specific** practices for disinfection; **how you launder gowns** *(on-site or using a service)*; **where you keep records,** such as training and medical records and the OSHA standard; **your office policy** on the hepatitis B vaccination; and **your office protocol** for handling regulated waste.

It must be accessible to employees, and it must be updated at least annually and whenever necessary to reflect office changes *(e.g., new procedures that affect occupational exposure, new positions)*. The plan must also be provided to OSHA upon request.

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Which agency is a branch of the **United States Department of Health and Human Services?**

- The Food and Drug Administration *(FDA)*
- The Drug Enforcement Administration *(DEA)*

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• **The Food and Drug Administration** *(FDA)*

The **FDA** determines which drugs and medical devices can be marketed in the United States; requires **proof of both safety and efficacy.**

The **Drug Enforcement Administration** *(DEA)* is a branch of the Department of Justice; this agency determines degree of control for substances with abuse potential *(Schedules I through V)*.

A test result which **erroneously assigns an individual** to a specific diagnostic or reference group, due particularly to insufficiently exact methods of testing is known as:

- A false negative test
- A true negative test
- A false positive test
- A true positive test

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- **A false positive test**

A test can produce two kinds of errors: a **false positive** result *(meaning that the test indicates presence of the disease when it is, in fact, not there)* or a **false negative** result *(meaning that the test indicates absence of the disease when it is in fact present)*.

In general, a population of tested individuals may be divided into four groups:

- **True Positives:** those who test positive for a condition and are positive *(i.e., have the condition)*
- **False Positives:** those who test positive, but are negative *(i.e., do not have the condition)*
- **True Negatives:** those who test negative and are negative
- **False Negatives:** those who test negative, but are positive

Test _____ is a medical term defined as the ratio of true negative tests to the total unaffected patients tested expressed as a percentage. It measures a screening test's ability to correctly identify the absence of disease.

- Specificity
- Sensitivity

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• **Specificity**

A test with **high** specificity has **few false positives.** Test specificity is **independent** of disease prevalence in the community.

Test **sensitivity** is a medical term defined as the ratio of true positive tests to the total number of affected *(positive)* patients tested expressed as a percentage. It measures a screening test's ability to correctly identify the **presence** of a disease. A test with **high** sensitivity has **few false negatives.** Test sensitivity is **independent** of disease prevalence in the community.

Note: Sensitivity and specificity are **inversely** proportional. As the specificity of a test increases, the sensitivity decreases.

$$\frac{\text{True positives}}{\text{True positives + False negatives}} = \text{probability of being positive given you tested positive}$$

$$\frac{\text{True negatives}}{\text{True negatives + False negatives}} = \text{probability of being negative given you tested negative}$$

$$\frac{\text{True negatives}}{\text{True negatives + False positives}} = \frac{\text{True negatives}}{\text{All unaffected}} = \text{Specificity}$$

$$\frac{\text{True positives}}{\text{True positives + False negatives}} = \frac{\text{True negatives}}{\text{All affected}} = \text{Sensitivity}$$

The measure of the **quality of care** provided in a particular setting is called:

- Quality assurance
- Quality evaluation
- Quality assessment
- Quality inspection

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• **Quality assessment**

Quality assurance is the measurement of the quality of care and the **implementation** of any necessary changes to either maintain or improve the quality of care rendered.

The **differences** in these definitions are important: **quality assessment** is limited to the appraisal of whether or not standards of quality have been met, whereas **quality assurance** includes the additional dimension of action to take the necessary corrective steps to improve the situation in the future.

The following concepts relate to quality assurance:

1. **Structure** → refers to the layout and equipment of a facility.
2. **Process** → involves the actual services that the dentist and assistant perform for the patients and how well they perform.
3. **Outcome** → is the change in health status that occurs as a result of the care delivered.

Which of the following indexes should be used to measure the presence and severity of periodontal disease?

- PMA index
- Plaque index
- Periodontal index
- Gingival index

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• **Periodontal index**

In this index, the condition of **both** the gingiva and the bone is estimated individually for each tooth in the mouth. A progressive scale that gives relatively little weight to gingival inflammation and relatively more weight to **alveolar bone resorption** is used for scoring. The scores from each tooth are added together, and the total is divided by the number of teeth present in the mouth. The result gives the periodontal disease index of the patient, which reflects the average status of periodontal disease in a given mouth without reference to the type or causes of the disease. The community's score is the average of individual scores of persons examined.

Remember: Gingivitis is most commonly scored with the **gingival index** *(GI)* of Löe and Silness. This index grades the gingiva on the four surfaces of each tooth. It is based on **inflammation** and **bleeding.**

Notes:

1. The **Gingival Index** *(GI)* and **Papillary, Marginal, and Attached Gingival Index** *(PMA index)* are confined to measurements **within the gingiva.**
2. The **Plaque Index** *(PI)* is used to determine **accumulation of plaque.**
3. The **Sulcus Bleeding Index** *(SBI)* is used to determine the **bleeding and gingival health.**
4. The **OHI and OHI-S** are debris indices
5. **DMFS and DMFT** are caries indices
6. The best time to perform an index *(perio. or plaque)* is at the beginning of the appointment.

A **system** where a **provider of coverage** contracts to pay for some of the patient's dental treatment is known as?

- First-party dentistry
- Second-party dentistry
- Third-party dentistry
- Fourth-party dentistry

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- **Third-party dentistry**

The major forms of third-party reimbursement currently in use are:

- Usual, customary, and reasonable *(UCR)* fee
- Table of allowance
- Fee schedules
- Capitation

Panel of providers:

- **Closed panel** → dental services provided by salaried dentists at specified locations only.
- **Open panel** → dental services provided by any dentist willing to accept third party payment.

Note: Dentistry is financed mainly through fee-for-service self-pay. 56% of all dental expenses are paid out of pocket by the patient. Third-party payers represented by private insurance pay about 33% of total dental expenses, followed by government-financed or public programs *(i.e., Medicaid, Veterans Affairs)*.

If the major purpose of an epidemiologist's research is to **determine caries susceptibility** as opposed to immediate treatment needs, the best caries index to use is:

- TSIF
- DMFS
- DMFT
- CPITN

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• **DMFT**

Measurements of dental caries are made with the DMF index. The **DMF** is an **irreversible index** and is applied only to **permanent teeth.** The DMF index can be applied to teeth **(DMFT)** or surfaces **(DMFS).** The DMFT score may range from 0 to 32, whereas the DMFS score may range form 0 to 160.

Note: The **primary dentition** uses the **def index,** where **d** represents decayed teeth; **e,** extracted teeth; and **f,** filled teeth.

The **results** of the DMFT index **yield** a group's **caries susceptibility.** It has received practically universal acceptance and is probably the best known of all dental indices.

Limitations of the DMFT index:

1. DMF values **are not related** to the number of teeth at risk.
2. DMF index can be **invalid in older adults** because teeth can become lost for reasons other than caries.
3. DMF index can be **misleading in children** whose teeth have been extracted for orthodontic reasons.
4. DMF **cannot** be used for **root caries.**
5. DMF **cannot** account for **sealed teeth.**

The **major objective** of public health programs is:

- Prevention
- Cost efficiency
- Teamwork
- All of the above

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• **Prevention**

The **fundamental principles** of public health are prevention, cost efficiency and teamwork. Prevention is the **major objective** of public health programs because it entails **ethics, teamwork and cost efficiency.**

- It is more ethical to prevent disease than to cure it
- Teamwork is necessary to handle large groups efficiently
- Cost efficiency plays a major role because prevention is cheaper than a cure

Education plays an important role in public health because it decreases the need for government intervention. In other words, when people learn why regulations are of value they will comply. **For example**, when people learn how many lives are saved yearly by wearing seatbelts, then they are more inclined to wear them.

Any school-based program for the promotion of oral health should have the following fundamental components:

1. **Oral health services,** meaning preventive procedures, health screening and treatment, referral and follow-up.
2. **Health instruction** to include both personal and community health topics.
3. **A healthy environment,** with attention to all aspects of the school environment that could affect the health of students or school personnel.

Note: The more successful school-based programs use a high degree of **active involvement of the participants.**

Dentists are:

- **Morally** obligated to report child abuse
- **Ethically** obligated to report child abuse
- **Legally** obligated to report child abuse
- All of the above

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• **All of the above**

Dentists are **morally, ethically and legally obligated** to report a suspected case of child abuse.

Once an injury of a suspicious nature is **observed,** the dentist's first and immediate responsibility is the protection of the child.

Reports should be made to the **designated state agency** *(social services or police).* Dentists must familiarize themselves with the exact procedures to be followed in their own states.

Notes:

1. **Child abuse most commonly** involves newborns and children up to the age of three years.
2. Dentists **are also** ethically obligated to identify and refer cases of **domestic violence.** Practitioners should become familiar with the physical signs of domestic violence, especially because 68% of battered women's injuries involve the face, 45% the eyes,and 12% the neck.

Which of the following refers to the number of **new cases** of a specific disease occurring within a population over a certain amount of time?

- Incidence
- Prevalence
- Epidemiology
- Frequency

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• **Incidence**

***Incidence** is **expressed as a rate** *(cases) / (population) / (time)*

Prevalence is the **proportion of persons** within a population suffering from a particular disease at a given point in time. It is expressed as a **percentage of the population.**

Remember: Incidence is a rate and requires a unit of time, whereas **prevalence is a proportion** and is expressed as a percentage of the population.

Epidemiology is the study of the incidence, prevalence, mode of transmission and control of diseases in a defined human population for the purpose of establishing programs to prevent and control their development and spread.

Note: Frequency is simply a count.

Random assignment and blinding are methods used in clinical trails to _____ and enhance study validity.

- Increase bias
- Decrease bias
- Have no change on bias

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• **Decrease bias**

Important: Both require justification, however, because when randomized and blinded, subjects have no say in their choice of experimental treatment nor do they have information about what experimental treatment they are receiving.

A **"randomized study"** is a study in which all subjects have an equal chance of being assigned to either the study or control group.

Statistical probability is such that the assumption can then be made that the groups differ from each other only in terms of the agent under study. Any uncontrolled variables influencing the outcome are likely to affect subjects in both groups equally. For this reason, researchers prefer the **random assignment method** for placing subjects into either the study or control group.

When subjects are **unaware** of whether they are in a test or control group, this is said to be a **blind study.** When neither participants **nor** examiners know the group allocations *(test or control groups)* it is called a **double-blind study. Note:** One means of achieving a **blinded study** is with the use of placebos.

Two variables used in research studies:

1. **Dependent variable** is the variable whose value depends on those of others; e.g., in the formula $x = 3y + z$, **x is the dependent variable.**
2. **Independent variable** is the variable whose value determines that of others; e.g., in the same formulas as above, **y and z are the independent variables.**

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH PH

The **most important concept** of C.E.A. Winslow's definition of public health is:

- To encourage mental and physical efficiency
- Promotion through organized community effort
- Individuals acting alone can solve any problem
- The science and art of preventing disease

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• **Promotion through organized community effort**

Public health is defined as "The science and art of preventing disease, prolonging life and promoting physical health and efficiency through organized community efforts."

Three principles of public health:

1. A problem exists
2. Solutions to the problem exist
3. The solutions to the problem are applied

A public health problem must meet the following criteria:

- A condition or situation that is a widespread actual or potential cause of morbidity and / or mortality
- Involves a perception on the part of the public, public health authorities, and the government that a public health problem is occurring

Dental public health is defined as "The science and art of preventing and controlling dental disease and promoting dental health through organized community efforts." It is that form of dental practice which serve the community as a patient rather than the individual. It is concerned with the dental health education of the public, and research and application of the findings of research with the administration of programs of dental care for groups, and the prevention and control of dental disease through a community approach.

The term **mortality,** used in epidemiology, refers to:

- Disease
- Reasons as to why disease and death occur in a population
- Death
- Birth

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• **Death**

Vital statistics are **quantitative methods** routinely collected by epidemiologists and public health professionals. They are used to monitor and evaluate the life history of a specific population. Vital statistics include:

Mortality *(also called death rate)* reflects the number of deaths caused by a **specific disease.** It is the ratio of the number of deaths caused by the disease to the total number of cases of the disease at a specific time.

Morbidity is the incidence of a specific disease within a given population.

Natality is the birth rate *(ratio of births to the general population).*

Birth-death ratio *(also called the vital index)* is the number of births in a given year divided by the number of deaths in a given year. It is an indication of the population growth, stability or reduction.

Crude death rate is the ratio of the number of deaths occurring within a given time period and population to the total population during that time.

Note: These statistics are **used** to identify community health needs, to estimate health care costs and to evaluate health program effectiveness.

A method of payment for dental services in which an individual or institutional provider is paid a fixed, per capita amount without regard to the actual number or nature of services provided to each patient is called a:

- Capitation fee
- Fixed fee
- Contractual fee
- Managed fee

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• **Capitation fee**

Managed care is an **arrangement** whereby a **third-party payer** *(e.g., insurance company, federal government or corporation)* **mediates between doctors and patients,** negotiating fees for services and oversees the types of treatment given.

Examples of managed-care practices:

- **HMO** *(health maintenance organization)*: is usually a self-contained staff-model practice in which no distinction is made between the providers of insurance and the providers of health care.
- PPO *(preferred provider organization)*
- IPA *(independent practice organization)*

Represent groups of doctors who practice in the community and are distinct from the insurance provider. However, an insurance agency contracts with the providers for discounted rates and may refer patients to these providers exclusively.

Note: PPOs and IPA's typically involve contracts between insurers and a number of dentists. Patients are allowed to choose from whom they will receive dental treatment from depending upon whether or not the dentists participates in the PPO arrangement. **Participants of HMOs** are much more limited in the selection of dentists from whom they can receive treatment.

Payment to the dentist for these managed-care programs is usually made on a **capitation basis.** A capitation fee is usually a fixed monthly payment paid by a carrier to a dentist based on the number of patients assigned to the dentist for treatment. This fee is the same regardless of how much or how little care is delivered.

The role of the professional in the practice of dentistry is described in the ADA's Principles of Ethics and Code of Professional Conduct *(ADA Code)*. The ADA Code is, in effect, a written expression of the obligations arising from the implied contract between:

- The dental profession and insurance companies
- The dental profession and public health board of directors
- The dental profession and society
- The dental profession and the local government

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• **The dental profession and society**

The dental profession holds a special position of trust within society. As a consequence, society affords the profession certain privileges that are not available to members of the public-at-large. In return, the profession makes a commitment to society that its members will adhere to high ethical standards of conduct. These standards are embodied in the ADA Principles of Ethics and Code of Professional Conduct *(ADA Code)*. The ADA Code is, in effect, a written expression of the obligations arising from the implied contract between the dental profession and society. Members of the ADA voluntarily agree to abide by the ADA Code as a condition of membership in the Association. They recognize that continued public trust in the dental profession is based on the commitment of individual dentists to high ethical standards of conduct.

The ADA Code has **three main** components:
• The Principles of Ethics
• The Code of Professional Conduct
• The Advisory Opinions

Three **ethical principles** in the code:
1. **Justice** → the quality of being impartial and fair.
2. **Autonomy** → to inform patients about treatment, to be truthful and also protect their confidentiality.
3. **Beneficence** → to be kind and to give the highest quality of care that one is capable of.

Remember: The dentist is responsible for providing information and dental care, **however**, ultimately the patient is responsible for maintaining his / her own oral health *(brushing, flossing, etc.)*.

Note: The **Good Samaritan law,** enacted in all states, provides immunity from suit for specified health practitioners who render emergency aid to victims of accidents, **provided** there is no evidence of gross negligence. **Important: Not all states** include dentists in the Good Samaritan law.

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH PH

All of the following are **true** concerning the Americans with Disabilities Act **except:**

- Dentists **cannot** deny anyone care due to a disability
- Dental offices **must undergo** structural changes to allow access for the disabled
- Dentists **cannot** dismiss employees due to a disability
- Patients with HIV **are not protected** under the Americans with Disabilities Act

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• **Patients with HIV are not protected under the Americans with Disabilities Act**

***This is **false;** patients with HIV **are protected** under the Americans with Disabilities Act.

Both state and federal statutes define disability as having the following:

1. A physical or mental impairment that **substantially limits** one or more of the major life activities of such individual.
2. A **record** of such impairment.
3. **Being regarded** as having such impairment.

According to the **plaque index** of Silness - Löe, the tooth #14 would have what score *(plaque index)*?

- 1.0
- 1.5
- 2.0
- 2.5

Tooth #14

Surface	Scores
Buccal	2
Lingual	1
Mesial	1
Distal	2

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• 1.5

*****Plaque index for this tooth = 2+1+1+2 / 4 = 1.5**

The measurement of the state of oral hygiene by Silness - Löe plaque index is based on recording the presence or absence of plaque on teeth. Each of the four surfaces of the teeth *(buccal, lingual, mesial and distal)* is given a score from 0 - 3. The scores from the four areas of the tooth are added and divided by four in order to give the plaque index for the tooth with the following scores and criteria:

Scores	Criteria
0	No plaque
1	A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be seen insitu only after application of disclosing solution or by using the probe on the tooth surface.
2	Moderate accumulation of soft deposits within the gingival pocket, or the tooth and gingival margin which can be seen with the naked eye.
3	Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

Notes:

1. In this system, the most stress is placed on the thickness of the plaque at the gingival margin area on all four surfaces of each tooth.
2. The plaque index has been extensively used **but** does not have universal acceptability.
3. **According to some studies, 80-90%** of children have inflammatory periodontal disease *(gingivitis or periodontitis)* by the age of 15. **Localized acute gingivitis** is the most common form. Epidemiologic studies show the strongest relationship between prevalence and severity of periodontal disease with oral hygiene and age.

The **gingival index** is a (an):

- Reversible index
- Irreversible index

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- **Reversible index**

Important: An **irreversible index** is one that measures conditions which cannot be reversed *(dental caries)*. A **reversible index** measures conditions that can be changed *(plaque and bleeding)*.

A **dental index** is a data collection instrument used to numerically express the oral health status of a population.

Common Indices:

- **Periodontal index** → a **reversible index** used to measure the periodontal condition of a population
- **Decayed-missing-filled teeth index** *(DMFT)* → an **irreversible index** used to determine total dental caries experience, past and present. It is used only on permanent teeth. It has received practically universal acceptance and is probably the best known of all dental indexes. The **primary dentition** uses the **DEFT index** *(decayed, extracted, filled teeth)*. **Note:** The **DMFS index** is the same as the DMFT index except it records tooth surfaces involved instead of teeth.
- **Gingival index** → a **reversible index** used to assess the severity of gingivitis based on color, consistency, and bleeding on probing.
- **Simplified oral hygiene index** → a reversible index used to measure oral hygiene status *(by estimating the tooth surface covered with material alba and / or calculus)*.
- **Plaque index** → a **reversible index** used to assess the thickness of plaque at the gingival margin.

All of the following are **advantages of using alcohols** (*70% isopropyl and 70% ethyl alcohol*) as surface disinfectants **except:**

- They are **bactericidal**
- They are **sporicidal**
- They are **tuberculocidal**
- They are **economical**

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• **They are sporicidal**

***This is **false;** they **are not** sporicidal.

Alcohol is probably the **most widely used antiseptic** and is used to reduce the number of microorganisms on the skin surface in the area of a wound. **Alcohol denatures proteins,** extracts membrane lipids and acts as a dehydrating agent, all of which contribute to its effectiveness as an antiseptic. Even some viruses *(lipophilic viruses only)* are inactivated by alcohol. The drawbacks of alcohol are that it evaporates too quickly and has diminished activity against viruses in dried blood, saliva and other secretions on surfaces *(this is due to the presence of tissue proteins and glycoproteins which render alcohol ineffective)*.

For the above reason, **alcohols are not regarded** as effective surface cleansing agents *(i.e., cleaning a dental operatory following patient treatment)*.

Notes:

1. **Isopropyl alcohol** (90% - 95%) is the major form in use in hospitals.
2. **Ethanol** (70%) is widely used to clean the skin prior to immunization or venupuncture.
3. **Iodine** is the most effective skin antiseptic used in medical practice. It acts as an oxidizing agent and combines irreversibly with proteins.

The **minimal** temperature required for sterilization by an **autoclave** is:

- 350° F *(177° C)*
- 250° F *(121° C)*
- 450° F *(232° C)*
- 89° F *(31° C)*

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• **250° F *(121° C)***

The proper time and temperature for **autoclaving** is 250° F *(121° C)* for 15–20 minutes. ***These conditions will yield **15 lbs. pressure of steam** per square inch.

Moist heat destroys bacteria by denaturation of the high protein-containing bacteria. The autoclave provides sterilization when used at 250° F *(121° C)* for 15–20 minutes **because** it applies the **heat under pressure,** which greatly speeds up the denaturation process when compared with boiling water. Usually only ten minutes is required to destroy all of the bacteria, **but** the increased time is allowed for penetration when the instruments are wrapped in thick towels.

The effectiveness of autoclaving is **best determined by culturing bacterial spores.** Spore testing of autoclave units is recommended **weekly** *(at a minimum)*.

Note: Precleaning is the **most important step** in instrument sterilization. Debris acts as a barrier to the sterilant and sterilization process. Ultrasonic instrument cleaning is the safest and most efficacious method of precleaning.

For **dry heat sterilization** at a temperature of 320° F *(160° C)*, how long does it take to sterilize instruments?

- 5 minutes
- 20 minutes
- 40 minutes
- 2 hours

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• **2 hours**

The proper time and temperature for **dry heat sterilization** is **320° F** *(160° C)* **for 2 hours. Note:** 340° F *(170° C)* for 1 hour is also effective.

Items which are usually sterilized by dry heat can be autoclaved. They should be removed **immediately** after the cycle to diminish the possibility of corrosion of the instruments and dulling of sharp points or edges *(carbon steel instruments)*.

Important: Dry heat destroys microorganisms by causing **coagulation of proteins.**

Advantages of dry heat:
• **Effective** and safe for sterilization of metal instruments
• **Does not** dull or corrode instruments

Disadvantages of dry heat:
• **Long** cycle
• **Poor** penetration
• **Will ruin** heat-sensitive materials

Note: Instruments **must be dry** before both dry heat sterilization and ethylene oxide sterilization. Water will **interfere** with the sterilization process.

For **unsaturated chemical vapor** sterilization at a temperature of 250° F *(121° C)*, how long does it take to sterilize instruments?

- 12 minutes
- 15–20 minutes
- 20–40 minutes
- 2 hours

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• **20–40 minutes**

The proper time and temperature for unsaturated chemical vaporization is **270° F** *(132° C)* **for 20–40 minutes.** *** These conditions will yield 20 lbs. pressure of sterilizing vapor.

The principal operation of this system is similar to that of steam sterilizers **but** instead of distilled water, a **solution of alcohol, formaldehyde, ketone, acetone and water** is used to produce the sterilizing vapor.

The temperature and pressure required for chemical vapor sterilizers **are greater** than those for the autoclave.

The **major advantage** of this system is that it **does not** rust or corrode metal instruments, including carbon steel. **Disadvantages include** instruments must be dried completely before processing, a special chemical solution must be used and it **will destroy** heat-sensitive plastics.

Note: Rapid heat transfer sterilization requires the following, 375° F *(191° C)* with a cycle time of 12 minutes for wrapped instruments and 6 minutes for unwrapped instruments.

How long does it take to **kill bacterial spores** when a dental instrument is placed in a 2% solution of glutaraldehyde?

- 10 minutes
- 1 hour
- 10 hours
- 24 hours

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• **10 hours**

2% glutaraldehyde is an **alkalizing agent** highly lethal to essentially all microorganisms if sufficient contact time *(10 hours)* is provided and there is absence of extraneous organic material.

Advantages of glutaraldehydes:
- Most potent category of chemical germicide
- Capable of killing spores *(after 10 hours)*
- EPA registered as chemical sterilant
- Can be used on heat sensitive materials

Disadvantages of glutaraldehydes:
- Long period required for sterilization
- Allergenic
- Not an environmental disinfectant
- Extremely toxic to tissues

Notes:

1. In hospitals, glutaraldehydes are used to sterilize respiratory therapy equipment.
2. Alcohols, chlorhexidine, and quaternary ammonium compounds are **disinfectants.**

Quaternary ammonium compounds, which are widely used for skin antiseptics, are classified as:

- Nonionic detergents
- Anionic detergents
- Cationic detergents

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• **Cationic detergents**

Detergents are "surface-active" agents composed of a long-chain, lipid-soluble, hydrophobic portion and a polar hydrophilic group, which can be a cation, an anion, or a nonionic group. These surfactants interact with the lipid in the cell membrane through their hydrophobic chain and with the surrounding water through their polar group and thus disrupt the membrane.

Quaternary ammonium compounds *(e.g., benzalkonium chloride)* are cationic detergents. They are used as **disinfectants** and **antiseptics.** Gram-positive bacteria are the most susceptible to destruction. These compounds **are not** sporicidal, tuberculocidal, or viricidal and are inactivated by anionic detergents *(soaps and the iron in hard water).*

Anionic surface-acting substances include synthetic anionic detergents and **soaps.** These substances alter the nature of interfaces to lower surface tension and increase cleaning. Their primary value appears to be their ability to **remove microorganisms mechanically** from the skin surface.

Note: Nonionic chemicals do not possess any antimicrobial properties.

The **killing or removal** of all microorganisms, including bacterial spores, is called:

- Disinfection
- Sterilization
- Cleaning
- Decontamination

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• **Sterilization** → refers to the absence of all living forms

Disinfection is the killing of many, **but not all,** microorganisms. It does not include the **destruction of spores.** The term disinfectant is reserved for chemicals applied to **inanimate surfaces** *(lab tops, counter tops, headrests, light handles, etc.)*. They **are not** considered safe for use on living tissue.

Antiseptics are chemical agents similar to disinfectants, but they **may be applied** safely to living tissue. **Note:** Soap **only removes** microorganisms.

Note: Remember the doctrine of sterilization → Do not disinfect what you can sterilize!!!

Remember:

1. The immersion of dental instruments in cold disinfectants will not destroy spores or the hepatitis viruses *(they are resistant to physical and chemical agents).*
2. **Liquids** are generally **sterilized by filtration**. The most commonly used filter is composed of nitrocellulose and has a pore size of 0.22um. This size will retain all bacteria and spores. Filters work by physically trapping particles larger than the pore size.

Which type of pathogens provide the **ultimate test** for efficacy of sterilization?

- Bacteria
- Spore-forming
- Virus
- Fungi

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• **Spore-forming**

Because bacterial spores are resistant to boiling (*100° C at sea level)*, they must be exposed to a higher temperature; this cannot be achieved unless the pressure is increased. For this purpose, an autoclave chamber is used in which steam at a pressure of 15 lb./in. reaches a temperature of 121° C and is held for 15–20 minutes. **This kills even the highly heat-resistant spores** of Clostridium botulinum, the cause of botulism, with a margin of safety.

Saturated steam *(autoclave)* has proven to be the most practical, the most economical, and the most currently effective sporicide. It is also the most efficient method for destruction of viral and fungal microorganisms.

The **steam autoclaves** are made to operate in the following ranges:
- 121° C *(250° F)* at a pressure of 15 pounds per square inch *(psi)* for 15–20 minutes.
- 134° C *(270° F)* at a pressure of 30 psi for a minimum of 3 minutes *("flash cycle")*.

To positively **destroy all living organisms,** the minimum, required temperature is 121° C *(250° F)*.

Note: The **autoclaving time** will vary directly with the type of the load placed into the chamber. The 3-minute "flash cycle" is best indicated for unwrapped instruments. When instruments are wrapped, a longer sterilization cycle is required to permit adequate penetration of steam for proper disinfection.

Which of the following is a **powerful oxidizing agent** that inactivates bacteria and most viruses by oxidizing free sulfhydryl groups?

- Alcohol
- Chlorine
- Formaldehyde
- Phenol

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• **Chlorine**

Chlorine is the active component of **hypochlorite** *(bleach)*, which is used as a disinfectant. Generic sodium hypochlorite solutions are generally recommended by the CDC as an alternative to other proprietary germicides for disinfection of environmental surfaces. A dilution of 1:100 with water *(approximately 500 ppm chloride)* is acceptable after proper precleaning of visible material from surfaces. It is best to renew the dilution at least weekly. Bleach solutions should be used with caution as they are corrosive to metals.

Disinfectants are antimicrobial agents that kill *(germicide)* or prevent the growth *(microbiostatic)* of pathogenic microorganisms. **Disinfectants are not considered safe for use on living tissue** *(as opposed to antiseptics which are)* and are applied only to inanimate objects *(counter tops, light handles, headrests, etc.)*.

Phenol was the original disinfectant used in hospitals, but is rarely used as a disinfectant today because it is too caustic.

Note: Concentration and **contact time** are critical factors that determine the effectiveness of an antimicrobial agent against a particular microorganism. Any or all of the three major portions of microbial cells can be affected: **the cell membrane, cytoplasmic contents** *(particularly enzymes)*, and **nuclear material.**

Remember for disinfectants:
1. **Water-based** is **better** than **alcohol-based.**
2. **Pump spray** is **better** than **aerosol spray.**

The only gaseous chemical agent that can be relied on for complete sterilization of dental instruments is:

- Hexachlorophene
- Phenol
- Ethylene oxide
- Isodine

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• **Ethylene oxide**

Ethylene oxide gas has been widely used as a sterilization agent, particularly for prepackaged, disposable plastic ware in hospitals. This gas is **fairly toxic** to humans and is also flammable, **so that its general use is limited.** Exposure of materials to ethylene gas must be performed in special sealed chambers. Items must be cleaned and dried thoroughly before the sterilization process. **Note:** Sterilization with ethylene oxide is a slow process *(10 - 16 hours)* depending on the material to be sterilized.

Advantages of ethylene oxide sterilization:

- Highly penetrative
- Does not damage heat-sensitive materials *(rubber, cotton, plastic, etc.)*
- Evaporates without leaving a residue
- Works well for materials that cannot be exposed to moisture

Important: Ethylene oxide functions as an alkylating agent by **irreversibly inactivating cellular nucleic acids** *(DNA)* and **proteins.**

The greatest occupational health care worker risk for **bloodborne infection** is:

- Hepatitis C virus
- Human Immunodeficiency Virus
- Hepatitis B virus
- Tuberculosis

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• **Hepatitis B virus**

The basis of the current standard *(formerly "universal")* infection control precautions was first recommended by the CDC in 1987. The purpose was to protect health care workers *(HCW)* from occupational transmission of all bloodborne infectious diseases during provision of patient care. While many health professionals focused on the Human Immunodeficiency Virus *(HIV)* as the major risk, accumulated evidence clearly demonstrated that the hepatitis B virus *(HBV)* was responsible for infection in 10–30% of exposed, susceptible HCW. Concentrations of HBV in the blood of a chronic carrier can range between 1,000,000 to 100,000,000 virions per ml, in contrast to significantly lower viral loads demonstrated for both HIV-infected persons and person with AIDS.

Viral concentrations detected in hepatitis C virus *(HCV)* infected individuals range between those noted for HBV and HIV. Thus, we target the most infectious bloodborne pathogen with our infection control standards. Precautions that minimize potential HBV spread, also by inference, prevent cross-infection of less infectious microorganisms.

Antimicrobial chemical agents which **destroy microorganisms** when applied onto inanimate surfaces such as counter tops or lights, are called:

- Antiseptics
- Sterilants
- Disinfectants
- Decontaminants

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• **Disinfectants**

Sterilization is defined as the use of physical or chemical procedures to destroy all microbial life, including bacterial endospores.

Disinfection is less lethal than sterilization, and involves a chemical process of microbial inactivation which destroys virtually all pathogenic microorganisms on inanimate surfaces *(i.e., counter tops, light handles, bracket trays)*, but not necessarily all microbial forms *(i.e., spores)*.

Antiseptics are agents that are used on living tissue. Examples include antimicrobial handwash agents and antimicrobial mouth rinses. Antiseptic compounds inhibit but do not necessarily destroy microorganisms. **Disinfectants** are used on non-living things such as floors, counter tops, etc. They are usually stronger and are too toxic to be used on living tissue. Disinfectants are lethal for pathogenic microorganisms. A common disinfectant is bleach.

Many people are confused about the difference between soap and detergent. Soaps and detergents are not the same thing, although both are **surfactants,** or surface active agents, which basically means a washing compound that mixes with grease and water. **Soaps** are made of materials found in nature. **Detergents** are synthetic *(although some of the ingredients are natural)*; they were developed during World War II when oils to make soap were scarce. **Note:** Plain soap physically removes microbes but **does not** kill them.

Note: Surfactants are commonly included in most disinfectants to ensure full wettability of all surfaces.

Which of the following is used as a **handwash agent?**

- Chlorhexidine gluconate
- Triclosan
- Isopropyl alcohol
- Both chlorhexidine gluconate and triclosan
- All of the above

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• **Both chlorhexidine gluconate and triclosan**

Chlorhexidine gluconate is a skin antiseptic and an antimicrobial agent. It is a relatively rapid-acting antiseptic that is effective against both gram-positive and gram-negative bacteria and some fungi. It combines excellent surface adherence characteristics and the best residual effect of any agent currently on the market. The antimicrobial action of chlorhexidine gluconate is attributed to the disruption of the microbial cell membrane and precipitation of cell contents.

Triclosan is a potent wide spectrum antibacterial and antifungal agent. It has been shown to be effective in reducing and controlling bacterial contamination on the hands. Triclosan appears to kill bacteria mainly by inhibiting fatty acid synthesis.

Isopropyl alcohol is also used for hand hygiene procedures, but products containing 60–80% alcohol **DO NOT** use water, hence the term **waterless hand hygiene.**

An **antibacterial solution** which directly kills bacteria is said to be:

- Bactericidal
- Bacteriostatic
- Substantive

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• **Bactericidal**

The use of bactericidal chemicals is preferable to those which are **"static"**. The latter do not directly kill or inactivate microbes, but instead inhibit their metabolism and replication. These affected organisms can remain viable but inactive for extended intervals. Application of **"cidal"** agents or processes are designed to ensure microbial inactivation.

Bactericidal products:

- Sporicidin
- Envirocide
- Mada Cide-1
- Biozide
- Cetylcide II
- SaniZide Plus

The marker microorganism for **intermediate surface** disinfection is:

- Bacillus stearothermophilus
- Pseudomonas aeruginosa
- Hepatitis B virus
- Mycobacterium tuberculosis

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• **Mycobacterium tuberculosis**

Antimicrobial activity against **Mycobacterium tuberculosis** is recognized as a significant benchmark criterion for disinfectant effectiveness. While tuberculosis is not transmitted via inanimate environmental surfaces, the morphology and structure of the tubercle bacilli make them relatively resistant to penetration by a number of low-level disinfectant chemicals.

High-level disinfection is a process in which chemical sterilants are used in a manner that kills vegetative bacteria, tubercle bacillus *(mycobacteria)*, lipid and nonlipid viruses, and fungi, but not all bacterial spores, if they are present in high numbers. **High-level disinfectants include:** glutaraldehyde, sodium hypochlorite 1,000 ppm *(1:50 dilution of household bleach)*, and boiling. **Note:** The application of high-level disinfection in dentistry is limited because virtually all dental instruments are heat-stable and can be sterilized in an autoclave.

Intermediate-level disinfection kills vegetative bacteria and fungi, tubercle bacillus, and lipid and nonlipid viruses. **Intermediate-level** agents, such as phenols, iodophors, sodium hypochlorite 500 ppm *(1:100 dilution of household bleach)*, and certain preparations containing alcohols *(60 - 90% isopropyl)* are able to penetrate the wax and lipid outer layers surrounding mycobacteria. Designed for cleaning of environmental surfaces.

Low-level disinfection kills only vegetative bacteria, some fungi, and lipid viruses, but not the tubercle bacillus. These products *(quaternary ammonium compounds and accelerated hydrogen peroxide products)* are designed for daily cleaning and disinfection of all surfaces in the office.

The antigens most responsible for an immediate **Type I** reaction to natural rubber latex are:

- Proteins
- Accelerators
- Corn starch powders
- Anti-oxidants

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- **Proteins**

Only a few of the more than 250 proteins found in the sap from the rubber tree **Hevea brasiliensis** are responsible for causing the Type I, immediate, IgE-mediated reactions to natural rubber latex. These are water-soluble macromolecules that can leach out of latex gloves when a person perspires, or be detected on the surfaces of other products containing natural rubber latex *(NRL)*.

Three types of reactions can occur in persons using latex products:

- **Irritant contact dermatitis:** it is the most common reaction to latex products. Signs include the development of dry, itchy, irritated areas on the skin, usually the hands.
- **Allergic contact dermatitis** *(delayed hypersensitivity)* results from exposure to chemicals added to latex during harvesting, processing, or manufacturing. These chemicals can cause skin reactions similar to those caused by poison ivy. As with poison ivy, the rash usually begins 24 to 48 hours after contact and may progress to oozing skin blisters or spread away from the area of skin touched by the latex.
- **Latex allergy** *(immediate hypersensitivity)* can be a more serious reaction to latex than irritant contact dermatitis or allergic contact dermatitis. Reactions usually begin within minutes of exposure to latex, but they can occur hours later and can produce various symptoms. Mild reactions to latex involve skin redness, hives, or itching. More severe reactions may involve respiratory symptoms such as runny nose, sneezing, itchy eyes, scratchy throat, and asthma *(difficult breathing, coughing spells, and wheezing)*. Rarely, shock may occur; but a life-threatening reaction is seldom the first sign of latex allergy. Such reactions are similar to those seen in some allergic persons after a bee sting.

Cleaning surfaces **prior** to disinfection in clinical settings is required to:

- Destroy all pathogens
- Inhibit pathogen growth
- Reduce the concentration of pathogens
- Weaken the virulence of pathogens

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• **Reduce the concentration of pathogens**

The simplest way to approach environmental surface disinfection is to adhere to a basic premise of aseptic technique → **clean it first.** All disinfectant products include specific label instructions for cleaning prior to disinfection. **Cleaning** is defined as the physical removal of debris.

Two effects result from efficient cleaning:

1. A reduction in the number of microorganisms present.
2. The removal of blood, tissue bioburden, and other debris which can interfere with disinfection.

The most **efficient** way to kill microbes is:

- Cold sterilization
- Proper handwashing with sterilizing antiseptics
- Heat sterilization
- Immersion of contaminated items in chemical sterilants

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• **Heat sterilization**

The use of heat has long been recognized as the **most efficient, reliable, biologically monitorable method of sterilization.** During a routine cycle using an autoclave, unsaturated chemical vapor sterilizer, or dry heat unit, cell death is accomplished via heat inactivation of critical enzymes and other proteins within microbial cells.

The recommendation stating that all reusable items that come in contact with a patient's blood, saliva, or mucous membranes **must be sterilized** using heat is now routinely accepted and used in dental facilities.

The most common form of an **adverse epithelial reaction** noted for health-care professionals is:

- Irritation dermatitis
- Type I immediate latex allergy
- Type IV, delayed latex allergy
- Superficial fungal infections on the fingers

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- **Irritation dermatitis**

A number of published reports have cited data suggesting that between **20–30%** of health care workers suffer from occasional or chronic dermatitis on their hands. The most common manifestation of the condition is irritation dermatitis. This reaction is caused by skin irritation from using gloves and possibly by exposure to other workplace products and chemicals. It can also result from repeated hand washing and drying, incomplete hand drying, use of cleaners and sanitizers, and exposure to powders added to the gloves.

Signs include the development of dry, itchy, irritated areas on the skin, usually the hands. Health care workers located in colder climates may also experience chapping during the winter months.

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH IC

The term **"antiseptic"** BEST relates to a (an):

- Autoclave
- Dry heat sterilizer
- Chemical used on contaminated counter tops
- Handwash agent

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• **Handwash agent**

The term **antiseptic** is used for antimicrobial agents that are applied onto **living tissues.** Liquid antimicrobial preparations for handwashing, such as chlorhexidine gluconate, parachlorametaxylenol, idophors, and triclosan are examples.

Chemical solutions, sprays, or wipes applied onto **inanimate** counters or other environmental surfaces are termed **disinfectants.**

Note: The primary reason for using chemical disinfectants and antiseptics is to control the number of pathogens and exhibit some killing action. These agents **are not** to be used for sterilization.

One of your patients develops a **Type I,** immediate allergic reaction to latex. When treating them and wearing gloves from now on, you can:

- Wear vinyl or nitrile gloves
- Wear hypoallergenic latex gloves
- Get an exemption and not wear gloves
- Refuse to treat them

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• **Wear vinyl or nitrile gloves**

A wide variety of latex-alternative infection control items have appeared in the marketplace within the last 10 years. The most widely recognized are newer generations of vinyl or nitrile gloves which do not cross-react with latex allergens. Products with the designation "hypoallergenic" are no longer to be labeled latex alternatives, as they contain latex with a chemical coating over the latex.

Note: Studies over recent years showed that not all latex-allergic persons were able to use hypoallergenic gloves, since many Type I allergic individuals still developed allergic manifestations when using these gloves.

Remember: Reactions usually begin within minutes of exposure to latex, but they can occur hours later and can produce various symptoms. Mild reactions to latex involve skin redness, hives, or itching. More severe reactions may involve respiratory symptoms such as runny nose, sneezing, itchy eyes, scratchy throat, and asthma *(difficult breathing, coughing spells, and wheezing)*. Rarely, shock may occur; but a life-threatening reaction is seldom the first sign of latex allergy. Such reactions are similar to those seen in some allergic persons after a bee sting.

The **complete destruction** of all forms of microbial life, including spores, **BEST** describes:

- Disinfection
- Sterilization
- Pasteurization
- Sanitization

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• **Sterilization**

Sterilization → the destruction or removal of **all forms of life,** with particular reference to microbial organisms; the limiting requirement is destruction of heat resistant spores.

Disinfection → the use of chemical agents to accomplish the destruction of pathogenic microorganisms, but not necessarily all pathogen or resistant spores, on **inanimate** surfaces.

Pasteurization → the treatment of dairy foods, such as milk, for short intervals with heat, to kill certain, disease-causing microorganisms; the target of pasteurization is the destruction of Mycobacterium tuberculosis.

Sanitization → the treatment of water supplies to reduce microbial levels to safe public health levels.

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH IC

Which of the following statements is **true?**

- Human Immunodeficiency Virus *(HIV)* is the most infectious target of Standard *(Universal)* Blood Precautions
- Hepatitis B Virus *(HBV)* is the most infectious target of Standard *(Universal)* Blood Precautions
- Hepatitis C Virus *(HCV)* is the most infectious target of Standard *(Universal)* Blood Precautions
- Epstein-Barr Virus *(EBV)* is the most infectious target of Standard *(Universal)* Blood Precautions

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- **Human Immunodeficiency Virus *(HIV)* is the most infectious target of Standard *(Universal)* Blood Precautions**

HIV is the most infectious bloodborne pathogen known, and infection control precautions aimed at preventing this viral transmission, have also been shown to be effective in preventing HIV and HBV cross-infection.

A particular kind of **antimicrobial treatment,** such as that for our drinking water which lowers the total microbial load to safe public health levels is called:

- Antisepsis
- Sanitization
- Disinfection
- Sterilization

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• **Sanitization**

Antiseptic → a chemical that can be administered safely to external body surfaces or mucous membranes to decrease microbial numbers. Antiseptics **cannot** be taken internally.

Disinfectant → a chemical agent used to destroy microorganisms on inanimate objects such as dishes, tables, and floors. Disinfectants **are not** safe for living tissues.

Disinfection → the process of **reducing** the numbers of or **inhibiting** the growth of microorganisms, especially pathogens, to the point where they no longer pose a threat of disease.

Sterilization is the process by which all microbial life is killed. Bacterial spores are the most resistant of microbial life to killing. If the sterilization process is effective in killing bacterial spores, all other pathogenic and nonpathogenic miroorganisms are presumed killed. The spore test, or biologic monitor, is the only true test of sterility.

The **routine use** of biologic monitors results in early detection of sterilization problems so that corrective measures can be taken.

OSHA / IC / BEHAV. SCI. / PUBLIC HEALTH IC

Which phrase describing an aspect of infection control is **not** appropriate?

- Exposure is not synonymous with infection
- Do not disinfect when you can sterilize
- Sterilization of all clinical instruments and inanimate environmental surfaces is mandatory
- Known AIDS patients can be treated using Standard Bloodborne Precautions

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- **Sterilization of all clinical instruments and inanimate environmental surfaces is mandatory**

It is **not possible, nor necessary** to sterilize all environmental surfaces which become contaminated during patient care. In many instances, because of the relatively low risk of microbial transmission, thorough cleaning of the surfaces is sufficient to break the cycles of cross-contamination and cross-infection.

Ideal chemical disinfectants should:

- Kill as many microbes as possible in the shortest time possible
- Not damage the object being decontaminated or humans or animals
- Not be affected by the presence of organic material
- Be compatible with soaps, detergents, and other chemicals
- Be inexpensive and stable during storage

All of the following statements concerning **hepatitis C** are true **except:**

- 80% of persons have no signs or symptoms
- It is transmitted primarily in infected blood
- There is a vaccine to prevent hepatitis C
- Signs and symptoms include: jaundice, fatigue, dark urine, abdominal pain, loss of appetite, and nausea

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- **There is a vaccine to prevent hepatitis C**

***This is **false;** there is no vaccine to prevent hepatitis C

Hepatitis C virus *(HCV)* is transmitted primarily in infected blood. Historically, parenteral drug abusers, persons receiving transfusions, organ recipients, and hemophiliacs receiving factor VIII or IX were shown to be at high risk of HCV infection. More recently, persons receiving tattoos or undergoing body piercings have been infected via contaminated, unsterilized needles.

An infection caused by normally non-pathogenic microorganisms in a host whose resistance has been **decreased** or compromised is known as a (an):

- Nosocomial infection
- Secondary infection
- Opportunistic infection
- Medical infection

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• **Opportunistic infection**

The percentage of people living with a wide variety of **immune compromised conditions** continues to increase. Along with the clinical manifestations of those types of diseases there can be accompanying deficiencies in aspects of host immune defenses. The severity of deficiency can range from mild to life threatening, and predispose the compromised person to infections by organisms which would not usually occur in other people with intact innate and specific immunity.

Immune-compromised persons are those that have a weakened, under-developed or malfunctioning immune system. This definition includes: HIV infection, cancer, an organ transplant, Primary Immune Deficiency disorders, some severe autoimmune disorders, persons on immunosuppressive medications and other illnesses that can weaken the immune system.

It is recommended that **face masks** be changed:

- Between patients
- Daily
- Twice per day

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• **Between patients**

Face masks should be changed **at least with every patient and more often** if heavy spatter is generated during treatment. If a mask is worn longer than 20 minutes in an aerosol environment, the outside surface of the mask becomes a nidus of pathogenic bacteria rather than a barrier.

In a **latex allergy,** the immune system identifies latex as a harmful substance. The immune system triggers certain cells to produce _____antibodies to fight the latex component *(the allergen)*.

- IgA
- IgD
- IgE
- IgG

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• **IgE**

Latex allergy is a reaction to certain **proteins** found in natural rubber latex, a product manufactured from a milky fluid derived from the rubber tree *(Hevea brasiliensis)* found in Africa and Southeast Asia.
If a person has a latex allergy, their body mistakes latex for a harmful substance. Their immune system triggers certain cells *(B-cells differentiate into plasma cells)* to produce immunoglobulin E *(IgE)* antibodies to fight the latex component *(the allergen)*. The next time the person comes in contact with latex, the IgE antibodies sense it and signal their immune system to release histamine and other chemicals into their bloodstream.

These chemicals cause a range of allergic signs and symptoms. Histamine is partly responsible for most allergic responses, including runny nose, itchy eyes, dry throat, rashes and hives, nausea, diarrhea, labored breathing and even anaphylactic shock.

Latex sensitivity can occur in these ways:

- **Direct contact:** The most common cause of latex allergy is **direct contact** with latex, such as by wearing latex gloves or by contact with latex-containing products.
- **Inhalation:** A person can develop a latex allergy by **inhaling latex particles.** Latex products, especially gloves, shed large amounts of latex particles, which can become airborne. Cornstarch is sometimes used on the inside of gloves to make them easier to put on and take off. The cornstarch absorbs latex proteins, but when the gloves are snapped during application or removal, the latex laden particles fly into the air. The amount of airborne latex from gloves differs greatly depending on the brand of glove used.

_____ is recommended for all instruments that are used in the **mouth.**

- Disinfection
- High-level disinfection
- Sterilization involving the use of heat
- Both disinfection and sterilization involving the use of heat

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• **Sterilization involving the use of heat**

The use of heat has long been recognized as the **most efficient, reliable, biologically monitorable method of sterilization**. During a routine cycle using an autoclave, unsaturated chemical vapor sterilizer, or dry heat unit, cell death is accomplished via heat inactivation of critical enzymes and other proteins within microbial cells.

The recommendation stating that all reusable items that come in contact with a patient's blood, saliva, or mucous membranes **must be sterilized** using heat is now routinely accepted and used in dental facilities.

Dry heat sterilization requires high temperatures and long exposure times to achieve sterilization. The recommended sterilization time for dry heat is 2 hours at 320° F *(160° C)*. **Rapid heat** transfer systems are available that operate at _____temperatures for _____time cycles.

- Lower, longer
- Higher, longer
- Lower, shorter
- Higher, shorter

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• **Higher, shorter**

This method of rapid heat transfer achieves sterilization in 12 minutes at 375° F *(190° C)* for wrapped items and in 6 minutes for unwrapped items. ***Important: Note:** The manufacturer's recommendations must be followed for these systems.

FDA-approved, forced air, dry heat convection ovens are appropriate for sterilization of heat-stable instruments and other reusable items employed in patient care. They use a higher temperature than other dry heat units, and there is controlled internal air flow within the chamber. **In contrast** to the traditional type of dry heat sterilizers, a rapid heat transfer unit can achieve sterilization of items in substantially shorter times, while still offering the advantages of dry heat.

Advantages of rapid heat transfer sterilization:
• It has a shorter cycle time than regular dry heat units
• It does not dull cutting edges
• Items are dry after cycle

Disadvantages of rapid heat transfer sterilization:
• Instrument must be dried before packaging and placement in chamber
• It destroys heat-labile items
• It cannot sterilize liquids
• It is generally unsuitable for dental handpieces
• Unwrapped items become contaminated quickly after cycle

Personal protective equipment clinic jackets should be:

- Short sleeve, high neck
- Long sleeve, high neck
- Long sleeve, turtle neck
- Whatever your preference

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• **Long sleeve, high neck**

For **optimal protection,** clinic jackets or coats **are required** to be long sleeved and high necked. This requirement was installed to minimize the potential for exposed skin to contact, and therefore become contaminated with, a patient's blood, saliva, or other potentially infectious material.

Contaminated laundry is defined by the Standard as laundry that has been soiled with blood or OPIM *(saliva)* or may contain sharps. The following rules apply to the handling of contaminated laundry in the dental office:

- It must be handled as little as possible
- Contaminated laundry should be bagged at the location of use
- It will not be sorted or rinsed in the location of use
- Bags for storage or transport will be labeled with a biohazard label or color coded red
- Contaminated laundry must be handled with gloves and other appropriate PPE

The **dental office** has several choices in dealing with contaminated laundry *(which will be protective gowns or scrubs in most offices):*

- Personnel may use disposable gowns
- The office may use an outside laundry service
- A washer and dryer may be used on-site
- An unincorporated dentist may take laundry home
- One employee may be appointed to take laundry to a laundromat using appropriate PPE. This person must be trained in handling contaminated laundry, must wear the appropriate PPE, and must transport the laundry in an orange / red bag or one that has the biohazard symbol.

PEDIATRIC DENTISTRY

Legend

Major Topic	Abbreviation
Abnormal Teeth	Abn Tth
Behavior Management	Behav Mngmt
Diseases & Conditions	Dis & Cond
Drugs	Drugs
Fluoride	Fluoride
General Information	Gen Info
Miscellaneous	Misc.
Primary Dentition	Prim Dent
Pulp Treatment	Pulp Tx
Restorative	Restorative
Tooth Development	Tth Dev
Tooth Trauma	Tth Trauma

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PEDIATRIC DENTISTRY

Drugs

If a child in your office requires **antibiotic** prophylaxis to prevent bacterial endocarditis, which regimen is correct?

- Amoxicillin 100 mg / kg 1 hr prior to dental appointment
- Amoxicillin 75 mg / kg 2 hrs prior dental appointment
- Amoxicillin 50 mg / kg 1 hr prior to dental appointment
- Amoxicillin 25 mg / kg 2 hrs prior to dental appointment

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- **Amoxicillin 50 mg / kg 1 hr prior to dental appointment**

Remember: 1 lb = .453 kg

Child Prophylactic Regimens for Dental Procedures		
Situations	**Agent**	**Regimen**
Standard general prophylaxis	Amoxicillin	Children: 50 mg/kg orally 1 hr before procedure
Unable to take oral medications	Ampicillin	Children: 50 mg/kg IM or IV within 30 min. before procedure
Allergic to penicillin	Clindamycin	Children: 20 mg/kg orally 1 hr before procedure
	Cephalexin **OR** Cephadroxil	Children: 50 mg/kg orally 1 hr before procedure
	Azithromycin **OR** Clarithromycin	Chilsren: 15 mg/kg orally 1 hr before procedure
Allergic to penicillin and unable to take oral medications	Clindamycin	Children: 20 mg/kg IV within 30 min. before procedure
	OR Cefazolin	Children: 25 mg/kg IM or IV within 30 min. before procedure

Which of the following is the **most frequently** utilized route of administration for sedation in pediatric patients?

- Oral
- Inhalation
- IV
- IM

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• **Inhalation** → the agent used most frequently is **nitrous oxide**

Nitrous oxide is a slightly sweet smelling, colorless, inert gas. It must always be coupled with **no less than 20% oxygen.** Nitrous oxide is quickly absorbed from the lungs and is physically dissolved in the blood. There is no biotransformation, and the gas is rapidly excreted by the lungs when the concentration gradient is reversed. It is recommended that the patient be **maintained on 100% oxygen for 3 to 5 minutes** after the sedation period.

Nitrous oxide basically creates an **altered state of awareness** with impaired motor function. It is a central nervous system depressant. It produces little analgesia. **The combined volume of gases being delivered** (*oxygen and nitrous*) **should be at least 3 to 5 liters/minute.** The operator should encourage the patient to breathe through the nose with the mouth closed.

Notes:

1. For **restorative dentistry,** nitrous oxide is usually all you need to treat a child who is **fearful** of the dentist (*along with local anesthesia*).
2. The feeling of floating or giddiness with tingling of the digits is the **proper response** to nitrous oxide.
3. Nitrous oxide is **stored as a liquid** under pressure, and is **not flammable** but will support combustion.
4. Nitrous oxide is much **less soluble in blood** than alveolar air, thus allowing for rapid changes in alveolar gas concentration.
5. **Concentration effect** → since nitrous is used at relatively high concentrations *(approximately 35%)* in dentistry, there is a higher concentration gradient and more rapid uptake.
6. **Diffusion hypoxia** → since nitrous oxide elimination is typically rapid, alveolar oxygen might get displaced. Avoid by maintaining on 100% oxygen for 3-5 minutes after sedation. Most machines are safely constructed to deliver at least 30% oxygen.

Which of the following drugs is **widely used** for pediatric sedation?

- Meperidine
- Chloral hydrate
- Barbiturates
- None of the above

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• **Chloral hydrate**

Chloral hydrate acts on the CNS to **induce sleep.** At normal doses, the sleep induction does not affect breathing, blood pressure or reflexes. It may be used before some surgeries or procedures to help relieve anxiety and to induce sleep. When used in combination with analgesics, it can help manage pain after surgery. It has an onset of action of 15 to 30 minutes when given by mouth. **Important:** Children often enter a period of excitement and irritability before becoming sedated. As with barbiturates, pain may cause paradoxical reactions.

Chloral Hydrate *(Noctec)*		
Weight (lbs.)	**Age**	**Dosage**
25-50	2-4	500-760 mg
50-75	4-7	750-900 mg
75-100	7 & up	1000-1500 mg

The **short acting barbiturates** secobarbital (*Seconal*) and pentobarbital (*Nembutal*) are sedative drugs. They are sometimes considered for pediatric conscious sedation by oral administration. They are of very limited value. They are nonanalgesic. They may cause hyper-excitability rather than sedation in some children.

Note: Chloral hydrate and the barbiturates are classified as **sedative-hypnotics** whose principal effect is sedation or sleepiness.

Meperidine (*Demerol*) is a synthetic opiate agonist. It is used to treat moderate to severe pain (*acute dental pain*). It may be used as a preoperative medication to relieve pain and allay anxiety.

A child eleven years old traumatized a **permanent** maxillary central incisor some time ago. The tooth has never been restored. It is now painful and there is evidence of swelling. A periapical x-ray discloses a **pathosis** associated with the apex. The suggested treatment is:

- Pulpotomy
- Extraction
- Pulpectomy
- Observation

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• **Pulpectomy** → root canal therapy

This is treated the same way as you would treat the adult patient. At age eleven the root of a maxillary central incisor should be completely formed, therefore an apexification procedure is **not indicated.** If the root were not fully formed, then an **apexification** process should be started. This involves the placement of calcium hydroxide pastes into the canal to stimulate continued apical closure.

The fact that the tooth is **painful** and there is swelling is a **contraindication** to a pulpotomy. You need healthy pulp tissue in the root for success of a pulpotomy. If the tooth were non-restorable, then a pulpectomy procedure would be contraindicated and the only alternative would be to extract the tooth.

Apexogenesis is a vital pulp therapy procedure performed to encourage continued physiological development and formation of the root end. This term is frequently used to describe vital pulp therapy performed to encourage the continuation of this process. **MTA** *(Mineral Trioxide Aggregate)* is frequently used for this procedure. The best sign for success of apexogenesis is **continuous completion of apex.**

Indirect pulp treatment is a procedure performed in a tooth with:

- A necrotic pulp
- A deep carious lesion adjacent to the pulp
- A periapical radiolucency
- Pulp tissue that is irreversibly infected due to caries or trauma

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• A deep carious lesion adjacent to the pulp

The caries near the pulp is left in place to avoid pulp tissue exposure and is covered with a biocompatible material *(i.e., calcium hydroxide)*. A radiopaque base such as calcium hydroxide, zinc oxide and eugenol, or glass ionomer cement is placed over the remaining affected dentin to stimulate healing and repair. The tooth then is restored with a material that seals the tooth from microleakage.

The **objectives** of indirect pulp capping: The restorative material should seal completely the involved dentin from the oral environment. The tooth's vitality should be preserved. No post-treatment signs or symptoms such as sensitivity, pain, or swelling should be evident. There should be no radiographic evidence of pathologic external or internal root resorption or other pathologic changes. There should be no harm to the succedaneous tooth.
Indications for **indirect pulp capping** → mostly for permanent teeth:
- Rampant caries
- Large carious lesions close to the pulp which **are not** chronically painful

Contraindications for indirect pulp capping: **Do not** use indirect pulp capping in cases where there is spontaneous pain, furcation involvement, or pulpal involvement. **Note:** Most pediatric dentists feel indirect pulp caps are **contraindicated** in the primary dentition.

Important: Adolescent patients with rampant caries may require **caries control** prior to final restorations. Caries control is used to arrest all deep active carious lesions and halt their advancement toward the pulp. This involves the removal of gross caries followed by **calcium hydroxide placement and an interim restoration such as IRM** (*reinforced ZOE*).

Note: The greatest problem in **pulpal diagnosis** is predicting the amount or extent of inflammation. Successful management of deep caries and pulpally involved teeth in children is **contingent** on the extent of inflammation in the pulp.

A **four-year old child** presents with acute pain associated with a **primary** mandibular second molar that has a large carious lesion with pulpal involvement. Radiographically, there is periapical pathology on the distal root. The child is very cooperative and is able to tolerate long appointments. What is the **preferred choice** of therapy for the primary mandibular second molar?

- Incision and drainage
- Pulpotomy
- Pulpectomy
- Extraction

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• **Pulpectomy**

Saving the tooth is very important so that a space maintainer will not be necessary. Constructing a space maintainer in cases where second primary molars are lost before eruption of first permanent molars is extremely difficult.

Since there is **periapical pathology** and the child is four years old, the treatment of choice is **pulpectomy**. If there wasn't any periapical pathology, a **formocresol pulpotomy** would be indicated. If the child were older and there was a periapical radiolucency but successful pulpectomy could not be accomplished, the treatment of choice would be extraction with **placement of a space maintainer.** This should be done to prevent damage to the surrounding bone and the developing permanent tooth.

When performing a **pulpectomy on primary teeth,** the root canals are debrided, enlarged, disinfected, and filled with a resorbable material such as non-reinforced zinc oxide-eugenol. The tooth then is restored with a restoration that seals the tooth from microleakage. **Note: ZOE** will cause a tissue reaction, but it will be minimal. ZOE is used because you want it to resorb when the primary tooth begins its normal resorption process.

Note: Chronic pulpal infection in **primary molars** is usually first noted on x-rays as a **change in the bony furcation.**

Which treatment is the proper one for a **Class II fracture** of a **permanent tooth** with an **immature apex?**

- Pulpectomy
- Apply calcium hydroxide to exposed dentin and restore tooth with a permanent restoration
- Pulpotomy
- Observe

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- **Apply calcium hydroxide to exposed dentin and restore tooth with a permanent restoration**

Emergency treatment for fractures → **permanent teeth with immature apices:**

Class I → smooth enamel edges, **restore tooth.**

Class II → apply **calcium hydroxide** to exposed dentin and **restore tooth** with a permanent restoration.

Class III → **immediately after injury, apply calcium hydroxide** over exposure and place a temporary restoration. If exposure is large or the injury was several hours or days ago, **perform a calcium hydroxide pulpotomy.** Once apex closes, do pulpectomy.

Class IV → **calcium hydroxide pulpotomy.** Once apex closes, do pulpectomy.

In an older child with a fully formed apex: If there is a pinpoint exposure and it's been a while (*day*) since the fracture, the treatment of choice would be conventional root canal therapy using gutta-percha. If it is seen immediately, then a direct pulp cap with calcium hydroxide is indicated, followed by a permanent restoration.

Which **pulpotomy technique** is recommended in the treatment of **primary** teeth that have had a **carious exposure**?

- Calcium hydroxide technique
- Formocresol technique

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• **Formocresol technique**

The **formocresol technique** is recommended in the treatment of primary teeth that have had a carious exposure. Selection of teeth is the same as for the calcium hydroxide technique and is also carried out in one appointment. After the coronal pulp has been removed *(amputated),* a cotton pellet **moistened with formocresol** is placed in contact with the pulp stumps and is allowed to **remain for 5 minutes.** The pellet is then removed and the pulp chamber is dried. A thick paste of zinc oxide-eugenol is placed over the chamber and the tooth is restored. **The success of a formocresol pulpotomy for a primary tooth depends primarily on a vital root tip.**

The reason for performing a pulpotomy using either technique is that the coronal pulp shows evidence of inflammation and degenerative change due to microorganisms located there. The pulpotomy procedure **preserves** the radicular vital pulp tissue when the entire coronal pulp is amputated. This procedure **allows resorption and exfoliation** of the primary tooth but preserves its role as a natural space maintainer.

Note: Formocresol (*formalin and cresol*) will cause surface fixation of the pulpal tissue accompanied by degeneration of the odontoblasts.

All of the following are **contraindications** to performing a direct pulp cap on a primary tooth **except:**

- Spontaneous pain from the tooth
- A pinpoint exposure with little or no hemorrhaging (*bleeding*)
- A large exposure
- Excessive hemorrhaging (*bleeding*)
- Radiographic evidence of internal resorption

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- **A pinpoint exposure with little or no hemorrhaging (*bleeding*)**

Direct pulp capping is primarily used on permanent teeth. The reason it is not widely used on primary teeth is because of the alkaline pH of CaOH. CaOH can effect (*irritate*) the pulp either mildly or **most often severely.** With a **mild irritation,** there is a mild inflammatory reaction which will resolve itself and regroup as reparative dentin. With **severe irritation,** there is a **probability of internal resorption.** In primary teeth this severe irritation resulting in internal resorption happens **more often than not.** In permanent teeth this **rarely occurs,** because the severe inflammatory response will cause reparative dentin to form.

Despite this, there are **indications** for performing a direct pulp cap on **primary** teeth:
- Tooth must be **asymptomatic**
- Must be a **small exposure** with **little** or no **hemorrhaging**

Mineral trioxide aggregate *(MTA)* was developed at the University of Loma Linda to seal communications between the root canal system and the external tooth surface at all levels, and recently indicated in pulp treatment as a direct pulp capping agent. Studies have shown that mineral trioxide aggregate presents similar responses to calcium hydroxide in the pulp tissue and periapical region when used for direct pulp capping.

Which **pulpotomy technique** is recommended in the treatment of a **permanent tooth** with a **carious exposure** which also has **immature root development?**

- Calcium hydroxide technique
- Formocresol technique

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• **Calcium hydroxide technique**

The **calcium hydroxide** technique is recommended in the treatment of permanent teeth when there is a pathological change in the pulp at the exposure site. This procedure is particularly indicated for **permanent teeth with immature root development and with healthy pulp tissue in the root canals**. The success of a pulpotomy in this case would be indicated when the root apex, if not completely formed, completes its full development in time (*apexogenesis).* This technique is completed in one appointment. Only those teeth free of symptoms of **painful pulpitis** (*severe toothache, lingering pain*) are considered. A dentin bridge will form at a level somewhat below the level of amputation of the coronal pulp. **Histologically,** you would see a necrotic layer immediately under the calcium hydroxide.

Remember: The reason for performing a **pulpotomy** using either technique is that the coronal pulp shows evidence of inflammation and degenerative change due to microorganisms located there.

The phenomenon of **"strawberry tongue"** is associated with:

- Herpangina
- Scarlet fever
- Diphtheria
- Mumps

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• **Scarlet fever**

Scarlet fever is an exotoxin-mediated disease arising from group A beta-hemolytic streptococcal infection. The peak incidence of scarlet fever occurs in children 4 to 8 years old. It is usually accompanied by symptoms of strep throat, such as sudden onset of fever, sore throat, headache, nausea, vomiting, abdominal pain, muscle pain, and fatigue.

An enlargement of the fungiform papillae extending above the level of the white desquamating filiform papillae gives an appearance of an unripe strawberry. During the course of scarlet fever, the coating disappears and the enlarged red papillae extend above a smooth denuded surface, giving the appearance of a **red strawberry** or **raspberry.** Penicillin is the drug of choice, Early diagnosis and treatment are important to prevent complications, which include local abscess formation, rheumatic fever, arthritis, and glomerulonephritis.

Herpangina is a viral infection, usually of young children, characterized by mouth ulcers, but a high fever, sore throat, and headache may precede the appearance of the lesions. The lesions are generally ulcers with a white to whitish-gray base and a red border - usually on the roof of the mouth and in the throat. The ulcers may be very painful. Generally, there are only a few lesions. The disease usually runs its course in less than a week. **Treatment is palliative.** The cause is often an infection by a strain of coxsackie A virus.

Diphtheria is an acute, contagious disease caused by the bacterium Corynebacterium diphtheria, characterized by the production of a systemic toxin. The toxin is particularly damaging to the tissue of the heart and CNS. **Immunization against diphtheria is available to all children in the U.S.**

All of the following statements concerning **acute necrotizing ulcerative gingivitis** are true **except:**

- It is also called Vincent's infection, Vincent's angina or "trench mouth"
- It is a gingival disease characterized by painful hyperemic gingiva, punched out erosions of the interproximal papilla, covered by a gray pseudomembrane with an accompanying fetid odor
- Risks include poor oral hygiene, poor nutrition, smoking, and emotional stress
- It usually affects children
- Fusiforms and spirochetes, as well as Prevotella intermedia, have been implicated in the etiology of ANUG

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• **It usually affects children**

***This is **false;** ANUG occurs in young to middle-aged adults.

ANUG is an acute fusospirochetal infection of the gingiva. It involves a progressive painful infection with ulceration, swelling and sloughing off of dead tissue from the mouth and throat due to the spread of infection from the gums. It is usually associated with poor oral hygiene and is most common in conditions where there is crowding and malnutrition. **It is rare in preschool children.**

It can be **easily diagnosed** because of the involvement of the interproximal papillae and the presence of a pseudomembranous necrotic covering of the marginal tissues. The clinical manifestations of the disease include inflamed, painful, bleeding gingival tissue; poor appetite; fever; general malaise; and a fetid odor. Treatment includes debridement, hydrogen peroxide mouth rinses, and antibiotic therapy.

Note: Atrophic gingivitis is characterized by gingival recession without a corresponding rate of alveolar bone loss. Minor marginal and papillary gingival inflammation is found. The predominant clinical finding is the recession.

A **Class I cleft palate** involves what structures?

- Hard and soft palates
- Soft palate only
- Alveolar process only
- Hard palate only

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- **Soft palate only**

Four Classes of Cleft Palate:

- **Class I** → involves **only the soft palate.**
- **Class II** → involves **soft and hard palates** but not the alveolar process.
- **Class III** → same as Class II **but with alveolar process involvement** on one side of the premaxilla.
- **Class IV** → involves the **soft palate** and **continues through the alveolus on both sides of the premaxilla.**

***Females more often affected**

Four Classes of Cleft Lip:

- **Class I** → a unilateral notching of the vermillion **not** extending into the lip.
- **Class II** → same as Class I **but** the cleft extends into the lip **but not** to the floor of the nose.
- **Class III** → same as Class II **but** extending into the floor of the nose.
- **Class IV** → any **bilateral clefting of the lip** whether incomplete notching or complete clefting.

***Males more often affected**

An **outstanding oral manifestation** of both ectodermal dysplasia and cleidocranial dysplasia is:

- Oversized crowns
- Elongated roots
- Enlarged mandible
- Retained primary teeth

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- **Retained primary teeth**

Cleidocranial dysplasia *(or dysostosis)* is a rare condition inherited as an autosomal dominant and characterized by partial or complete absence of the clavicles, defective ossification of the skull, and faulty occlusion due to missing, misplaced, or supernumerary teeth. It is equally common in males and females. Prolonged retention of primary teeth and delayed or complete failure of eruption of permanent teeth are characteristic features. The presence of numerous supernumerary and unerupted permanent teeth is very common.

Ectodermal dysplasia is a hereditary condition characterized by abnormal development of the skin and associated structures (*hair, nails, teeth, and sweat glands*). There are several different types of ectodermal dysplasia of which x-linked anhidrotic ectodermal dysplasia is the most common. This condition affects only males. Teeth develop abnormally and there may be complete failure of the teeth to develop (*anodontia*) or **oligodontia** (*partial anodontia*). The anterior teeth often have a conical shape, which is characteristic of oligodontia associated with ectodermal dysplasia.

Note: Anhidrotic ectodermal dysplasia is characterized by **lack of perspiration** caused by the partial or complete absence of sweat glands.

Important: Whether retained primary teeth cause a change in the path of eruption of succeeding teeth is uncertain.

Upon oral examination, a three year-old child is found to have intensely inflamed gingiva, a sore throat, fever, lymphadenopathy, and small fluid-filled vesicles on the mucosa of the lips, tongue and gingiva. **The diagnosis is probably:**

- Chicken pox
- Primary (*acute*) herpetic gingivostomatitis
- Scarlet fever
- Acute necrotizing ulcerative gingivitis (*ANUG*)

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• **Primary (*acute*) herpetic gingivostomatitis**

***Other symptoms may include **fever** and **malaise.**

This disease is self-limiting, and the acute phase generally lasts 7-10 days. Oral fluids are very important in children so that they **do not become dehydrated.**

Differences Between ANUG and Acute Herpetic Gingivostomatitis	
ANUG	**Acute Herpetic Gingivostomatitis**
Debridement helps clear up infection	Debridement doesn't help clear up infection
Antibiotics will reduce acute symptoms	Antibiotics do not help this viral infection
Rarely occurs in preschool-aged children	Seen in preschool-aged children
Onset is slow	Onset is rapid
Caused by spirochetes, fusobacterium, and prevotella intermedia	Caused by virus
Necrotic, foul-smelling ulcers of the gums and throat	Round ulcers with red areolae on the lips, tongue, gingiva and cheeks

Important: The main differential diagnosis for **primary herpetic gingivostomatitis** in patients with predominately gingival involvement without or with few discrete lesions is **acute necrotizing ulcerative gingivitis** *(ANUG)*. Patients with ANUG also present with a sudden onset of a sore mouth. However, ANUG can be differentiated from primary herpes by the fact that in ANUG the interdental papillae are necrotic while in primary herpes, the interdental papillae are intact. In individuals with **primary herpes** manifesting multiple oral ulcerations, **aphthous stomatitis** must be considered in the diagnosis. However, primary herpes can be distinguished from aphthous stomatitis by lesion location and history. Aphthous ulcers occur only on mobile or unattached mucosa and there is a history of recurrence. **In contrast,** primary herpetic lesions occur on both mobile and attached mucosa and there is no history of previous episodes. Most patients with aphthous stomatitis do not have systemic symptoms such as fever.

_____develops as the infection spreads from the bone to the adjacent soft tissue. Subsequently, inflammation and edema occur, and the patient develops a poorly localized swelling. On palpation the area is often sensitive, but the sensitivity is not discrete.

- Suppuration
- Osteomyelitis
- Cellulitis
- Periapical osteitis

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• **Cellulitis**

3 clinical stages of odontogenic infection:

1. **Periapical osteitis:** occurs when the infection is localized within the alveolar bone. Although the tooth is sensitive to percussion and often slightly extruded, there is no soft tissue swelling.
2. **Cellulitis:** develops as the infection spreads from the bone to the adjacent soft tissue. Subsequently, inflammation and edema occur, and the patient develops a poorly localized swelling. On palpation the area is often sensitive, but the sensitivity is not discrete.
3. **Suppuration** then occurs and the infection localizes into a discrete, fluctuant abscess.

Cellulitis may be caused by a **necrotic primary or permanent tooth.** It often causes considerable swelling of the face or neck, and the tissue appears discolored. It is a very serious infection and it can be life-threatening. The child will appear acutely ill and may have a very high temperature with malaise and lethargy. **Note:** The **most common causative organisms** are Group A Streptococci and Staphylococcus aureus.

Important: Cellulitis in a child is harder to treat because **dehydration** occurs more frequently, rapidly, and severely in children than in adults.

If it involves the submandibular, sublingual, and submental space it is called **"Ludwig's angina".** In this condition, the tongue and floor of the mouth become elevated and the patient's airway is obstructed and swallowing is impossible. The treatment for cellulitis should include having the child go to the hospital if the signs and symptoms warrant it. In the case of Ludwig's angina, **it is mandatory.**

Historically the incidence of dental decay in persons with **Down syndrome** has been reported to be _____. The rate of periodontal disease in these persons has been reported to be _____.

- Extremely high, extremely low
- Relatively the same as the general population, extremely high
- Extremely low, relatively the same as the general population
- Extremely low, extremely high

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• **Extremely low, extremely high**

Down syndrome is a congenital defect caused by a chromosomal abnormality *(trisomy 21)*. The primary skeletal abnormality affecting the orofacial structures in Down syndrome is an **underdevelopment or hypoplasia** of the midfacial region. The bridge of the nose, bones of the midface and maxilla are relatively smaller in size. In many instances this causes a **prognathic Class III** occlusal relationship which contributes to an **open bite.** The tongue may protrude and appear to be too large. With age, both the tongue and the lips in people with Down syndrome tend to develop cracks and fissures. This is a result of **chronic mouth breathing.** The eruption of teeth in persons with Down syndrome is usually **delayed** and may occur in an unusual order. There is an extremely high rate of missing teeth in both the primary and permanent dentitions. The roots of the teeth in patients with Down syndrome tend to be small and conical.

The **clinical features** of Down syndrome are fairly recognizable and include:

- Delayed physical and mental development
- Short, stocky build
- The face is broad and flat, with slanting eyes and a short nose
- The ears are small and low set
- Heart defects are common. **Important:** SBE prophylaxis is required for dental treatment

The child with Down syndrome is said to be affectionate, fearful of quick movements, but capable of learning dental procedures. These children need a comprehensive preventive program. These patients often have difficulty accepting dental care but cooperation can be improved by using **gradual exposure** to the dental office.

All of the following are clinical features of insulin-dependent diabetes mellitus *(IDDM)* **except:**

- Polydipsia and polyuria due to osmotic diuresis
- Malaise
- Often the patient is obese
- Bacterial and fungal infections
- Cramps
- Blurred vision
- Abdominal pains

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- **Often the patient is obese**

***This is **false;** IDDM is characterized by **weight loss** due to dehydration and catabolism.

Diabetes is a disease in which your body is unable to properly use and store glucose. Glucose backs up in the bloodstream causing your blood glucose or "sugar" to rise too high. Though people with diabetes experience many symptoms, the presence of the **classical triad** is always present. This **consists** of:

- **Polydipsia** → being very thirsty
- **Polyphagia** → being very hungry
- **Polyuria** → having to frequently urinate

Juvenile diabetics tend to be unstable and brittle. They are prone to ketoacidosis. A serious complication that can develop is **blindness.**

Oral complications of uncontrolled Type 1 diabetes:

- Xerostomia
- Infection
- Poor healing
- Increased incidence and severity of **periodontal disease,** due to:
  - Increased plaque
  - Elevated glucose levels
  - Loss of collagen in gingival tissue
  - Reduced circulation in tiny blood vessels in the gingival tissues *(caused by diabetes)* interferes with nutrition and healing in the gingival tissues
- Burning mouth syndrome

Remember: People with Type 1 diabetes are **totally insulin deficient** and therefore must take daily insulin injections to survive.

Which of the following is the most common **malignant** tumor presenting in neonates?

- A lymphangioma
- A hemangioma
- A neurofibroma
- A neuroblastoma

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• **A neuroblastoma**

Neuroblastoma is one of the most common solid tumors of early childhood usually found in babies or young children. The disease originates in the adrenal medulla or other sites of sympathetic nervous tissue. The most common site is the **abdomen** *(near the adrenal gland)* but can also be found in the chest, neck, pelvis, or other sites. Most patients have widespread disease at diagnosis.

Hemangiomas are vascular birthmarks in which the proliferation of blood vessels leads to a mass that resembles a neoplasm. Hemangiomas differ from other vascular birthmarks in that they are biologically active; their growth is independent from the growth of a child. Most hemangiomas appear within a week or two after birth. They are 5 times more common in girls than boys. They are common on **lips, tongue and buccal mucosa.** These lesions appear as flat or raised, usually deep red or bluish red and seldom well-circumscribed. They are removed surgically, others require no treatment.

A **lymphangioma** is a fairly well-circumscribed nodule or mass of lymphatic vessels. They occur most frequently in the neck and axilla. These lesions appear as red to blue translucent enlargements that are compressible and spongy. They are treated by excisional biopsy.

A **neurofibroma** is a moderately firm, encapsulated tumor resulting from the proliferation of Schwann cells. They occur on the tongue, buccal mucosa, vestibule and palate. These lesions appear as solitary or multiple submucosal enlargements. May become malignant *(5-15%).* Multiple lesions are associated with **neurofibromatosis** *(von Recklinghausen's disease).*

An outstanding oral manifestation of **achondroplasia** is:

- Rampant caries
- Periodontal disease
- Overcrowding of teeth
- Supernumerary teeth

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- **Overcrowding of teeth**

Achondroplasia is the most common form of short-limb dwarfism. It occurs in all races and with equal frequency in males and females. An individual with achondroplasia has a disproportionate short stature → the head is large and the arms and legs are short when compared to the trunk length. Other signs are a prominent forehead and a depressed bridge of the nose. Many of these children die during the first year of life. Deficient growth in the cranial base is evident in many children that survive. **The maxilla may be small with the resultant crowding of the teeth.**

Note: A **Class III** malocclusion is **very common.**

Remember → the **oral manifestations** of the following disorders in children:

- **Gigantism** → **enlarged tongue**, mandibular prognathism, teeth are usually tipped to the buccal or lingual side, owing to enlargement of the tongue. Roots may be longer than normal.

- **Pituitary dwarf** → the eruption rate and the shedding of the teeth are **delayed**, clinical crowns appear smaller as do the roots of the teeth, the dental arch as a whole is smaller causing malocclusion, and the **mandible is underdeveloped.**

All of the following are oral manifestations of Apert syndrome **except:**

- Prominent mandible
- High arched palate
- Bifid uvula
- Cleft palate
- Severely crowded maxillary teeth
- Class II malocclusion
- Shovel-shaped incisors
- Supernumerary teeth

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• **Class II malocclusion**

***This is **false;** a **Class III** malocclusion is common.

Apert syndrome is a genetic defect and falls under the broad classification of cranial / limb anomalies. It is primarily characterized by specific malformations of the skull, midface, hands and feet.
Note: The retrusion of the midface is often corrected by performing a **Lefort III** surgical procedure.

Remember:

1. **Crouzon syndrome** is an uncommon, autosomal dominant craniofacial disorder characterized by craniosynostosis and dysmorphic facial features.
   Clinical features include:
   - Early childhood, no gender predilection
   - Maxillary hypoplasia, reduced width of the dental arch and crowded teeth
   - Short upper lip
   - Short head, widely spaced eyes, shallow orbits and protruding eyeballs
   - Calcified stylohyoid ligaments
   - Possible unilateral or bilateral posterior crossbite
2. **Rieger's syndrome** is characterized by delayed sexual development and hypothyroidism. This syndrome has important dental considerations, which include: hypodontia, an underdeveloped premaxillary area, cleft palate and a protruding lower lip.
3. **Treacher Collins Syndrome,** also called mandibulofacial dysostosis, is an autosomal dominant disorder characterized by certain facial features including downward slanting palpebral fissures, sunken cheekbones, receding chin, and malformed ears. Other clinical features include:
   - Narrow face
   - Severe mandibular hypoplasia
   - Possible widely-spaced, hypoplastic and / or displaced

Which of the following is **true** concerning a young epileptic who has a grand mal seizure in the dental office?

- It is **generally fatal**
- It is best **treated by injecting insulin**
- They generally recover **if restrained from self-injury and oxygen is maintained**
- It can be **prevented with antibiotics**

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• **They generally recover if restrained from self-injury and oxygen is maintained**

Grand mal epilepsy is the **most common** form of seizure disorder *(present in 90% of epileptics)*. Approximately 60% of epileptics have this form alone, whereas 30% have other seizure types in addition to grand mal. Grand mal epilepsy **may occur** in any age group. Grand mal seizures usually last from 2 to 5 minutes.

A seizure often has **three** distinct phases: **aura, ictus, and postictal** state. The first phase involves alterations in smell, taste, visual perception, hearing, and emotional state. This is known as an **aura,** which is actually a small partial seizure that is often followed by a larger event. The seizure is known as **ictus.** Following a seizure, the person enters into the **postictal** state. Drowsiness and confusion are commonly experienced during this phase. The postictal state is the period in which the brain recovers from the insult it has experienced.

The treatment for the **ictus phase** includes positioning the patient in a supine position, preventing injury to the convulsing patient, initiating basic life support *(head tilt)*, and if cyanosis occurs, administering oxygen.

Important: If the ictus phase of the seizure lasts more than 5 minutes, EMS should be called. **Status Epilepticus is a medical emergency.** If the dentist is trained to do so, an IV line should be started, and a dose of 25–50 ml of 50% dextrose should be given immediately in case the cause of the seizure is hypoglycemia. If there is no response, the patient should be given 10 mg of diazepam IV over a 2–minute period. The patient's vital signs must be monitored, because the diazepam may cause respiratory depression. The dose of diazepam may be repeated after 10 minutes, if necessary.

The most common of the **craniofacial malformations** is:

- Bifid tongue
- Macroglossia
- Cleft palate and cleft lip
- Anodontia

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• **Cleft palate and cleft lip**

***Cleft palate and cleft lip account for half of the total number of defects. Of all cases, 25% are cleft palate alone and 75% are cleft lip with or without cleft palate.

The lip and primary palate begin to develop at **four to five weeks** gestational age. The two medial nasal swellings and the maxillary swellings fuse to form the upper lip. Failure of this fusion results in cleft lip. Clefts of the lip are more frequent in **males.** Cleft lip involvement is **more frequent** on the left side than the right.

The secondary palate develops at approximately **nine weeks** developmental age. The paired palatal shelves arise from the intraoral maxillary processes. These shelves, originally in a vertical position, reorient to a horizontal position as the tongue assumes a more inferior position. The shelves then fuse anteriorly with the primary palate, which arises from the median nasal process posteriorly and with one another. Failure of fusion results in a cleft palate. Cleft palate is more frequent in **females.**

The most severe handicap imposed by cleft palate is an impaired mechanism preventing normal speech and swallowing. The child will almost always need orthodontic treatment once the palate is surgically repaired. Also, speech therapy will be needed because these patients have problems related to the **inability of the soft palate to close** the air flow into the nasopharynx. Orthognathic surgery may be needed to correct the general concave appearance of the face. This concave appearance is generally due to deficient maxillary growth.

Which type of leukemia is referred to as the "leukemia of childhood"?

- Acute myeloid leukemia
- Chronic myelocytic leukemia
- Acute lymphocytic leukemia
- Chronic lymphocytic leukemia

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• **Acute lymphocytic leukemia** *(ALL)*

Acute lymphocytic *(lymphoblastic)* **leukemia** is a life-threatening disease in which the cells that normally develop into lymphocytes *(lymphoblasts)* become cancerous and rapidly replace normal cells in the bone marrow. The peak age is around four years old, and it is the form of acute leukemia that is **most responsive** to therapy. It can be successfully treated, with a 50–70% 5–year survival rate.

The early signs of **acute leukemia** in a child include fatigue, pallor, weight loss and easy bruising. This will progress to fever, hemorrhages, extreme weakness, bone and joint pain, and repeated infections.

Oral findings include:
• Gingival oozing, petechiae, hematoma, or ecchymosis
• Oral ulceration, pharyngitis, and gingival infection which is unresponsive to conventional therapy
• Submandibular lymphadenopathy

Note: Candidiasis is **common** in children with leukemia because they are especially susceptible to this fungal infection. Nystatin rinses or popsicles are effective in clearing up this infection.

Hodgkin's Lymphoma or Hodgkin's Disease is a malignant growth of cells in the lymph system. Hodgkin's Disease is the better known form of lymphoma (*the other lymphomas are grouped into what is called the* ***Non-Hodgkin's*** *Lymphomas)*. The most common symptom of **Hodgkin's** disease is painless swelling of the lymph nodes in the neck, underarm, or groin. The common symptoms of **Non-Hodgkin's** disease include: painless swelling in the lymph nodes in the neck, underarm, or groin; persistent fever; feeling of fatigue; unexplained weight loss; itchy skin and rashes; small lumps in skin; bone pain; swelling in the abdomen; liver or spleen enlargement.

Congenital porphyria produces distinct discoloration of the teeth. Discoloration occurs due to excess porphyrins in the blood during mineralization of the teeth. Affected teeth are usually _____, but _____when viewed under a UV light.

- Pinkish-brown, scarlet
- Black, brown
- Blue-green, scarlet
- Yellowish-gray, green

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- **Pinkish-brown, scarlet**

Congenital porphyria is inherited as an autosomal recessive trait. The skin becomes light brown and sensitive to sunlight and photosensitivity is expressed as large bullous lesions. Scarring is frequent.

Other causes of intrinsic tooth discoloration:

- Children with **cystic fibrosis** have teeth that are **dark in color,** ranging from yellowish-gray to dark brown. This may be related to the usual high doses of tetracycline given to children with cystic fibrosis.
- **Erythroblastosis fetalis** is characterized by an excessive destruction of erythrocytes. The primary teeth may have a characteristic blue-green color.
- **Tetracycline therapy** can cause the crowns of teeth to become discolored, ranging from **yellow to brown and from gray to black.** The drug will stain permanent teeth that have not completed enamel formation at the time the drug is given. **For example:** If a five year-old child receives tetracycline therapy, the teeth affected will be the canines, premolars, and second molars. **Important:** The incisors and first molars have already completed enamel formation. **Note:** Review the card that shows when **enamel calcification** is completed on the various teeth.
- **Amelogenesis imperfecta:** teeth vary in color from white opaque to yellow to brown.
- **Dentinogenesis imperfecta:** opalescent teeth.
- **Dental fluorosis:** yellow to brown pigmentation.
  Hyperbilirubinemia: jaundice-like yellow-green tint on the tooth surfaces.

Gingivostomatitis is a condition that is:

- Common, particularly among elderly-people
- Common, particularly among middle-aged adults
- Common, particularly among children
- Uncommon

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• **Common, particularly among children**

Gingivostomatitis is a disorder involving sores on the mouth and gingiva that result from a viral infection *(HSV-1)*. It is characterized by inflammation of the gingiva and mucosa and multiple mucosal ulcerations.

Important: Acute *(primary)* herpetic gingivostomatitis generally affects children under the age of three. There are **prodromal symptoms** *(fever, malaise, irritability, headache, dysphagia, vomiting and lymphadenopathy)* that occur 1-2 days prior to the local lesions *(ulcers)* in the oral cavity.

The treatment in children should be directed toward the relief of the **acute** symptoms so that fluid and nutritional intake can be maintained. Symptomatic treatment for primary herpes consists of rinsing with a 50:50 suspension of Benadryl Kaopectate and / or Viscous Lidocaine. The anti-viral drug used most frequently today to shorten the duration and severity of the primary infection is acyclovir *(Zovirax)*. It is prescribed *(400 mg. q.i.d.)* for 1-2 weeks.

Complications are unusual. However children can become dehydrated and can transmit the infection to their eye*(s)* by rubbing. Herpes keratitis if not properly treated can lead to blindness over time.

Common sequelae to acute herpetic gingivostomatitis include:

- Recurrent herpes labialis *(cold sores on the lips)*. Exposure to sunlight may be responsible for the appearance of the cold sores on the lips. Acyclovir 5% ointment may be applied topically at the first symptom of the lesion *(tingling sensation)*.
- Child will have circulating anti-herpes antibodies *(HSV-1 antibodies)*.

Nursing-bottle caries is a widespread carious destruction of the deciduous teeth. Which of the deciduous teeth are **most commonly** affected?

- Mandibular incisors
- Maxillary first molars
- Mandibular first molars
- Maxillary incisors

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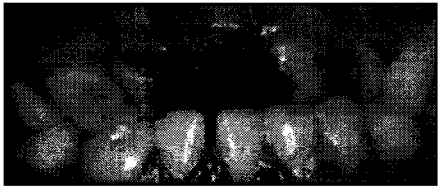
• Maxillary incisors

Inappropriate feeding of children can lead to typical nursing pattern decay. There is early carious involvement of the maxillary anterior teeth followed by the maxillary and mandibular first molars and mandibular canines. The mandibular incisors are in general less affected since the tongue covers them. **Note:** Nursing-bottle caries is also called baby bottle tooth decay *(BBTD)*, bottle-mouth syndrome, early childhood caries, nursing caries, bottle caries and infant caries. **Nursing-bottle caries** is a rampant decay that results from sleep time bottle-feeding combined with the activity of Streptococcus mutans. The stagnation of milk about the necks of anterior teeth and the fermentation of the disaccharide lactose, a sugar found in milk, contribute to this caries process as well.



Preventive measures include:

- Infants should **not be** put to sleep with a bottle containing a liquid **other than water**
- Infants should be encouraged to drink from a cup prior to their first birthday
- Infants should be weaned from the bottle at 12-14 months of age
- Infants should start to supplement their diet with nonliquids at 4-6 months of age
- Juices should only be offered from a cup
- Oral hygiene should be started with eruption of the first primary tooth
- Within six months of eruption of the first tooth *(no later than the first birthday)* it is time for the **first dental visit**

The **most common** congenitally missing **primary tooth** is the:

- Primary mandibular canine
- Primary maxillary lateral incisor
- Primary maxillary canine
- Primary mandibular first molar

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• **Primary maxillary lateral incisor** → **Note:** This is rare

The most common congenitally missing **permanent tooth** is the third molar, followed by the mandibular second bicuspid and the maxillary lateral incisor.

Notes:

1. The maxillary lateral incisor is most often **atypical in size** *(peg-shaped, etc.)*.
2. A patient who has permanent central incisors, permanent canines, and primary canines anterior to the premolars most likely has **congenitally missing** permanent lateral incisors.

Heredity is **most frequently** responsible for the congenital absence of teeth. The roots of the primary tooth will resorb slower than normal without the presence of the permanent tooth. **As a general rule,** if only one tooth is or a few teeth are missing, the absent tooth will be the most distal tooth of any given type. If a molar tooth is congenitally missing, it is almost always the third molar. If an incisor is missing, it is nearly always the lateral. If a premolar is missing, it almost always is the second rather than the first. Rarely is a canine the only missing tooth.

Important: In the case of a **congenitally missing second bicuspid,** you want to hold onto the primary second molar as long as possible. If it is still present it may be ankylosed. Cessation of eruption is **most diagnostic** of an ankylosed primary molar.

Remember: Space maintenance is of utmost importance because whenever primary or permanent teeth are congenitally missing or lost prematurely and arch integrity is lost, loss of space and arch length, perimeter, or circumference may result. Migration of primary and / or permanent teeth can occur and the available space may be reduced by an amount sufficient to cause some degree of crowding in the permanent dentition.

A **15-month-old** child would normally have all of the following teeth erupted **except** the:

- Primary lateral incisors and canines
- Primary canines and first molars
- Primary canines and second molars
- Primary central and lateral incisors
- Primary first and second molars

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• **Primary canines and second molars**

Rule of four → this simplified rule will enable you to determine the number of teeth present at any given time. It implies the eruption of **four teeth** every **four months** beginning with **four teeth** at age seven months.

Rule of Four		
Age *(in Months)*	Number of Teeth Erupted	Specific Teeth
7	4	4 → mand. incisors
11	8	8 → mand. and max. central and lateral incisors, four first molars
15	12	12 → mand. and max. central and lateral incisors, four first molars
19	16	16 → mand. and max. central and lateral incisors, four first molars and four canines
23	20	20 → mand. and max. central and lateral incisors, four first molars, four canines, and four second molars

Example from question on front of card: At age 15 months, 12 teeth are erupted → four centrals, four laterals, and four first molars.

Listed below are the **usual events** in the histogenesis of a tooth. Place them in their **correct sequence** → from what happens first to what happens last.

- Deposition of the first layer of dentin
- Differentiation of odontoblasts
- Deposition of the first layer of enamel
- Elongation of the inner enamel epithelial cells of the enamel organ

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Histogenesis of a tooth in sequence

1. **Elongation of the inner enamel epithelial cells of the enamel organ** → this influences mesenchymal cells on the periphery of the dental papilla to differentiate into odontoblasts *(#2 below)*
2. **Differentiation of odontoblasts**
3. **Deposition of the first layer of dentin**
4. **Deposition of the first layer of enamel**

Tooth development is dependent on a series of sequential cellular interactions between **epithelial** and **mesenchymal components** of the tooth germ. Once the ectomesenchyme **influences the oral epithelium** to grow down into the ectomesenchyme and become a tooth germ, the above events occur.

Notes:

1. Some texts include the **deposition of root dentin and cementum** as #5 in the histogenesis of a tooth.
2. **Korff's fibers** is a name given to the rope-like grouping of fibers in the periphery of the pulp that seem to have something to do with the formation of the dentin matrix.

Remember: Histogenesis means the **formation and development** of the tissues of the body, in this case the tooth.

The stage of tooth development in which the **physiological process of differentiation** occurs to its fullest extent is called:

- Initiation
- Bud stage
- Cap stage
- Bell stage
- Appositional stage
- Maturation stage

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• **Bell stage**

Six stages of tooth development *(odontogenesis)*

1. **Initiation** stage involves the physiological process of **induction.** Occurs at the beginning of the 5th week. Initial interaction between oral epithelium and mesenchyme *(ectomesenchyme)*, formation of **dental lamina.**
2. **Bud stage** involves the physiological process of **proliferation.** Occurs at the beginning of the 8th week. Growth of the dental lamina into 10 buds per arch. Underlying mesenchyme also undergoes proliferation and the shape of the tooth becomes evident. Enamel organ starts to form.
3. **Cap stage** involves the physiological processes of **proliferation and differentiation,** which occurs in two ways (1) **morphodifferentiation** → to change into other shapes (2) **histo-differentiation** → to branch into different tissues. Occurs from the 9th to the 10th week.

   ***By the end of the cap stage, the **tooth germ is complete** and consists of the enamel organ, the dental papilla, and the dental sac.

4. **Bell stage** → differentiation occurs to its furthest extent resulting in four different cell types in the bell-shaped **enamel organ:** OEE→ cuboidal, IEE→ columnar, Stellate reticulum → network of star-shaped cells, Stratum intermedium→ flat to cuboidal. The **dental papilla** also differentiates into two layers **(1)** outer cells and **(2)** central cells. The **dental sac** increases in collagen fibers. Occurs between the 11th and 12 weeks.
5. **Appositional stage** → cells that were differentiated into specific tissue-forming cells begin to **deposit the specific dental tissues** *(enamel, dentin, cementum, and pulp)*
6. **Maturation stage** → mineralization begins at the DEJ and continues until total development accomplished, taking approximately two years to complete.

As the crown portion of the tooth nears completion, the outer enamel epithelium and the inner enamel epithelium combine at the cervical loop region to form a bilayered structure termed:

- The dental lamina
- Hertwig's epithelial root sheath

Notes:

1. Tooth development is **initiated by** the mesenchyme's inductive influence on the overlying ectoderm.
2. The enamel of a tooth is **derived from** the ectoderm of the oral cavity. All other tissues of the tooth differentiate from the associated mesenchyme *(mesoderm)*.
3. Ectodermal cells are **responsible** for determining crown root and shape.

The **first deciduous** *(primary)* **tooth** to erupt is the:

- Mandibular central incisor
- Mandibular first molar
- Maxillary central incisor
- Maxillary first molar

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• **Mandibular central incisor**

Permanent Teeth Eruption Chart			
	Tooth	First Evidence of Calcification	Eruption
Maxillary	Central	3-4 months	7-8 years
	Lateral	10 months	8-9 years
	Canine	4-5 months	11-12 years
	First premolar	1 1/2 - 1 3/4 years	10-11 years
	Second premolar	2 - 2 1/4 years	10-12 years
	First molar	At birth	6-7 years
	Second molar	2 1/2 - 3 years	12-13 years
	Third molar	7-9 years	17-21 years
Mandibular	Central	3-4 months	6-7 years
	Lateral	3-4 months	7-8 years
	Canine	4-5 months	9-10 years
	First premolar	1 3/4 - 2 years	10-12 years
	Second premolar	2 1/4 - 2 1/2 years	11-12 years
	First molar	At birth	6-7 years
	Second molar	2 1/2 - 3 years	11-13 years
	Third molar	8-10 years	17-21 years

Primary Teeth Eruption Chart			
	Tooth	First Evidence of Calcification	Eruption
Maxillary	Central Incisor	4 mos. *in utero*	7 1/2 mos.
	Lateral Incisor	4 1/2 mos. *in utero*	8 mos.
	Canine	5 1/2 mos. *in utero*	16-20 mos.
	First molar	5 mos. *in utero*	12-16 mos.
	Second molar	6 mos. *in utero*	20-30 mos.
Mandibular	Central Incisor	4 1/2 mos. *in utero*	6 1/2 mos.
	Lateral Incisor	4 1/2 mos. *in utero*	7 mos.
	Canine	5 mos. *in utero*	16-20 mos.
	First molar	5 mos. *in utero*	12-16 mos.
	Second molar	6 mos. *in utero*	20-30 mos.

Notes:

1. The **first permanent** tooth to erupt is the **mandibular first molar,** followed shortly thereafter by the maxillary first molar.
2. The **first permanent** tooth to begin calcifying **is the mandibular first molar** *(at birth).*
3. The **first succedaneous** tooth to erupt is the **mandibular** central incisor.

Remember: The mandibular first molar and the maxillary first molar **are not** succedaneous teeth.

Ordinarily, a **6-year-old child** would have what teeth **clinically visible** in the mouth?

- All *(20)* primary teeth and 4 permanent first molars
- 18 primary teeth and 2 permanent mandibular central incisors
- 18 primary teeth, 2 permanent mandibular central incisors, and 4 permanent first molars

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- **All *(20)* primary teeth and 4 permanent first molars** *("6 year molars")*

Remember:

- The permanent mandibular centrals erupt between the ages of 6 –7.
- The permanent maxillary centrals erupt between the ages of 7–8.

Note: A **7–year-old** child would have the following teeth present clinically:

- **18 primary and 6 permanent teeth** → the **6 permanent teeth** include:
  - — Mandibular first molars *(2)* → right and left
  - — Maxillary first molars *(2)* → right and left
  - — Mandibular central incisors *(2)* → right and left

***All of the primary teeth **except** the two mandibular central incisors **(20 – 2 = 18)**.

Which primary mandibular molar has a **prominent transverse ridge** that unites the mesiobuccal and mesiolingual cusps?

- The primary mandibular **first** molar
- The primary mandibular **second** molar

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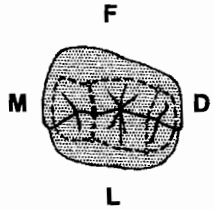
• **The primary mandibular first molar**

This **transverse ridge** separates the mesial portion from the remainder of the occlusal surface.

Other characteristics of the primary mandibular first molar:

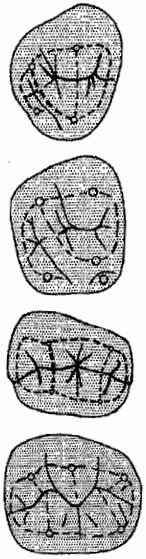
- It **does not resemble** any other primary or permanent tooth.
- The mesiobuccal cusp is always the **largest** and **longest** cusp, occupying nearly two-thirds of the buccal surface.
- The mesiolingual cusp is **larger, longer,** and **sharper** than the distolingual cusp.
- Crown is **wider mesiodistally** than high cervico-occlusally.
- The mesial marginal ridge is **very well developed** and resembles a cusp.
- It has a **prominent** mesiobuccal **cervical ridge.**
- Class II cavity preparations **are difficult** due to morphology.
- It has **no** central fossa.



Occlusal view of primary mandibular right first molar

Match the primary molar tooth on the left with the appropriate drawing on the right.

- Primary mandibular right first molar
- Primary mandibular right second molar
- Primary maxillary right first molar
- Primary maxillary right second molar



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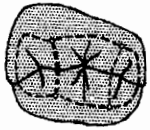
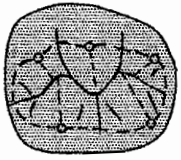
- Primary mandibular right first molar →→→→→→

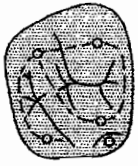


- Primary mandibular right second molar →→→→



- Primary maxillary right first molar →→→→→→→

- Primary maxillary right second molar →→→→→



The **permanent mandibular first molar** has a morphology that closely resembles the:

- Primary mandibular first molar
- Primary mandibular second molar
- Primary maxillary first molar
- Primary maxillary second molar

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• **Primary mandibular second molar**

Note: Amalgam prep outlines on these two teeth **also resemble** one another.

Differences include:

- Relative size of the **distal cusp.** The primary molar has its mesiobuccal, distobuccal, and distal cusp almost equal in size. The distal cusp of the permanent molar, however, is smaller than the other two cusps.
- From the buccal aspect, the primary mandibular second molar has a narrow mesiodistal dimension at the cervical portion of the crown when compared with the dimension mesiodistally on the crown at the contact level. The mandibular first **permanent** molar, accordingly, is wider at the cervical portion.
- **Groove patterns** are different on the occlusal surface.
- The primary molar has more divergent roots to allow for the eruption of the second pre-molar.
- The primary molar has a more prominent facial crest of contour.

Permanent mandibular right first molar

Primary mandibular right second molar

Notes:

1. The **primary teeth** that present the most **noticeable morphologic deviations** from the permanent teeth are the **first molars.**
2. The **primary second molar** has the **greatest faciolingual diameter** of all primary teeth.

Which tooth is the only anterior tooth in either dentition to have a shorter inciso-cervical height than the mesio-distal width?

- The primary mandibular central incisor
- The primary mandibular lateral incisor
- The primary maxillary lateral incisor
- The primary maxillary central incisor

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• **The primary maxillary central incisor**

The primary **maxillary central** incisor resembles the permanent maxillary central in shape. It is much smaller in size than the permanent maxillary central and has a more pronounced cervical line. The crown is the only anterior tooth in either dentition to have a shorter inciso-cervical height than the mesio-distal width. This tooth erupts with **no mamelons,** and the labial surface is convex and smooth.

The primary **maxillary lateral** incisor is similar to the central incisor **except** it is smaller. Another difference is that it is longer than it is wide. The incisal edge of the primary maxillary lateral incisor is more rounded on the mesial and distal sides than the straight incisal edge of the central incisor.

The primary **mandibular central** incisor more closely resembles the permanent mandibular lateral incisor than its central incisor counterpart. The crown of the tooth is slightly wider than the permanent lateral incisor. The shape and form of the incisal edge is almost exactly the same as that of the permanent lateral. The root is slender and rather long. Mesial and distal surfaces of the root are flat, while lingual and labial surfaces are convex.

The primary **mandibular lateral** resembles the primary mandibular central incisor **except** that it is slightly longer and wider. The cingulum and the mesial and distal marginal ridges are more pronounced and the fossa is not as shallow. The root curves toward the distal at the apex.

When **extracting primary incisors** in which the roots have been partially resorbed due to pressure from the developing permanent teeth, the **facial** part of the remaining primary root will usually be the longest and the most securely attached to the gingiva.

Note: Labial and lingual cervical ridges **are prominent** on all primary incisors.

Morphologically, the **primary maxillary second molar** strikingly **resembles** the:

- Permanent maxillary third molar
- Permanent maxillary second molar
- Permanent maxillary first molar
- Permanent mandibular second molar

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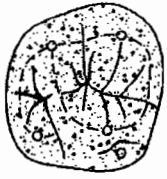
- **Permanent maxillary first molar** → but the primary maxillary second molar is smaller

In general, the **primary second molars** are larger than the first primary molars and resemble the form of the first permanent molars.

Primary maxillary second molar:
- The **faciolingual measurement** of the crown is greater than the **mesiodistal measurement**
- May have a **fifth cusp** *(of Carabelli)*
- Has a **prominent mesiobuccal** cervical ridge
- Has an **oblique ridge**
- MB cusp is **almost equal in size or slightly larger** than the ML cusp
- The largest and longest pulp horn is the **MB**

Primary maxillary right second molar



Permanent maxillary right first molar

The **primary maxillary canine** differs from the **permanent maxillary canine** in which **two** ways?

- The **cusp** on the primary canine is much shorter and rounded
- The **mesial cusp ridge** *(MCR)* is longer than the distal cusp ridge *(DCR)*
- The **cusp** on the primary canine is much longer and sharper
- The **mesial cusp ridge** *(MCR)* is shorter than the distal cusp ridge *(DCR)*

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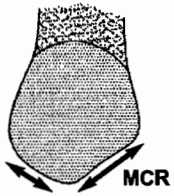
- **The mesial cusp ridge *(MCR)* is longer than the distal cusp ridge *(DCR)***
- **The cusp on the primary canine is much longer and sharper**

***This is the **opposite** of all other canines *(including the primary mandibular canine)*. Obviously they **differ in other ways,** but these two differences are the most significant.
Note: The primary maxillary canine **also appears** especially wide and short.

Primary maxillary right canine



Permanent maxillary right canine

Remember:
1. Comparing the **primary** maxillary and mandibular canines:
   - The mandibular canines crown and root are shorter
   - The mesiodistal width of the mandibular canine at the root trunk is greater
   - In a labiolingual dimension, the maxillary canine is much larger
   - The distal cusp ridge is longer than the mesial cusp ridge on the mandibular canine but the opposite is true of the maxillary canine *(this allows for proper intercuspation)*
2. The **permanent maxillary canines** are the teeth that are most likely to be crowded out of the arch, as opposed to the centrals, laterals, etc.
3. The **permanent mandibular second premolars** are most likely to be malposed in cases of mandibular arch space discrepancy.

The primary maxillary **first molar** appears to be intermediate in form and development between a premolar and a molar. Which cusp is the widest and longest?

- Distolingual
- Distobuccal
- Mesiolingual
- Mesiobuccal

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- **Mesiobuccal**

Characteristics unique to **primary maxillary first molars,** which somewhat resemble permanent maxillary premolars:

- There are often four cusps: two larger cusps *(like a maxillary premolar)*, the mesiobuccal cusp, which is the widest and longest, the mesiolingual cusp, which is smaller but the sharpest, and two smaller cusps, the distobuccal and the inconspicuous, sometimes absent, distolingual.
- A notch *(distal to center)* divides the large mesiobuccal cusp from the indistinct distobuccal cusp *(buccal view)*.
- The crown is wider faciolingually than mesiodistally like a maxillary premolar, but unlike other primary molars *(occlusal view)*.
- The mesial marginal ridge is directed distolingually *(occlusal view)*.
- There are three fossae: a large mesial triangular fossa, a medium central fossa, and a minute distal fossa *(occlusal view)*.
- The grooves form an "H" pattern (*somewhat similar to a maxillary premolar).*

Primary maxillary right first molar

The **permanent mandibular second premolar** typically erupts when a child is about:

- 5–6 years old
- 8–9 years old
- 11–12 years old
- 13–14 years old

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• **11–12 years old**

***When a tooth clinically erupts in the mouth, **one-half to two-thirds** of the root structure has usually developed. Apex is fully developed two to three years after eruption.

Tooth	First Evidence of Calcification	Enamel Complete	Eruption	Root Completion
Maxillary				
Central	3-4 months	4-5 years	7-8 years	10 years
Lateral	10 months	4-5 years	8-9 years	11 years
Canine	4-5 months	6-7 years	11-12 years	13-15 years
First premolar	1 1/2 -1 3/4 years	5-6 years	10-11 years	12-13 years
Second premolar	2 - 2 1/4 years	6-7 years	10-12 years	12-14 years
First molar	At birth	3-4 years	6-7 years	9-10 years
Second molar	2 1/2 -3 years	7-8 years	12-13 years	14-16 years
Third molar	7-9 years	12-14 years	17-21 years	19-21 years
Mandibular				
Central	3-4 months	4-5 years	6-7 years	9 years
Lateral	3-4 months	4-5 years	7-8 years	10 years
Canine	4-5 months	6-7 years	9-10 years	12-14 years
First premolar	1 3/4 - 2 years	5-6 years	10-12 years	12-13 years
Second premolar	2 1/4 - 2 1/2 years	6-7 years	11-12 years	13-14 years
First molar	At birth	2 1/2 - 3 years	6-7 years	9-10 years
Second molar	2 1/2 -3 years	7-8 years	11-13 years	13-14 years
Third molar	8-10 years	12-14 years	17-21 years	19-21 years

The crowns of all 20 primary teeth begin to **calcify** between:

- 1 to 2 months in utero
- 2 to 3 months in utero
- 4 to 6 months in utero
- 8 to 9 months in utero

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• **4 to 6 months in utero**

***On the **average** they take 10 months for completion of calcification.

The primary teeth begin to form in utero at about **6 weeks.** Hard tissue formation occurs in all primary teeth by the **18th week** in utero.

The **permanent teeth** begin to develop at approximately **four months of age in utero.** Maxillary and mandibular first molars begin to **calcify at birth.** They are the first to begin calcification. The mandibular third molars are generally the last teeth to begin calcifying. This happens at about 8-10 years of age.

Notes:

1. The **largest primary** tooth is the mandibular second molar.
2. The mandibular lateral incisor is the **smallest primary** tooth.
3. The **largest permanent** tooth is the maxillary first molar.
4. The mandibular central incisor is the **smallest permanent** tooth.

Which teeth are **succedaneous** teeth?

- The permanent maxillary and mandibular **premolars**
- The permanent maxillary and mandibular **first** molars
- The permanent maxillary and mandibular **second** molars
- The permanent maxillary and mandibular **third** molars

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• **The permanent maxillary and mandibular premolars**

A permanent tooth that moves into a position formerly occupied by a primary tooth is called a succedaneous tooth. In **each quadrant,** five permanent teeth, the incisors, canine, and premolars, succeed or take the place of the five primary teeth.

Nonsuccedaneous teeth include:
• The permanent maxillary and mandibular **first** molars
• The permanent maxillary and mandibular **second** molars
• The permanent maxillary and mandibular **third** molars

***These teeth **do not** move into a position formerly occupied by a primary tooth
***These teeth **do not succeed** deciduous teeth

Note: The **last primary tooth** to be **replaced** by a permanent tooth is usually the maxillary canine *(the permanent maxillary canine usually erupts between the age of 11-12).* The permanent mandibular canine usually erupts between the age of 9-10.

Remember: Permanent molars **do not** replace primary teeth *(see above).*

All of the following syndromes demonstrate both hyperdontia and hypodontia **except:**

- Crouzon's disease
- Gardner's syndrome
- Down's syndrome
- Hallerman-Streiff syndrome

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- **Gardner's syndrome**

Syndromes Demonstrating Hypodontia	
Condition	**Characteristics**
Ectodermal dysplasia *(hypohidrotic type)*	Hypotrichosis, aplasia of sweat or sebaceous glands
Chondroectodermal dysplasia	Polydactyly, mesomelic dwarfism, hidrotic ectodermal dysplasia
Achondroplasia	Short-limbed dwarfism, macrocephaly, frontal bossing
Rieger's syndrome	Iris dysplasia, midface hypoplasia, protruding umbilicus
Incontinentia pigmenti	Alopecia, pigmented macules, mental retardation
Seckel syndrome	Dwarfism, microcephaly, facial hypoplasia, low-set lobeless ears
Syndromes Demonstrating Supernumerary Teeth	
Condition	**Characteristics**
Cleidocranial dysplasia	Aplastic clavicles, frontal bossing, hypoplastic midface
Gardner's syndrome	Osteomas, epidermoid cysts, odontomas, intestional polyps
Down's syndrome	Brachycephaly, mental retardation, epicanthal folds
Crouzon's disease	Craniosynostosis, exophthalmos, hypoplastic midface
Sturge-Weber syndrome	Angiomatosis & calcification of leptomeninges, seizures, portwine nevi of the face
Oral-facial-digital syndrome I	Hypoplastic alar cartilage, cleft tongue, clinodactyly
Hallermann-Streiff syndrome	Dyscephaly, mandibular hypoplasia, hypotrichosis

***Syndromes manifesting both **Hyperdontia** and **Hypodontia** → Crouzon's disease – Down's syndrome – Oral-facial-digital syndrome I – Hallerman-Streiff syndrome

The **deciduous** dental formula of man is:

- $I\frac{1}{1}C\frac{1}{1}B\frac{1}{1}M\frac{2}{2} = 10 \times 2 = 20$
- $I\frac{2}{2}C\frac{1}{1}M\frac{2}{2} = 10 \times 2 = 20$
- $I\frac{2}{2}C\frac{1}{1}M\frac{3}{3} = 12 \times 2 = 24$
- $I\frac{2}{2}C\frac{1}{1}B\frac{2}{2}M\frac{3}{3} = 16 \times 2 = 32$

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- $I\frac{2}{2}\,C\frac{1}{1}\,M\frac{2}{2}$ **= 10 x 2 = 20**

$I\frac{2}{2}\,C\frac{1}{1}\,M\frac{2}{2}$ = 5 per quadrant = 10 per arch
= 5 per quadrant = 10 per arch **= 20 total teeth**

I = Incisors
C = Canines
M = Molars

Note: There are no **premolars** *(bicuspids)* in the deciduous dentition.

The **permanent** dental formula of man is:

- $I\frac{2}{2}\,C\frac{1}{1}\,B\frac{3}{3}\,M\frac{2}{2} = 16 \times 2 = 32$
- $I\frac{2}{2}\,C\frac{1}{1}\,B\frac{1}{1}\,M\frac{3}{3} = 14 \times 2 = 28$
- $I\frac{2}{2}\,C\frac{1}{1}\,B\frac{2}{2}\,M\frac{3}{3} = 16 \times 2 = 32$
- $I\frac{2}{2}\,C\frac{1}{1}\,M\frac{3}{3} = 12 \times 2 = 24$

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- **$I\frac{2}{2}\ C\frac{1}{1}\ B\frac{2}{2}\ M\frac{3}{3}$ = 16 x 2 = 32**

$I\frac{2}{2}\ C\frac{1}{1}\ B\frac{2}{2}\ M\frac{3}{3}$ = 8 per quadrant = 16 per arch
= 8 per quadrant = 16 per arch **= 32 total teeth**

I = Incisors

C = Canines

B = Bicuspids *(premolars)*

M = Molars

When do the **permanent** teeth begin to **calcify?**

- At birth
- One month
- Four months
- One year

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- **At birth** → the first molars begin to calcify at birth. Tooth buds generally initiated **after birth** are the premolars and second and third molars.

Approximate time when calcification begins for the primary and permanent dentitons

Permanent Dentition		
Maxillary	Central	3-4 months
	Lateral	10 months
	Canine	4-5 months
	First premolar	1 1/2 -1 3/4 years
	Second premolar	2-2 1/4 years
	First molar	At birth
	Second molar	2 1/2 - 3 years
	Third molar	7-9 years
Mandibular	Central	3-4 months
	Lateral	3-4 months
	Canine	4-5 months
	First premolar	1 3/4 - 2 years
	Second premolar	2 1/4 - 2 1/2 years
	First molar	At birth
	Second molar	2 1/2 - 3 years
	Third molar	8-10 years

Primary Dentition		
Maxillary	Central incisor	4 mos. in utero
	Lateral incisor	4 1/2 mos. in utero
	Canine	5 1/2 mos. in utero
	First molar	5 mos. in utero
	Second molar	6 mos. in utero
Mandibular	Central incisor	4 1/2 mos. in utero
	Lateral incisor	4 1/2 mos. in utero
	Canine	5 mos. in utero
	First molar	5 mos. in utero
	Second molar	6 mos. in utero

The **primary mandibular canines** are usually **exfoliated** when a child is about:

- 6 – 8 years old
- 7 – 9 years old
- 9 –12 years old
- 14 –16 years old

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• **9 – 12 years old**

Usual Exfoliation Age of Primary Teeth	
Maxillary Teeth	**Shed**
Central incisor	6-7 years
Lateral incisor	7-8 years
Canine *(Cuspid)*	10-12 years
First molar	9-11 years
Second molar	10-12 years
Mandibular Teeth	**Shed**
Second molar	10-12 years
First molar	9-11 years
Canine *(Cuspid)*	9-12 years
Lateral incisor	7-8 years
Central incisor	6-7 years

Primary teeth are exfoliated by the phenomenon called resorption of the primary root. The permanent tooth in its follicle attempts to force its way in to the position held by its predecessor. The pressure brought to bear against the primary root evidently causes resorption of the root, which continues until the primary crown has lost its anchorage, becomes loose, and is finally exfoliated.

If during a routine exam, you note that a permanent tooth is trying to erupt while the primary tooth is still firmly in place, the best treatment is to **extract the primary tooth** and allow the permanent tooth to erupt.

The **primary maxillary lateral incisor** typically erupts when a child is about:

- 8 months old
- 16 – 22 months old
- 13 – 19 months old
- 25 – 33 months old

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- **8 months old**

Primary Teeth Eruption Chart		
	Tooth	**Eruption**
Maxillary	Central incisor	7 1/2 mos.
	Lateral incisor	8 mos.
	Canine	16-20 mos.
	First molar	12-16 mos.
	Second molar	20-30 mos.
Mandibular	Central incisor	6 1/2 mos.
	Lateral incisor	7 mos.
	Canine	16-20 mos.
	First molar	12-16 mos.
	Second molar	20-30 mos.

***Eruption dates are variable. Some infants get them early, others do so late.

- When a tooth clinically erupts in the mouth, **one-half to two-thirds** of the root structure has usually developed.
- A primary tooth usually takes 1.5 to 2 months from the beginning of clinical eruption until it reaches the occlusal plane. Canines take the longest to erupt.
- **Calcification of the roots** is normally completed by the age of 3 or 4.
- **Calcification of the primary teeth** begins in the second trimester of pregnancy.

All of the following statements are true **except:**

- The primary teeth are **darker in color** than the permanent teeth
- The pulp cavities are **proportionately larger** in the primary teeth
- In general, the crowns of primary teeth are **more bulbous and constricted** than the permanent counterpart
- The crown surfaces of all primary teeth **are much smoother** than the permanent teeth *(in other words, there is less evidence of pits and grooves)*

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- **The primary teeth are darker in color than the permanent teeth**

***This is **false;** the primary teeth are lighter in color than the permanent teeth.

More comparisons of primary and permanent teeth:

- The crowns of the **primary anterior teeth** are wider mesiodistally and shorter incisocervically than their permanent counterparts
- The crowns of the **primary molars** are shorter and more narrow mesiodistally at the cervical third than the permanent molars
- The roots of the **primary anterior teeth** taper more rapidly than do those of the permanent anteriors
- The roots of the **primary molars** are longer and more slender than those of the permanent molars
- The enamel ends abruptly at the cervical line on **primary teeth,** rather than becoming thinner as on permanent teeth
- The buccal and lingual surfaces of **primary molars** are flatter above the crest of contour than on permanent molars
- **Primary molar** roots are more divergent *(relative to their crown width)* compared to their permanent counterparts to allow room for the developing permanent dentition

In most cases, the proper treatment for an **intruded primary anterior tooth** is:

- Repositioning
- Extraction
- Intermaxillary fixation
- Administer no treatment

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- **Administer no treatment**

Immediate attention should be given to soft-tissue damage. However, an x-ray of the area should be taken. Re-eruption usually occurs in 2 – 4 months. If the intruded incisor is contacting the permanent tooth bud, the primary tooth should be extracted.
Note: Damage to the succedaneous permanent tooth, including hypoplastic defects, dilaceration of the root, or arrest of tooth development, has been reported.

During the first six months after the injury, you may observe that there is **pulpal necrosis** which usually manifests as a gray or gray-black color change in the crown of the involved primary tooth at any time after the injury. The tooth can then be endodontically treated, **if necessary**, as long as the tooth is sound in the socket and no pathologic root resorption is evident. **Note:** If the tooth is **asymptomatic,** leave it alone.

Important: Repositioning displaced primary teeth that that are mobile **is not** recommended. Extraction is recommended due to the potential of aspiration in young children.

Discolored primary teeth that are symptom-free and show **no radiographic changes** are best treated by:

- No treatment
- Extirpation of the pulp tissue followed by the placement of ZOE paste in the root canal space
- Extraction
- Pulpotomy

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- **No treatment**

***They should be examined periodically by taking a radiograph.

Primary teeth will **often darken** *(become gray)* after injury. This is due to **pulp bleeding** and the diffusion of biliverdin into the dentinal tubules.

Facts about darkened teeth:

- 80% of primary incisors that are darkened due to injury are **asymptomatic.**
- Occasionally these teeth will lighten.
- 15% of these teeth will need to be removed in one year's time. This is **due to repeated trauma.**
- 85% of these teeth **will remain** until normal exfoliation.

As a result of trauma to the **primary dentition,** you should not expect to have problems with the successors unless the crown is not calcified. In this case, you will see **hypocalcification** in the tooth. This is **most common** with the mandibular incisors.

Note: Underdeveloped motor coordination is the **most common cause** of dental trauma in very young children.

Remember: Recently traumatized teeth **may give false negative** responses to pulp vitality tests. This impaired nerve conduction may be temporary or permanent, only time will tell.

A simple fracture of the crown of a tooth **involving little or no dentin** would be classified as:

- An Ellis Class I fracture
- An Ellis Class II fracture
- An Ellis Class III fracture
- An Ellis Class IV fracture

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• **An Ellis Class I fracture**

***Treatment includes **enameloplasty** and / or **bonding.**

Class II → extensive fracture of the crown involving considerable **dentin** but not the pulp. The standard of care in the past called for covering exposed dentin with calcium hydroxide or a glass component cement to seal out the oral flora. It is now recommended also to place a glass ionomer type restoration.

Class III → extensive fracture of the crown with a **pulpal exposure.** Treatment includes pulp therapy via pulp capping, pulpotomy, or pulpectomy. This is followed by restoration of the tooth.

Class IV → a fracture in which the **entire crown** has been lost. Treatment includes pulpectomy. Restoration of tooth involves the use of stainless steel crowns or celluloid crowns.

Note: Most often a **Class III fracture** in a primary tooth will lead to pulpal necrosis. A root canal using **ZOE paste** as a filling material is indicated. This paste will resorb with resorption of the roots of the primary tooth *(unlike gutta-percha).*

Other Ellis classifications:
- **Ellis Class V:** teeth avulsed or lost as a result of trauma
- **Ellis Class VI:** facture of the root with or without loss of crown structure
- **Ellis Class VII:** displacement of the tooth with or without loss of crown structure
- **Ellis Class VIII:** fracture of the crown en masse and its replacement
- **Ellis Class IX:** traumatic injuries to the primary dentition

Root fractures in which part of the tooth are **more likely** to undergo repair?

- Coronal
- Mid-root
- Apical third of the root

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• **Apical third of the root**

Root fractures occur in only 7% of dental injuries. **Horizontal** root fractures occur in anterior teeth, and are caused by direct trauma. **Vertical** root fractures usually occur in molars, and may be caused by clenching or trauma to the mandible. Vertical root fractures are more difficult to detect, and may not be found until extensive tooth destruction has occurred.

A **horizontal** root fracture is classified based on the location of the fracture in relation to the root apex. Horizontal root fractures may occur in: the apical third, middle third, or cervical third of the root.The prognosis worsens the further cervically the fracture has occurred. Tooth fractures are often not apparent during a clinical examination, and can usually only be diagnosed using appropriate radiographs. Radiographs with at least two views are required for making this diagnosis. Root fractures involving primary teeth are **relatively uncommon** because the more pliable alveolar bone allows displacement of the tooth. When it does occur, it should be treated in the same manner as that recommended for permanent teeth; **however, the prognosis is less favorable.** The pulp in a permanent tooth with a fractured root has a better chance to recover, since the fracture allows immediate decompression and circulation is more likely to be maintained.

Fractures in the **apical third** are often repaired without treatment. Many people recommend a relatively long stabilization period of 2 to 3 months for teeth with fractured roots. A **longer stabilization** seems to encourage the more favorable type of healing with calcified tissue. Heavy wires are **recommended** when one is stabilizing teeth with fractured roots. **Note:** Splinting **is not** recommended in the primary dentition. The occlusion should be **adjusted** so that the injured tooth is not further damaged during normal masticatory function. Follow-up radiographs and pulp tests should be made at frequent intervals during the 6-month period after the injury. These teeth often remain functional and vital. **Remember: Fractured maxillary anterior teeth** occur most often in children with Class II, Division I malocclusion *(maxillary anteriors are flared).*

What is the **most reliable method** to determine the pulp vitality in the case of a **recently** traumatized primary tooth?

- Radiograph
- Electric pulp test
- Thorough intraoral exam
- There is no reliable method

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• **There is no reliable method**

Often, traumatized teeth **will not** respond to vitality testing. Pulp vitality testing **is not** routinely performed in the primary dentition. This is because primary teeth do not respond to such tests reliably and because the test requires a relaxed and cooperative patient objectively reporting a reaction.

Congestion of blood within the pulp chamber a short time after injury can often be detected in the exam. Shining a bright light on the facial surface and holding the mirror to view the lingual will usually show a reddish hue **which is indicative of pulpal hyperemia.** If this color change is evident after several weeks, it is often indicative of a poor prognosis. Electric pulp tests are seldom reliable to determine pulp vitality if taken immediately after the injury. The thermal test is the **most reliable test,** especially in primary incisors. Failure of a tooth to respond to heat is **indicative of pulpal necrosis.**

Remember from **Endodontics** section: The chief cause of failure of replantation of permanent teeth is **external root resorption.**

A 15-year-old female has lived in a non-fluoridated area all of her life. Which of the following is **most likely to occur** in this young lady when she moves to a community where the drinking water naturally contains **6 ppm of fluoride?**

- 50% reduction in dental caries
- Moderate dental fluorosis
- An increase in the amount of fluoride stored in her bones
- Gastrointestinal problems

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• **An increase in the amount of fluoride stored in her bones**

Moderate fluorosis **will not occur** since by age 15 all of her dentition has undergone complete enamel calcification *(with the possible exception of the third molars).*

A 50% reduction in dental caries **is not** probable for the reason listed above as well.

Notes:

1. Water fluoridation and diet supplementation **may** affect tooth morphology, while self and professionally applied topical treatments will **not.**
2. The following types of fluoride are often added to water supplies:
   - Sodium fluoride
   - Hydrofluosilicic acid
   - Sodium silicofluoride
3. Up to a level of 1 ppm fluoride, there is an inverse relation between dental decay and fluoride concentration. As fluoride concentration increases beyond 1 ppm, there is an increased prevalence of fluorosis and no increase in the reduction of dental decay.
4. Of the 50 largest cities in the United States, 43 have community water fluoridation. Fluoridation reaches 62% of the population through public water supplies, more than144 million people.
5. Water fluoridation costs, on average, 72 cents per person per year in U.S. communities.

Fluoridation of community water has been credited with reducing tooth decay by 50% – 60% in the United States since World War II. More recent estimates of this effect show decay reduction at _____which reflects that even in communities that are not optimally fluoridated, people are receiving some benefits from other sources *(e.g., bottled beverages, toothpaste)*.

- 5% – 15%
- 10% – 25%
- 18% – 40%
- 45% – 55%

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• **18% – 40%**

Major mechanisms of action for fluoride in caries inhibition:

1. The topical effect of constant infusion of a low concentration of fluoride into the oral cavity is thought to increase **remineralization** of the enamel.
   - **Remineralization:** deposition of calcium, phosphate, and other ions into areas previously demineralized by caries or other causes.
   - Porous or slightly demineralized enamel has a **greater capacity** to acquire fluoride than adjacent sound enamel *(3-5x more!)*.
   - Greater capacity of demineralized enamel to absorb fluoride = decreased enamel dissolution.
2. Fluoride **inhibits glycolysis** in which sugar is converted to acid by bacteria. When this process is inhibited, the caries process is also inhibited.
3. During tooth development, fluoride interacts with hydroxyapatite to form fluorapatite. Fluorapatite is **less acid soluble** and therefore resistant to caries.

Fluoride mouth rinses **have been shown** to have the greatest effect on **newly erupted teeth,** making it essential to have rinsing continued into the teen years to protect both the second and third permanent molars. It seems that fluoride rinses are most beneficial to smooth tooth surfaces, although there are some benefits to pits and fissures as well.

Notes:

1. Fluorine, from which fluoride is derived, is the 13th most abundant element and is released into the environment naturally in both water and air.
2. Fluoride is naturally present in all water. Community water fluoridation is the addition of fluoride to adjust the natural fluoride concentration of a community's water supply to the level recommended for optimal dental health, approximately 1.0 ppm *(parts per million)*. For warmer or colder climates, the amount can be adjusted from 0.6 to 1.2 ppm.

Which of the following **fluoride therapies** should be recommended to a 13-year-old child who is prone to decay and lives in a community where the water is fluoridated at an appropriate level?

- Professionally applied fluoride every six months
- Fluoride toothpaste
- Dietary fluoride supplements
- A low concentration fluoride mouth rinse
- A high concentration fluoride mouth rinse

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- **Professionally applied fluoride every six months**
- **Fluoride toothpaste**
- **A low concentration fluoride mouth rinse**

Fluoride supplements would be **contraindicated** since the community water is fluoridated at an appropriate level.

High concentration fluoride mouth rinses **must be** professionally prescribed and require a prescription. These mouth rinses have been shown to reduce root surface hypersensitivity. **OTC mouth rinses:** Act, Fluoriguard, Prevident Rinse, contain 0.2 - 0.5% NaF.

Fluoride in toothpastes:
- Stannous fluoride *(SnF_2)*
- Sodium monofluorophosphate *($NaPO_3F$)*
- Sodium fluoride *(NaF)*
- Sodium fluoride and Calcium Phosphate *(NaF / $CaPO_4$)*

***For **children:** recommend a **pea-sized** amount of toothpaste to avoid fluorosis

Fluoride **concentration** in all major dentifrices in USA is **0.1%** *(1,000 ppm)*:
equivalent to:
- 0.22% NaF
- 0.76% Na MFP
- 0.4% SnF_2

It is best to use which of the following for a topical fluoride application in the office for a patient that has porcelain, glass ionomer or composite restorations?

- Acidulated fluoride *(APF)* solutions
- Stannous fluoride
- Neutral sodium fluoride

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- **Neutral sodium fluoride**

Important: Acidulated fluoride *(APF)* solutions and stannous fluoride remove the glaze from porcelain, glass ionomer, and composite restorations.

Topical fluoride applications → applied in office twice a year:

- **2% sodium fluoride (NaF)** → advantages are neutral pH, more acceptable taste than stannous fluoride and **no** adverse effect on restorative materials.
- **8% stannous fluoride (SnF_2)** → is a nonstable solution, it tastes bad, stains silicate restorations and restoration margins.
- **1.23% acidulated phosphate fluoride (APF)** → advantages are a more acceptable taste than stannous fluoride and can be applied to both arches simultaneously. **May cause etching of porcelain and some composite restorations.**

Topical fluoride *(along with occlusal sealants)* is the primary preventive agent during adolescence *(past the age of 12)* because the entire dentition except for the third molars normally erupts by age 13. Therefore, fluoride tablets may not be as beneficial.

Remember: Caries activity is directly proportional to the **consistency** of fermentable carbohydrates ingested, the **frequency** of ingesting fermentable carbohydrates and the **oral retention** of fermentable carbohydrates ingested.

Match up the **fluoride concentration** with the appropriate **type of professionally applied topical fluoride.**

Topical Fluoride	Concentration
Sodium Fluoride (NaF)	8.0%
Acidulated Phosphate Fluoride	2.0%
Stannous Fluoride (SnF2)	1.23%

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- **Sodium Fluoride → 2.0 %**
- **Acidulated Phosphate Fluoride → 1.23 %**
- **Stannous Fluoride → 8.0 %**

Fluorapatite is the **most stable** reaction product of a topical application of fluoride.

Fluoride Concentrations Found in Other Compounds			
Type of Fluoride	Dentifrice	Brush-On-Gel *(home use)*	Oral Rinse
Sodium Fluoride (Naf)	.22%	1.1%	0.05% daily-swallow 0.2% weekly-expectorate
Sodium Monofluorophosphate ($NaPO_3F$)	0.76%	Not available	Not available
Acidulated Phosphate Fluoride (APF)	Not available	0.5%	0.04% daily-swallow or expectorate
Stannous Fluoride (SnF_2)	.4%	0.4%	0.1% daily

All of the following are factors to consider when deciding whether a **fluoride supplement** should be prescribed to a child **except:**

- The amount of fluoride in the child's drinking water
- The age of the child
- The type of topical fluoride applied professionally in the office
- How responsible is the person who will be administering the supplement, whether it be the patient or the patient's parents

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- **The type of topical fluoride applied professionally in the office**

Dietary Fluoride Supplementation Schedule			
Age	<0.3 ppm F	0.3-0.6 ppm F	>0.6 ppm F
Birth-6 months	0	0	0
6 months-3 years	0.25 mg	0	0
3-6 years	0.50 mg	0.25 mg	0
6 years up to at least 16 years	1.00 mg	0.50 mg	0

Remember: It is **not necessary** to prescribe fluoride supplements to a child who is consuming water which has been optimally fluoridated.

Important: Prenatal fluoride supplements **are not approved** by the FDA and are not recommended. However, prenatal flouride does not cross the placental barrier. No studies to date support the administration of prenatal fluorides to protect the primary dentition against caries.

Amelogenesis imperfecta *(AI)* is a relatively rare group of inherited disorders characterized by abnormal enamel formation. **Type I** amelogenesis imperfecta is also known as:

- Hypoplastic Amelogenesis Imperfecta
- Hypocalcified Amelogenesis Imperfecta
- Hypomaturation Amelogenesis Imperfecta

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• **Hypoplastic Amelogenesis Imperfecta**

Amelogenesis imperfecta is an inherited condition, which is transmitted as a dominant trait and causes the **enamel of teeth to be soft and thin.** The teeth are easily damaged and susceptible to decay. The pulpal outline appears to be normal and the root morphology is normal. The treatment of amelogenesis imperfecta depends on its severity and the demands of esthetic improvement. Because the **dentin is normal**, the teeth can be prepared for full crowns.

1. **Type I** *(Hypoplastic)* → inadequate deposition of enamel matrix. Whatever is deposited calcifies normally. Teeth have thin enamel that may be pitted. Color varies from normal to opaque white – yellow - brown.
2. **Type II** *(Hypomaturation)* → adequate deposition of enamel, but the enamel crystal does not mature normally. The result is soft pigmented enamel that is rough. Color varies from creamy opaque to marked yellow-brown. Dentin sensitivity and open bite are common.
3. **Type III** *(Hypocalcified)* → Inadequate mineralization. The result is a soft rough enamel surface that get lost a few years after eruption. Color varies from opaque white to yellow-brown. Dentin sensitivity and open bite are common.

Remember: Dentinogenesis imperfecta *(DI)* is also an inherited condition, which is transmitted as a dominant trait and causes the dentin of teeth to be undermineralized. It can affect both the deciduous and permanent dentition. The crowns are bulbous. The teeth may appear gray or brown with opalescent dentin that overgrows and obliterates the pulp cavity. The teeth have short roots and tend to wear rapidly. There are **three types:**

1. **Type I** → associated with osteogenesis imperfecta. Child may also have **blue sclera**, fragile bones and hearing loss.
2. **Type II** → most common, DI without osteogenesis imperfecta.
3. **Type III** *(Brandywine type)* → characterized by multiple periapical radiolucencies, and large pulp chambers may lead to multiple pulp exposures.

Concrescence involves fusion of:

- Enamel only
- Dentin and enamel
- Cementum only
- Dentin and cementum

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• **Cementum only**

Concresence is actually a form of fusion, it involves the union of the roots of two or more normal teeth caused by confluence of their cemental surfaces. It is thought to arise as a result of traumatic injury.

Gemination is a process in which a single tooth germ splits or shows an attempt at splitting to form two completely or partially separated crowns. This process results in incomplete formation of two teeth. The two teeth share the same root canal. It most frequently occurs in the incisor region. **Twinning** is more complete than gemination and results in the formation of two separate teeth from one tooth bud *(one extra tooth in the arch)*.

Fusion of teeth is a condition produced when two tooth buds are joined together during development and appear as a macrodont *(a single large crown)*. It may involve the entire length of two teeth *(enamel, dentin, and cementum)* or just the root *(dentin and cementum)*.This condition is usually seen in the incisor area. The single crown may have two roots or a grooved root, but there are usually two root canals. **Note:** It is often difficult to distinguish between gemination and fusion.

Remember: The fusion, or gemination of teeth, occurs during the initiation and proliferation stages of tooth development.

The term **Dens in dente** *(also called dens invaginatus)* means a **"tooth within a tooth"** and is caused by an invagination of all layers of the enamel organ into the dental papilla. It **most frequently** involves the maxillary lateral incisor. The pulp is usually exposed and is therefore necrotic or inflamed. The pulp canals of these teeth should be cleaned and **filled. Note:** This invagination can range in severity from an accentuated lingual pit to a "Dens in dente".

A defect in tooth enamel that results in less quantity of enamel than normal is referred to as:

- Enamel hypoplasia
- Enamel hypocalcification
- Fluorosis
- Enamel pearl

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• **Enamel hypoplasia**

Enamel hypoplasia is a developmental dental defect in which the enamel of the teeth is hard in context, but thin and deficient in amount. It is caused by **defective enamel matrix formation** with a deficiency in the cementing substance.

Clinical features:

- Pits, grooves, lines or larger areas of missing enamel surface
- Reduction in enamel thickness
- Possible occlusal distortion, esthetic problems, sensitivity
- Yellowish or brownish discoloration
- May be localized or present on numerous teeth and all or part of the surfaces of each affected tooth may be involved

This condition, which effects both the primary and permanent teeth can be transmitted genetically or be caused by environmental factors, as with vitamin deficiency, **fluorosis** or metabolic disturbances during the prenatal period. **It is a common dental sequelae in a child with a history of generalized growth failure in the first six months of life.** Hypoplastic areas on teeth are seen if a child has illnesses in early childhood. **See picture #9 in booklet.**

Note: Hypoplastic enamel, which is a dental manifestation of hypoparathyroidism, can be prevented by early treatment with vitamin D.

Enamel hypocalcification is a condition in which the enamel is normal but the enamel quality is poor. It is caused by the defective maturation of ameloblasts *(defect in mineralization of the formed matrix)*. The teeth are chalky in consistency, the surfaces wear down rapidly and a yellow to brown stain appears as the underlying dentin is exposed. This condition affects both the primary and permanent teeth.

For the typical child, bite-wing x-rays should be taken:

- When the parents think it is time to take them
- At the first dental visit
- When the proximal surfaces cannot be visualized and clinically examined
- After the second dental visit when the child is more relaxed

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• **When the proximal surfaces cannot be visualized and clinically examined**

***In other words, after the **establishment of contacts** on the posterior teeth.

A child should have his / her first pediatric visit by their first birthday. Following that, if the child's teeth are spaced far apart and there is no clinical evidence of decay, bite-wings are not needed until the establishment of contacts on the posterior teeth. At age six a child should have their first panoramic x-ray in order to get all vital information on developing teeth, roots and any possible malocclusion. X-rays for growth and development depend on the patient's stage of tooth eruption. The frequency of radiographs should depend on the child's risk for decay. Situations that make a child at higher risk for decay include lack of fluoride in the drinking water, high sugar diet, history of cavities, poor oral hygiene, and many others.

Notes:

1. The nice thing about panoramic x-rays is that they are taken **without placement** of the film in the mouth so it does not alarm the nervous child.
2. Children are often **"entertained"** by the panoramic unit.
3. The **drawback** of a panorex is that there is a **loss of image detail** *(it is hard to diagnose early carious lesions)*. Bite-wing x-rays **are required** for the diagnosis of carious lesions.

All of the following statements are true **except:**

- The occlusal anatomy of primary teeth is not as defined as that of permanent teeth **therefore** amalgam preps can be more conservative
- Enamel and dentin are thicker in primary teeth, **therefore** amalgam preps are deeper
- The pulpal horns of primary teeth are longer and pointed, **therefore** amalgam preps must be conservative to avoid a pulpal exposure
- Primary molars have an exaggerated cervical bulge that makes matrix adaption much more difficult

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- **Enamel and dentin are thicker in primary teeth, therefore amalgam preps are deeper**

***This is **false;** the enamel and dentin are **thinner** in primary teeth, **therefore** amalgam preps are **shallower.** The thickness of coronal dentin in primary teeth is about **one-half** that of permanent teeth.

Other important morphologic considerations of primary teeth include:

- Primary molars have an exaggerated cervical constriction **which requires special care in the formation of the gingival floor in Class II preps.**
- Enamel rods in the gingival third of primary teeth extend occlusally from the DEJ, **eliminating the need in Class II preps for the gingival bevel** which is always required when preparing Class II preps on permanent teeth.

Important: When preparing a Class II amalgam prep on a primary tooth, there are several other recommendations for the proximal box preparation:

- The proximal box should be broader at the cervical than at the occlusal aspect.
- The buccal, lingual, and gingival walls should all break contact with the adjacent tooth, just enough to allow the tip of an explorer to pass.
- The buccal and lingual walls should create a 90-degree angle with the enamel.

The pH of **acidulated phosphate fluoride gels** is between:

- 3 and 4
- 5 and 6
- 7 and 8
- 9 and 10

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• **3 and 4**

Important:
*** The **pH of APF** is approximately 3.0 to 3.5 *(acidic)*
*** The **pH of NaF** is approximately 9.2 *(basic)*
*** The **pH of SnF_2** is approximately 2.1 to 2.3 *(acidic)*

Note: Acidulated fluoride *(APF)* solutions and stannous fluoride *(SnF_2)* should not be used on patients with porcelain, glass ionomer, and composite restorations. They have been shown to remove the glaze from the surface of these restorations. Neutral sodium fluoride *(NaF)* is best to use if these restorations are present. **Also,** acidulated fluorides should be **avoided** on implant patients → **may corrode the surface of titanium implants.**

When painting fluoride on, it is very important to **isolate the teeth** with cotton rolls. When using fluoride trays, cotton rolls may be placed in the premolar areas to increase patient comfort and to help keep the fluoride in place. Patients are asked not to brush, rinse, eat or drink for 30 minutes after a fluoride treatment so that the fluoride is left undisturbed and is able to continue reacting with the hydroxyapatite for some time after the initial application. Fluoride treatments should be applied for four minutes, although there are now some one-minute products that are being marketed.

Emergency treatment in the dental office for a child who has accidentally ingested a large amount of fluoride should include all of the following **except:**

- Induce vomiting mechanically or with the help of Ipecac syrup
- Call 911
- Have patient drink a large quantity of milk
- Crush ammonia vaporole under the patient's nose to keep the patient awake and conscious

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- **Crush ammonia vaporole under the patient's nose to keep the patient awake and conscious**

In **acute fluoride toxicity,** the goal is to minimize the amount of fluoride absorbed. Therefore, syrup of ipecac is administered to induce vomiting. Calcium-binding products, such as milk or milk of magnesia, decrease the acidity of the stomach, forming insoluble complexes with the fluoride and thereby decrease its absorption. **Note:** EMS *should be activated (911).*

In **acute fluoride toxicity,** symptoms may appear within 30 minutes of ingestion and persist for up to 24 hours. Patients may experience some nausea, vomiting, diarrhea, and abdominal cramping. This may be due to the fact that 90-95% of ingested fluoride is absorbed through the stomach and small intestines. Fluorides are primarily eliminated from the body by way of the kidneys. However, the **fluoride** that does remain in the body is **found mostly** in skeletal tissue.

In **acute fluoride poisoning** *(which is rare)*, the most common causes of death are cardiac failure and respiratory paralysis. Fluoride toxicity shows up in the bones as osteosclerosis. The ingestion of 5 gm of fluoride or greater in an adult results in death within a couple of hours if the person does not receive medical attention. In a child, ingestion of a single dose greater than 400 mg results in death in about 3 hours. Doses of 100 - 300 mg in children result in nausea and diarrhea.

Note: If a **six-year old child** were receiving fluoridated water in the amount of 3 ppm, the result would most likely be fluorosis but **not** systemic toxicity. On the other hand, if a child in the same age range *(6-7)* were receiving 8 ppm of fluoridated water, there would be a good chance of **systemic toxicity** and moderate to severe fluorosis occurring.

All of the following statements concerning dental fluorosis are true **except:**

- It is a diffuse symmetric hypomineralization disorder of ameloblasts
- It is reversible
- It only occurs with exposure to fluoride when the enamel is developing *(calcification period)*
- It is a toxic manifestation of chronic *(low-dose, long-term)* fluoride intake

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- **It is reversible**

***This is **false;** it is **irreversible.**

Dental fluorosis *(fluoride mottling)* is a condition of enamel hypoplasia characterized by white chalky spots or brown staining and pitting of primary and permanent teeth due to an increased level of fluoride affecting enamel matrix formation and calcification by impairment of ameloblastic function.

The severity of the mottling increases with an increasing amount of fluoride in the drinking water. Thus, there is little mottling at a level below 0.9 to 1.0 ppm of fluoride in the drinking water, whereas children living in temperate zones where the water supply contains a higher content of fluoride are most often affected.

Note: If a child lives in a temperate zone most of his / her childhood *(through age 9 or 10)*, **all permanent teeth** will most likely be affected. **Remember:** The third molars begin calcification between 7 and 9 years of age. If the child moves before this age, the third molars might not be affected.

Important:
- Dental fluorosis **is not caused** by the repeated **topical application of fluoride.**
- The **systemic** distribution of fluoride may affect tooth morphology. The systemic distribution of fluoride is accomplished by fluoride in the water, fluoride tablets, and vitamins that contain fluoride.

What is the most **effective method** of reducing the dental caries problem in the general population?

- School water fluoridation
- Fluoridation of the communal water supply
- Fluoride rinses at home
- Frequent dental visits
- Patient education

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• **Fluoridation of the communal water supply**

The **optimal concentration** in the communal water supply varies with mean annual temperature. In most states, it is 1 ppm. **Fluoride supplements** are recommended if the water fluoride content is less than 0.7 ppm. These supplements are normally not indicated beyond 13 years of age.

The **school water fluoridation** optimal concentration is 4.5 times that of city water supplies because of **less water consumption** at school.

Other facts concerning fluoride:

- It is deposited in calcified tissues *(skeletal)*. It normally accumulates slowly in bones as a person ages.
- Proximal tooth surfaces derive the **greatest benefit** from fluoridation.
- It is **excreted** by the kidney.
- Dental fluorosis can occur in **permanent** and **deciduous** teeth.
- The U.S. Public Health Department sets the optimal fluoride level at **0.7 to 1.2 ppm** for public water.
- The cariostatic effect of fluoride is produced during the **calcification** stage of tooth development.
- It **converts** hydroxyapatite to fluorapatite.

Key point to remember: The most desirable form of fluoride in enamel for caries prevention is **fluorhydroxyapatite** and the most efficient means of forming this reaction product occurs with prolonged exposure of enamel to low concentrations of fluoride.

When operative or surgical procedures are performed on the **mandibular** primary or permanent teeth, which nerve **must be** blocked?

- Posterior superior alveolar nerve
- Lingual nerve
- Inferior alveolar nerve
- Long buccal nerve

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• **Inferior alveolar nerve**

In the mandibular arch, the only guaranteed way to accomplish profound pulpal anesthesia is to perform an inferior alveolar nerve block. Primary incisors, however, can be anesthetized using supraperiostial injections – which anesthetizes branches of the incisive nerve. **Note:** Local infiltration **can be** used for anesthetizing maxillary primary teeth. Adequate diffusion of the local anesthetic **readily occurs** in children because their bones are less dense than those of adults.

Important: Remember that the mandibular foramen in a child is located slightly below the plane of occlusion. In addition, the foramen is located more anterior than in adults – due to the narrow anteroposterior width of a child's mandible. **Note:** An imperfect injection technique is the most common cause of problems with getting a child patient numb.

Remember: Young children don't always understand what **"numb lip"** means when you ask them this following a mandibular block. The **best indicator** of a profound block would be to probe the labial-attached gingiva between the lateral incisor and canine with an explorer. If this is done **without a reaction** from the child, he / she is "numb."

Notes:

1. The maximum dose for lidocaine injection in children is 4.5 mg / kg per appointment. For example: a 35 lb child should never receive more than 2 carpules of 2% lidocaine during a dental appointment.
2. Bupivicaine *(Marcaine)* is an amide local anesthetic with a high toxic potential, and should not be used in children.
3. Remember to warn the child not to bite the "numb" cheek or lips. Give the warning during the dental appointment as well at the end of the appointment.

When placing a Class II amalgam in a primary tooth, the **width of the isthmus** should be:

- About one-half the intercuspal width
- About one-third the intercuspal width
- About two-thirds the intercuspal width
- About three-quarters the intercuspal width

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- **About one-third the intercuspal width**

***Important:** If fracture of the amalgam occurs, it is **most likely** to occur here.

Other basic principles in the preparation of cavities in primary teeth include:

- The **Class I** and **II** preparations should include those areas that have caries and those areas that retain plaque and are potential carious areas *(pits and fissures).* **Note:** This "extension for prevention" is **only when restoring with amalgam.** It is not necessary to "extend for prevention" when restoring with composite resin or resin modified glass ionomer, it is possible to seal the remaining pit and fissures.
- **Flat pulpal floor**
- **Beveled** *(rounded)* **axio-pulpal line angle.** This will help reduce stress in the amalgam and provide greater bulk of material in this area.
- **Rounded angles** throughout the preparation. This will result in less concentration of stresses and will allow more complete condensation of the amalgam material into the extremities of the preparation.
- In **Class II** preparations, the facial and lingual walls of the proximal box should be carried to self-cleansing areas and should be parallel to the external surfaces and converge slightly.
- The gingival margin **need not be beveled** in Class II preps. The enamel rods in this area incline occlusally.
- In **Class II** preparations, the gingival floor is **not ideal in most cases** as the preparation gets deeper in this area. This is **due to the cervical constriction** found in this area on primary molars.

When preparing a primary tooth for a **stainless steel crown**, the cusps are reduced:

- Approximately 1 to 1.5 mm
- Approximately 5 to 6.5 mm
- Approximately 10 mm
- Approximately 15 mm

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- **Approximately 1 to 1.5 mm**

***This **reduction** will allow clearance with the opposing teeth and prevents traumatic occlusion after placement of the stainless steel crown.

Posterior stainless steel crowns on primary teeth are a fast, predictable, durable, and relatively inexpensive restorative technique. Primary teeth have a limited lifespan compared to the permanent dentition; as a result, a restoration needs to last only until exfoliation. Because primary teeth are smaller than permanent teeth, a given amount of decay causes the tooth structure to become thinner and less stable than it would be in a larger permanent tooth. The larger pulp space of primary teeth limits the depth of amalgam preparations; these factors result in less stable Class II amalgam restorations among primary molars. Premature loss of a Class II amalgam can lead to the mesial migration of posterior teeth with a corresponding loss of arch length. In contrast, stainless steel crowns encompass the entire tooth; this full coverage aspect also offers a significant degree of protection from recurrent decay.

Two commonly **used types** of stainless steel crowns:
1. Pretrimmed crowns
2. Precontoured crowns

Preparing the tooth for a stainless steel crown:

- Reduce proximal surfaces *(mesial and distal)* → vertical reductions are made and carried gingivally to the extent that contact with the adjacent tooth is broken.
- The cusps are reduced *(approximately 1 to 1.5 mm)*.
- Removal of all sharp line angles. **Note:** It is usually not necessary to reduce the buccal or lingual surfaces. This will aid in retention of the crown *(undercut area)*, however, it may be necessary to reduce the **distinct buccal bulge,** particularly on the primary first molar.
- **Fitting the crown** → in festooning and trimming the crown, greater length is necessary in the region of the mesiofacial bulge on a primary first molar.

All of the following statements are true **except:**

- Dental decay in primary teeth is an infectious process that can be very painful, spread, and affect the development of the adult teeth
- Dental decay in primary teeth most often means there will be dental decay in the adult teeth
- Primary teeth are slightly more opaque on x-ray film than permanent teeth because of a lower inorganic content
- Dental decay in primary teeth tends to progress more rapidly from initial surface demineralization to involvement of the dentin
- The enamel layer of primary teeth is thinner in all dimensions as compared to permanent teeth

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- **Primary teeth are slightly more opaque on X-ray film than permanent teeth because of a lower inorganic content**

***This is **false;** primary teeth are slightly less opaque on x-ray film than permanent teeth because of a higher inorganic content. **Remember:** There must be 30–60% loss in mineralization before caries is radiographically evident with standard D–and E-speed intraoral films. Therefore, the clinical progress of a carious lesion is advanced, sometimes significantly, compared with its radiographic progress.

Decay in primary teeth needs to be treated the **same way** as it is treated in permanent teeth. To just **"watch"** this decay may result in loss of tooth structure and eventual loss of the primary tooth with resultant loss in arch length.

Notes:

1. **Amalgam** is still used to some degree to restore primary teeth. The most important modification in its use is in the **cavity preparation.** Properly contoured restorations are important to **maintain** proximal contacts and the length of the dental arch.
2. Composite resin and resin modified glass ionomers are now commonly used as well to restore primary teeth.

Remember:

- Caries is an **infectious disease.** It needs to be eliminated by removing the caries and restoring or extracting the tooth. **Note:** To ensure **arch integrity,** restoring the involved tooth, if possible, is preferred.
- For a **clinically caries-free child,** the first bite-wing x-ray should be taken **when the spaces** between the posterior teeth have closed.

PEDIATRIC DENTISTRY Behav Mngmt

Which of the following is the **most important** technique of behavioral management in the pediatric dental patient?

- Tell-show-do
- Voice control
- Positive reinforcement
- Distraction
- Non-verbal communication

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- **Tell-show-do**

- This technique is the **backbone** of the educational phase of developing an accepting, relaxed child dental patient.
- In this technique, you **tell** the child what is going to happen, you **show** the child what is going to happen, and then you **perform** the actual procedure in the mouth.
- This technique works **especially well** when treating a child with a **different** cultural background.

Important: The clinical examination of the **infant and toddler** should be accomplished with the parents' assistance in a **non-threatening environment.** Most often, it is **neither necessary nor recommended** that the dental chair be used. The parent and dentist sit facing each other in a knee-to-knee position, supporting the child with the head cradled on the dentist's lap.

Remember:

- **Aggressive behavior** in the dental office is **usually** a fear reaction.
- The **most realistic approach** to managing a **difficult child** it the dental office is to attempt to **recondition** the child through techniques of applied psychology.

When treating a child who is obviously **afraid**, the dentist should:

- Use **restraint**
- Use the **hand-over-mouth technique** *(HOME)*
- Permit the child to **express his fear**
- **Avoid all reference** to the child's fear

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• **Permit the child to express his fear** → identify the fear

All behavioral patterns are motivated by **anger** and **fear.** The crying child is **NOT** an abnormal child. Anger is **easier to treat** than fear. **Fear** is most likely to be exhibited by a young child on his first visit to the dentist. This is related to the anxiety over being separated from a parent. **The parent**, *not the dentist*, **has the greatest influence** on the child's reaction at this initial visit.

Techniques to control certain types of children:

• **The angry child:**
  - **Separate** the **parent** and the **child**
  - Place the child in the chair **abruptly and be firm**
  - Use the **"hand-over-mouth"** technique → get the **parent's permission !!!**
  - **Display authority** and **command respect of the child** by continuing with treatment if he / she is uncooperative
  - **Comfort parent** at the end of the visit
  - **Compliment child** at the end of the visit

• **The fearful child:**
  - Have the **parent stand quietly behind the chair**
  - **Dentist must be consistent** in tonal quality
  - Permit the child to express his fears → identify the fear
  - Change the child's focus **off** fear
  - Lastly, **sedation**

The process of shaping a patient's behavior through appropriately timed feedback is called:

- Tell-show-do
- Voice control
- Positive reinforcement
- Distraction
- Nonverbal communication

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• Positive reinforcement

American Academy of Pediatric Dentistry's Standard for Behavior Management				
Communicative Management	**Description**	**Objectives**	**Indications**	**Contraindications**
Tell-show-do	Explanations tailored to cognitive level, followed by demonstration, followed by actual procedure	- Allay fears shape patient's response - Give expectations of behavior	All patient's who can communicate regardless of method of communication	None
Voice control	Modulation on voice volume, tone, or pace to influence and direct patient's behavior	- Gain patient's attention - Avert negative or avoidance behaviors - Establish authority	Uncooperative or inattentive but communicative child	Children who are unable to understand due to age disability, medication, or emotional immaturity
Positive reinforcement	Process of shaping patient's behavior through appropriately timed feedback *(e.g., praise, facial expression)*	Reinforce desired behavior	Any patient	None
Distraction	Diverting patient's attention from perceived unpleasant procedure	Decrease likelihood of unpleasant perception or threshold	Any patient	None
Nonverbal communication	Conveying reinforcement and guiding behavior through contact, posture, and facial expressions	- Enhance effectiveness of other communicative management techniques - Gain or maintain patient's attention and compliance	Any patient	None

All of the following procedures have **proved beneficial** in treating a mentally retarded child **except:**

- Speak slowly and in very simple terms
- Listen carefully to the patient
- Schedule long appointments
- Ask the patient if there are any questions about anything you will be doing

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- **Schedule long appointments**

***This is **false;** you should keep appointments short.

In addition the following procedures are also helpful when treating mentally retarded children:

- **Give a tour to the patient** before attempting to do any treatment. Introduce the patient to the office personnel.
- **Give only one instruction at a time.** Reward the patient with compliments after the successful completion of the procedure.
- **Schedule the patient early in the day.** The staff, the dentist, and the patient are less fatigued at this time.

In treating mentally retarded children, the following is usually found:

- They can be controlled in the same ways as normal children.
- They respond similarly to normal children of the **same mental age.**
- They **respond inconsistently,** have **short attention spans**, and are **restless** and **hyperactive** when undergoing dental care.

Important: The dentist should assess the degree of mental retardation **by consulting** the patient's physician before starting dental treatment.

All of the following statements concerning **recurrent aphthous ulcers** *(canker sores)* are true **except:**

- They occur in women more than men
- They may occur at any age, but usually first appear between the ages of 10 and 40
- The cause is a coxsackie virus
- They appear to be associated with stress
- They usually appear on nonkeratinized oral mucosa including the inner surface of the cheeks and lips, tongue, soft palate and the base of the gingivae

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• **The cause is a coxsackie virus**

***This is **false;** the cause is **unknown,** however evidence supports they are related to the focal immune dysfunction where T lymphocytes play a major role.

These lesions appear as painful white or yellow ulcers surrounded by a bright red area. Lay persons refer to aphthous ulcers as **"canker sores".** They can be triggered by **stress**, dietary deficiencies *(especially iron, folic acid, or vitamin B12)*, menstrual periods, hormonal changes, food allergies, and similar situations.

They usually begin with a **tingling or burning sensation,** followed by a red spot or bump that ulcerates. Pain spontaneously decreases in 7 to 10 days, with complete healing in 1 to 3 weeks.

Important: Recurrent aphthous ulcers and lesions of intraoral herpes are distinguished largely on their location. Recurrent aphthous ulcers occur primarily on mobile mucosa while lesions of intraoral herpes occur on tissue bound to periosteum.

Three Classifications

1. **Recurrent aphthous minor** *((0.5 mm–10mm in diameter)* are common, last over 2 weeks
2. **Recurrent aphthous major** *(10–20 mm in diameter)* are much less common, last over 2 weeks and heal with scarring
3. **Recurrent herpetiform:** multiple, small, diffuse, painful, superficial ulcers

***Patients with frequent recurrences should be screened for **diabetes mellitus** or **Behcet's syndrome.**

Topical steroids have been suggested for the relief of symptoms as follows:

• **Triamcinolone** *(Kenalog)* **in Orabase 0.1%**
  Disp: 5 gm tube
  Sig: Coat the lesion with a thin film after each meal and at bedtime

The **management** of a child who must **undergo dental extractions** is based on which of the following factors?

- The **age** and **maturity** of the child
- The **past medical** and **dental experiences** that might influence the behavior of the child
- The **physical status** of the child
- The **length** of time and **amount** of manipulation necessary to accomplish the surgery
- All of the above

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• **All of the above**

The age and maturity of the child **often determine** the type of anesthesia best suited for the intended procedure. Children below the age of reason generally are best managed under general anesthesia, since a slight amount of discomfort is always associated with the administration of a local anesthetic. It is very important to have **total anesthesia** before starting the procedure. Use both buccal and palatal infiltration on maxillary teeth and block anesthesia on mandibular teeth with infiltration, if necessary.

The very young patient is **best managed** under general anesthesia, usually of the inhalation type or in combination with small doses of intravenous barbiturates. The **most common** premedication prior to general anesthesia is **versed.**

Note: Premedication with a **barbiturate** may cause paradoxical excitement in a young child.

Remember: After extracting a tooth on a child patient, the **biggest post-operative concern** is the prevention of lip biting.

All of the following instances, **except one**, may make the use of a rubber dam impractical. **Which is the exception?**

- The presence of **fixed** orthodontic appliances
- A patient with **congested nasal passages** or other **nasal obstruction**
- A very **nervous** or **anxious** patient
- A recently erupted tooth that **will not** retain a clamp

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• **A very nervous or anxious patient**

One of the **main advantages** of using a rubber dam is that it can **aid** in the management of the child. It seems to quiet and calm the patient because the dam acts as a separation or barrier, both physically and psychologically.

Other advantages include:

1. Better **access** and **visualization**
2. Control of **saliva** and **moisture** in the operating field
3. **Decreased** operating time
4. Provides **protection** from aspiration or swallowing of foreign bodies
5. The child becomes primarily a nasal breather when the rubber dam is in place. This then **enhances the effects** of nitrous oxide if applicable.

Cretinism is a deficiency disease, caused by the congenital absence of:

- Insulin
- Thyroxine
- Calcitonin
- Epinephrine

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• **Thyroxine**

***Thyroxine is a hormone secreted by the thyroid gland.

Cretinism is severe hypothyroidism in a child and is characterized by defective mental and physical development. Cretins have dwarfed bodies, with curvature of the spine and a pendulous abdomen. Their limbs are distorted, their features are coarse, and their hair is harsh and scanty. Severe mental retardation is caused by the improper development of the CNS. **Note:** If this condition is recognized early, it can be markedly improved with the use of thyroid hormones.

Dental findings in a child with cretinism *(hypothyroidism)* include an **underdeveloped mandible** with an overdeveloped maxilla, **enlarged tongue** which may lead to malocclusion, delayed eruption of teeth, and deciduous teeth being retained longer. An anterior open bite is common and flaring of the anterior teeth often occurs. This may be related to the abnormal size of the tongue.

Additional **intraoral** findings include: thickened lips due to glycosaminoglycan deposits, unerupted **yet fully developed** permanent dentition.

Remember: Severe hypothyroidism in adults is called **myxedema.**

All of the following statements are **true** concerning cystic fibrosis **except:**

- Cystic fibrosis is an inherited disease of the exocrine glands, affecting approximately 30,000 children and adults in the United States
- Currently, most individuals with cystic fibrosis die in middle age – many in their 40s and 50s from lung failure
- Cystic fibrosis causes the body to produce an abnormally thick, sticky mucus, due to a faulty transport of sodium and chloride within cells lining organs such as the lungs and pancreas
- Cystic fibrosis has a variety of symptoms. The most common of which are: a very salty tasting skin, persistent coughing, wheezing or pneumonia, excessive appetite but poor weight gain, and bulky stools
- An individual must inherit a defective copy of the CF gene – one from each parent – to have cystic fibrosis

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- **Currently, most individuals with cystic fibrosis die in middle age – many in their 40s and 50s from lung failure**

***This is **false;** currently, most individuals with cystic fibrosis **die young – many in their 20s and 30s** from lung failure.

The glands **most affected** are those in the pancreas, the respiratory system, and sweat glands. Cystic fibrosis is usually recognized in infancy or early childhood, occurring chiefly among Caucasians. **Early signs** are a chronic cough; frequent, foul-smelling stools *(steatorrhea)*; and persistent upper respiratory infections. The **most reliable diagnostic tool** is the sweat test, which shows elevations of both sodium and chloride.

Note: In CF cells, salt **does not** move properly because the protein product of the CF gene is defective and makes a faulty channel for the chloride to exit.

Oral complications of cystic fibrosis: chronic mouth breathing *(due to the occlusion of nasal cavity and maxillary sinus)*, higher incidence of open bite, high palatal vault, discolored teeth–more pronounced at the cervical and first seen at the CEJ where enamel is the thinnest. Tetracycline is often given for recurrent pulmonary infections and may be implicated as a cause of staining.

Remember: Pierre Robin Syndrome is a hereditary disorder that presents the following in the neonate: **micrognathia** *(smallness of the jaws)*, **glossoptosis** *(downward displacement or retraction of the tongue)*, and a high-arched or **cleft palate.** Most children will require orthodontics.

The sum of the mesiodistal widths of the **primary molars** in any one quadrant is:

- 5–10 mm greater than the permanent teeth that succeed them → premolars
- 2–5 mm less than the permanent teeth that succeed them → premolars
- 2–5 mm greater than the permanent teeth that succeed them → premolars
- 5–10 mm less than the permanent teeth that succeed them → premolars

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- **2–5 mm greater than the permanent teeth that succeed them → premolars**

***Also,** the enamel on the occlusal surfaces of primary molars is of uniform thickness and is approximately 1 mm thick, as opposed to that of permanent molars, which is 2.5 mm thick.

Characteristics of primary molars *(as compared to permanent molars):*

- **Crowns** are **shorter** with pronounced **buccal and lingual cervical ridges** and **a constricted cervical area.**
- The **occlusal table** is **narrower faciolingually.**
- **Anatomy is shallower** *(i.e., the cusps are short, the ridges are not as pronounced and the fossae are not as deep).*
- **A prominent mesial cervical ridge** *(makes it easy to distinguish rights from lefts).*
- **Roots** are **longer and more slender** than the roots of the permanent molars. The roots are **extremely narrow mesiodistally** and **very broad lingually.**
- **Roots** are **very divergent and less curved.** There is **little or no root trunk.**

Remember: Leeway space is the size differential between the primary posterior teeth *(canine, first and second molars)*, and the permanent canine and first and second premolar. Usually the sum of the primary tooth widths is greater than that of their permanent successors. So when these primary teeth fall out, there is usually a slight amount of space *(about 3.1mm per side in the mandibular arch and 1.3mm per side in the maxillary arch).*This space is often used to help relieve crowding. If nothing is done to preserve this space, the permanent first molars almost always drift forward to close it.

The principal characteristics of **ADHD** are all of the following **except:**

- Inattention
- Mental retardation
- Hyperactivity
- Impulsivity

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• **Mental retardation**

Attention Deficit Hyperactivity Disorder *(ADHD)* is a condition that becomes apparent in some children in the preschool and early school years. It is hard for these children to control their behavior and/or pay attention. It is estimated that between 3 and 5 percent of children have ADHD, or approximately 2 million children in the United States. This means that in a classroom of 25 to 30 children, it is likely that at least one will have ADHD.

The cause is unknown. The disorder is 10 times more common in males than females. Typically affected children, whether intellectually handicapped or not, perform poorly in school because of the inability to attend to tasks at hand or to sit still during the school day. **Note:** If there are any questions concerning the ability of the child to handle dental treatment, contact the childs' physician. **In most cases, the child doesn't need any special treatment.**

Methylphenidate *(Ritalin)* is a mild CNS stimulant that is often used in children with ADHD. It should not be used in children under 6 years of age. Among the more serious adverse reactions are nervousness, insomnia, and anorexia.

Note: Amphetamines *(for example: Dextroamphetamine)* are also used to treat ADHD.

"Koplik's spots" are associated with:

- Smallpox *(Variola)*
- German measles *(Rubella)*
- Mumps
- Measles *(Rubeola)*

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• **Measles** *(Rubeola)* ***Before immunization,** measles was very common during childhood so that 90% of the population had been infected by age 20.

Measles *(also called Rubeola)* is a highly contagious viral illness characterized by a fever, cough, and a spreading rash. It is caused by a **paramyxovirus.** The incubation period is 1 to 2 weeks before symptoms generally appear. **The oral lesions are pathognomonic of this disease.** These characteristic "Koplik's spots" usually occur on the buccal mucosa. They are 1-2 mm, yellow-white necrotic ulcers that are surrounded by a bright red margin.

Rubella *(or German measles)* is a fairly benign viral disease. The symptoms usually include a red, bumpy rash, swollen lymph nodes *(most often around the ears and neck),* and a mild fever. Some people will feel a little achy. The virus can manifest in the oral cavity as small petechiae-like spots of the soft palate. The defects of congenital infection from an infected mother are more severe → enamel defects, hypoplasia, pitting and abnormal tooth morphology.

Smallpox *(Variola)* is an acute viral disease, it manifests itself clinically by the occurrence of a high fever, nausea, vomiting, chills, and headache. The skin lesions begin as small macules and papules which first appear on the face, but rapidly spread to cover much of the body. **Oral manifestations** include ulceration of the oral mucosa and pharynx. In some cases, the tongue is swollen and painful, making swallowing difficult.

Mumps is an acute contagious viral infection **characterized chiefly** by unilateral or bilateral swelling of the salivary glands, usually the parotid *(parotitis)*. Although it is usually a disease of childhood, mumps may also affect adults. The papilla of the opening of the parotid duct on the buccal mucosa is often puffy and reddened.

Which of the following is the most common **primary** tooth to be retained?

- Maxillary second molars
- Maxillary first molars
- Mandibular second molars
- Mandibular first

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- **Mandibular first molars**

Over-retained primary teeth in the mixed dentition:
- May prevent the normal eruption of the permanent teeth
- May be caused by the abnormal root resorption of the primary teeth
- Are often treated by extraction

Be careful in extracting these teeth. The succedaneous tooth bud may be in close proximity. This is especially true when placing the beaks of forceps into bifurcations of primary molars in older children. The **most frequent cause** of fracture of root tips in extracting a primary molar is root resorption between the apex and the bifurcation.

Note: If a permanent tooth bud is **accidentally extracted** while removing a primary molar, the best treatment is to **immediately orient** the tooth bud, replant the bud using digital pressure, and suture. The best way to extract a **primary molar** that has the permanent tooth bud close to it is to **section the tooth** and remove the parts individually.

At the age of **six years** a child's **head** is what percentage of its **adult size?**

- 30%
- 50%
- 80%
- 90%

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- **90%** → this is typical of **all neural tissues** in the body.

Miscellaneous Facts That You May Need to Know

- **At birth,** the jaw is large enough to **accommodate all primary teeth** if they were to erupt simultaneously.
- **At birth,** the **width of the face** has reached the greatest percentage of its adult size *(as opposed to height and depth).*
- **At birth,** the **palate is pretty flat,** in adults, it is vault-shaped *(this occurs by deposition of alveolar crestal bone).*
- **At birth,** a newborn **cannot differentiate** between sour, salt, or a bitter taste.
- **At birth,** the **cranial vault** is very near the size it will eventually attain in adulthood *(as compared to the cranial base, mandible, mid-face, etc.).* The brain and the cranial base are **fully developed** by age six.
- **In early life, tonsils** function to filter bacteria and program the production of antibodies.
- **From age 6-12,** the body's **lymph tissue is 200%** of its normal adult mass. Consequently, enlarged tonsils in a six-year-old are, at age twelve, most likely to be smaller. This is because lymphoid tissue in the nasopharynx **decreases at puberty.** At the same time, **genital tissue is developing.**

All of the following are **true** when comparing the normal child periodontium to the normal adult periodontium **except:**

- There is **greater** blood and lymph supply
- The alveolar crest is **flatter**
- The cementum is **thicker and more dense** than that of the adult
- Gingival pocket depths are **larger**
- Attached gingiva is **not as wide**

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- **The cementum is thicker and more dense than that of the adult**

***This is **false;** the cementum is **thinner and less dense** than that of the adult. Cementum tends to increase with age.

More comparisons of the child periodontium to the adult periodontium:

- **Gingival tissues are more red.** This is so because in the child the gingiva is more vascular, thinner and less keratinized.
- **Lack of stippling** → the connective tissue of the lamina propria is shorter and flatter.
- **Flabbier tissue** → this is due to a decreased density of connective tissue.
- **Rounded and rolled gingival margins** → this is probably due to normal eruption patterns.
- **The PDL fibers run parallel to the teeth.** In adults, the PDL's are more horizontal against the tooth. The PDL is also wider in the child. This is why you may see mobility in the child's teeth as well as a decreased resistance to forces. The fiber bundles of the PDL increase with age.
- **Alveolar bone is thinner,** especially in the area of the lamina dura. There are fewer trabeculae in the alveolar bone of the pediatric patient.

The **minimum** number of lobes from which any tooth may develop is:

- Two
- Three
- Four
- Five

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- **Four**

Tooth development begins with increased cell activity in growth centers in the tooth germ. A growth center *(lobe)* is an area of the tooth germ where the cells are particularly active. These lobes are **primary centers** of calcification and are primary sections of formation in the development of the crown of a tooth. They are represented by a **cusp** on posterior teeth and **mamelons** and **cingula** on anterior teeth. They are always separated by **developmental grooves**, which are very prominent in the posterior teeth and form specific patterns. With anterior teeth, their presence is **much less noticeable** and these lobes are separated by what are known as **developmental depressions.**

Summary of number of lobes:

- **All anterior teeth** → three labial and one lingual *(cingulum)*.
- **Premolars** → three buccal and one lingual. **Exception:** The mandibular second premolar has three buccal and two lingual lobes.
- **First molars** *(maxillary and mandibular)* → five lobes, represented by five cusps → one lobe for each cusp.
- **Second molars** *(maxillary and mandibular)* → four lobes, one for each cusp.
- **Third molars** → at least four lobes, one for each cusp ***variations are seen.

Usually mamelons are **worn off** after the tooth comes into functional position. The presence of mamelons in a teenager or an adult is **evidence of malocclusion.** Most likely there is an **anterior open bite relationship** where the incisors do not touch.

ORAL SURGERY & PAIN CONTROL

Legend

Major Topic	Abbreviation	Major Topic	Abbreviation
Adrenal cortex	Adren Cort	Fractures	Fractures
Anatomy	Anat	General Information	Gen Info
Anesthesia	Anesth	Grafts	Grafts
Biopsy	Biopsy	Implants	Implants
Disorders / Conditions	Disord / Cond	Miscellaneous	Misc.
Drugs	Drugs	Temporomandibular Joint	TMJ
Exodontia	Exo		

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Which of the following is the **most common** disorder affecting the TMJ?

- Internal derangement
- Degenerative joint disease *(DJD)*
- Myofascial pain dysfunction *(MPD)* syndrome

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• **Myofascial pain dysfunction (MPD) syndrome**

Disorders and diseases of the TMJ:

Myofascial pain dysfunction *(MPD)* **syndrome** → is considered to be the **most common cause** of TMJ pain. This is a disorder characterized by pain and masticatory **muscle spasm** and limited jaw opening. The condition is characterized by a unilateral dull, aching pain which increases with muscular use. Common complaints associated with referred pain include headache, otalgia, tinnitus, burning tongue and sometimes decreased hearing.

Internal derangement → is a disorder in which the articular disc is pulled anteriorly by the superior head of the lateral pterygoid muscle. It has two forms: **with reduction** *(when the disc is anterior to the condyle at rest; the misplaced disc returns to the head of the condyle for at least part of the mouth's opening-closing cycle)* and **without reduction** *(when the disc is always anterior)*. Derangement **with reduction** occurs in about 1/3 of the population at one time or other. Its only symptom may be a **clicking or popping sound** when the mouth is opened. Pain may occur, particularly when chewing hard foods. Derangement **without reduction** usually produces no sound, but maximum opening between the tips of the upper and lower incisors is reduced to <= 30 mm from the normal 40 to 45 mm.

Degenerative joint disease *(osteoarthritis)* → can be either primary or secondary. Primary disease is seen in old people and is a disease of wear and tear. Patients are usually asymptomatic, and when symptomatic, the complaints are usually mild. Secondary degenerative arthritis occurs secondary to trauma or chronic bruxism. It occurs in younger people and the symptoms are much more severe.

MPD syndrome is believed to be a stress related disorder. This increase in stress produces an increase in mandibular muscle tension and in combination with teeth clenching results in muscle spasm, pain, and dysfunction. **Note: MPD** often responds to an acrylic night guard *(also called an occlusal separator or occlusal appliance)* along with a soft diet, limited talking, and elimination of gum chewing. Moist heat applied to the face and nonsteroidal anti-inflammatory agents are also helpful during the acute phase.

Which surgical approach is the **best** to expose the TMJ?

- Preauricular
- Submandibular
- Both are the same

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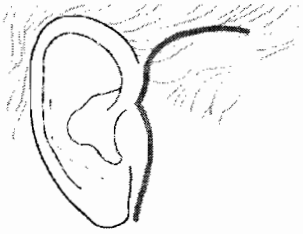
- **Preauricular**

Surgical approaches to the TMJ:

- **Preauricular** → the best incision to expose the TMJ. A perpendicular incision is made just anterior to the external ear parallel to the superficial temporal artery. The incision extends from one inch above the zygomatic arch to the lower extremity of the ear. The condyle is approached from behind. **Note:** With this approach, care must be taken not to damage either the facial nerve or the vessels that richly supply this area.



- **Submandibular approach** *(Risdon approach)* → this is the standard surgical approach to the **ramus of the mandible and neck of the condyle**. It is not the best approach for procedures within the joint space itself.

Remember: The most common cause of TMJ ankylosis is **trauma**. However, **ankylosis** is the most common complication of **rheumatoid arthritis.**

Which cranial nerve controls movement of the masseter and the temporalis muscles?

- Trigeminal *(CN V)*
- Olfactory *(CN I)*
- Oculomotor *(CN III)*
- Abducens *(CN VI)*
- Trochlear *(CN IV)*
- Optic *(CN II)*

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• **Trigeminal *(CN V)***

Summary of Cranial Nerves I —VI		
NO.	**Nerve**	**Main Functions**
I	Olfactory	**Sense** of smell
II	Optic	**Sense** of sight
III	Oculomotor	**Motor** supply to superior rectus, inferior rectus, medial rectus, and inferior oblique *(muscles that move eye)*; eyelid muscles; internal eye muscles that control lens shape and pupil size
IV	Trochlear	**Motor** supply to superior oblique muscle *(that moves eye)*
V	Trigeminal	**Ophthalmic division: Sensory** input from cornea; skin of scalp, forehead, and eyelids; mucous membrane of paranasal sinuses and nasal cavity. **Maxillary division: Sensory** input from skin of face over maxilla; upper teeth and gums; mucous membrane of nose, maxillary sinus, and palate. **Mandibular division: Motor** supply to muscles of mastication *(masseter, temporalis, medial & lateral pterygoids)* – **Sensory** input from skin of face over mandible; lower teeth and gums; temporomandibular joint; mucous membrane of floor of mouth and anterior part of tongue
VI	Abducens	**Motor** supply to lateral rectus muscle *(that moves eye)*

Important: Cranial nerves **III** *(oculomotor)*, **VII** *(facial)*, **IX** *(glossopharyngeal)*, and **X** *(vagus)* all have **parasympathetic** activity.

Which of the following is the **most common error** in blood pressure measurement?

- Applying the blood pressure cuff **too tightly**
- Applying the blood pressure cuff **too loosely**
- **Overinflating** the blood pressure cuff
- **Underinflating** the blood pressure cuff
- The use of **too large or too small** cuffs

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- **The use of too large or too small cuffs**

Important points concerning the measurement of blood pressure:

- Before performing a blood pressure reading, the patient should be comfortably seated with the back and arm supported, the legs uncrossed, and the upper arm at the level of the right atrium.
- Proper cuff size selection is critical to accurate measurement. The bladder length and width of the cuff should be 80% and 40%, respectively, of the arm circumference. Blood pressure measurement errors are generally worse in cuffs that are too small vs those that are too big.
- Blood pressure measurement in sitting and recumbent positions is acceptable. The diastolic blood pressure can be expected to be about 5 mm Hg higher in the sitting position.
- A difference in blood pressure between the two arms can be expected in about 20% of patients. The higher value should be the one used in treatment decisions.
- When measuring blood pressure, the cuff should be inflated to 30 mm Hg above the point at which the radial pulse disappears. The sphygmomanometer pressure should then be reduced at 2 to 3 mm/second. Two readings should be performed at least one minute apart.

Important: Use of the **wrong cuff size** can result in erroneous readings. A normal adult blood pressure cuff placed on an obese patient's arm will produce **falsely elevated** readings. This same cuff applied to the very thin arm of a child will produce **falsely low** readings.

Arrange the following **five phases of healing** of an extraction site in their correct order:

- Replacement of the connective tissue by fibrillar bone
- Hemorrhage and clot formation
- Replacement of granulation tissue by connective tissue and epithelialization of the site
- Recontouring of the alveolar bone and bone maturation
- Organization of the clot by granulation tissue

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Five Phases of healing of an extraction site:

1. Hemorrhage and clot formation
2. Organization of the clot by granulation tissue
3. Replacement of granulation tissue by connective tissue and epithelialization of the site
4. Replacement of the connective tissue by fibrillar bone
5. Recontouring of the alveolar bone and bone maturation

Note: Glucocorticoids have been shown to have the greatest effect on granulation tissue → **they retard healing.** This is believed to be due to the fact that:

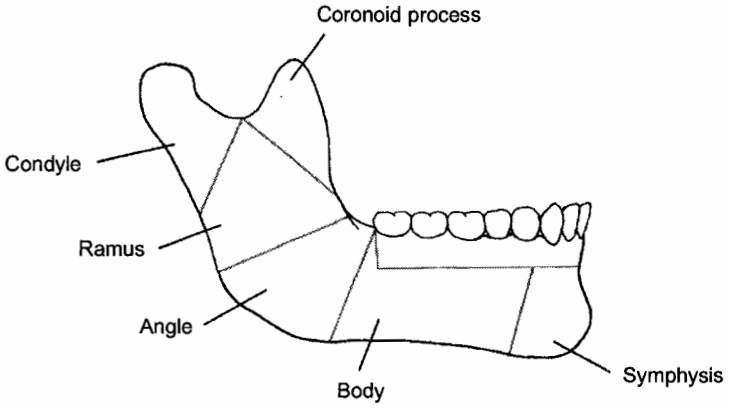
- Glucocorticoids interfere with the migration of neutrophils and mononuclear phagocytes into a site of inflammation; the phagocytic and digestive ability of macrophages is also reduced.
- Glucocorticoids inhibit formation of granulation tissue by retarding capillary and fibroblast proliferation and collagen synthesis.

The same stages that occur in normal wound healing of soft tissue injuries *(inflammation, fibroplasia, and remodeling)* also occur in the repair of injured bone. However, **osteoblasts** and **osteoclasts** are also involved to repair damaged bone tissue.

Bone heals by **primary** and **secondary** intention as does soft tissue.

- **Primary intention** bone repair involves both endosteal and periosteal proliferation. This type of bone repair occurs when either the bone is incompletely fractured or a surgeon closely reapproximates the fractured ends of a bone. **Little fibrous tissue is produced with minimal callus formation.**
- **Secondary intention** bone repair involves mostly **endosteal proliferation.** If fractured bones remain more than a millimeter apart, this type of repair takes place. Contains **a lot of fibrous tissue and a callus is formed.** This callus eventually ossifies.

Mandibular fractures can occur at any of the following sites. The **least common** site for a mandibular fracture to occur is:



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• **Coronoid process**

The **location** and **extent** of mandibular fractures are determined largely by the direction and intensity of the blow and the specific points of weakness in the mandible.

Common Sites of Mandibular Fractures	
Fracture Type	**Prevalence**
Body	30 – 40 %
Angle	25 – 31 %
Condyle	15 – 17 %
Symphysis	7 – 15 %
Ramus	3 – 9 %
Coronoid process	1 – 2 %

The **condylar neck** *(15 – 17% of fractures)* is a safety feature which allows the blow to the jaw to be dispersed at this point rather than driving the condyle into the middle cranial fossa. **Bilateral** dislocated fractures of the condylar necks will cause an anterior open bite and the inability to protrude the mandible. A **unilateral** fracture through the neck may cause forward displacement of the head of the condyle due to pull of the lateral pterygoid muscle.

The **symphysis area** or chin *(7 – 15% of fractures)* is usually where blows are sustained. These blows often result in fractures of the **subcondylar region.**

Remember: The patient's mandible **will deviate to the side of injury** upon opening.

Which of the following is considered to be the normal prothrombin time *(PT)?*

- < 7 seconds $\pm$ 2 seconds
- < 11 seconds $\pm$ 2 seconds
- < 14 seconds $\pm$ 2 seconds
- < 25 seconds $\pm$ 2 second

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• **< 11 seconds ± 2 seconds**

***To be a good candidate for surgery, the PT time should be **within 5 - 7 seconds** of the control sample.

Other tests used to measure a patient's clotting mechanisms:

- PTT *(partial thromboplastin time)* → detects coagulation defects of the intrinsic system. **Basic test for hemophilia.** Normal value - 25-36 seconds.
- Bleeding time → *(Ivy method)*, normal value = less than 9 minutes.
- Platelet counts → normal value = **1**50,000 - **4**50,000 per 1 cu mm of blood. The **minimal platelet count** for oral surgery is **50,000.**
- **INR** → is now the **preferred lab test** for assessing anticoagulant therapy in the patient taking warfarin.

The **INR** now is the preferred laboratory test for assessing anticoagulant therapy in the patient taking warfarin. The PT test has been used but this test was of limited value because of the variability among laboratories. To promote standardization of the PT test, the World Health Organization developed an international reference thromboplastin from human brain tissue and recommended that the PT ratio be expressed as the INR. While the INR range for minor oral surgery is subjective, local hemostatic measures generally are very effective if the INR **is less than 4.** No oral surgery, even of a minor nature, should be performed if the INR is **greater than 5.** For most patients on long-term warfarin therapy, the target INR is 2–3; these patients can be managed safely and effectively in the dental office. The normal INR for a non-anticoagulant patient **is 1.**

Important: Perhaps the **single most important** consideration in ruling out hemorrhagic disorder is **history.**

What nerve supplies sensory innervation to the anterior two-thirds of the tongue?

- Ophthalmic division *(V–1)*
- Maxillary division *(V–2)*
- Mandibular division *(V–3)*

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• **Mandibular division *(V–3)***

Remember: The trigeminal nerve contains **no** parasympathetic component at its origin.

Sensory innervation of V–3:

- Cheek
- Mandibular buccal gingiva

} **long buccal** nerve *(sensory only)*

- Jaw joint *(TMJ)*
- Auricle
- External auditory meatus

} **auriculotemporal** nerve *(sensory only)*

- Floor of mouth
- Mandibular lingual gingiva
- Anterior two-thirds of tongue

} **lingual** nerve *(sensory only)*

- Mandibular teeth
- Skin of chin and lower lip

} **Inferior alveolar** nerve *(mixed sensor and motor)*

Innervation of the tongue:

Motor: Hypoglossal *(XII)* nerve supplies both the intrinsic and extrinsic muscles of the tongue.

Sensory: Anterior 2/3: taste → chorda tympani branch of the facial nerve *(VII)*
sensation → lingual branch of mandibular division of trigeminal nerve
Posterior 1/3: both taste and general sensation by the glossopharyngeal nerve

A posterior superior alveolar *(PSA)* nerve block will provide anesthesia for:

- The second and third molars **along with** the mucoperiosteum of the palate
- The first and second molars **along with** the mucoperiosteum of the palate
- The first, second, and third molars **but not** the mucoperiosteum of the palate
- The first, second, and third molars **along with** the mucoperiosteum of the palate

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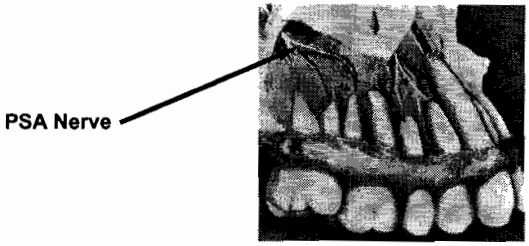
• **The first, second, and third molars but not the mucoperiosteum of the palate**

***For **extraction** of any or all of the three molar teeth, a greater palatine injection should be given to anesthetize the mucoperiosteum of the palate.



The PSA injection **does not always** anesthetize all of the roots of the **maxillary first molar tooth.** Therefore, if anesthesia of this tooth for either restorative dentistry or extraction is required, an infiltration injection also should be performed over the second premolar tooth.
Note: Patients experience **few subjective signs** of anesthesia after receiving a posterior superior alveolar nerve block, as compared to an inferior alveolar nerve block *(numb lip).*

Important: If a patient's face becomes distended and swollen **after** a posterior superior alveolar nerve block, **the following treatment is recommended:**
• Place cold packs and pressure on the affected side
• Explain to the patient that he / she may become black and blue on that side
***This is caused by **an intravascular** injection.

The propylene glycol in IV valium can cause:

- Cellulitis
- A unilateral facial paralysis
- Phlebitis
- Syncope

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• **Phlebitis**

Phlebitis is irritation or inflammation of a vein, it is sometimes seen after IV administration of valium. This is usually attributed to the presence of **propylene glycol** in the mixture.

Phlebitis is more likely to occur if a vein in the hand or wrist is used and may be more common following repeated injections, **especially in** heavy smokers, the elderly, and women taking oral contraceptives.

Common signs and symptoms of phlebitis:
• Pain
• Tenderness
• Edema
• Erythema
• Streaking of the limb

Treatment: Remove IV catheter, elevate limb, apply warm moist heat, initiate IV antibiotics → preferably cefazolin *(Ancef)*, 1 gm IV bolus push every 8 hours, for appropriate staphylococcus coverage.

Thrombosis is the formation of a blood clot that may partially or completely block a blood vessel. A clot located in an inflamed, blood vessel is called **thrombophlebitis.** A clot formation in a deeper vein is called a **deep vein thrombosis.** The primary danger in thrombophlebitis is that the clot may move and travel to a vital organ, such as the lung, causing serious damage.

Which of the following is a weak anesthetic and is used with other agents, such as thiopental, to produce surgical anesthesia?

- Halothane
- Enflurane
- Isoflurane
- Nitrous oxide
- Desflurane
- Sevoflurane

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• **Nitrous oxide**

Currently used inhaled general anesthetics include:
Halothane *(Fluothane)* is a powerful anesthetic. Very rarely, it can be toxic to the liver in adults, causing death. Halothane has a pleasant odor, and was frequently the anesthetic of choice for use with children, but since the introduction of sevofluorane in the 1990's, halothane use has declined.

Enflurane *(Ethrane)* is less potent and results in a more rapid onset of anesthesia and faster awakening than halothane. It has been found to increase intracranial pressure and the risk of seizures; therefore, its use is contraindicated in patients with seizure disorders.

Isoflurane *(Forane)* is not toxic to the liver but can cause some cardiac irregularities. Isofluorane is often used in combination with intravenous anesthetics for anesthesia induction. Awakening from anesthesia is faster than it is with halothane and enfluorane.

Desfluorane *(Suprane)* may cause coughing and excitation during induction and is therefore used with intravenous anesthetics for induction. It is rapidly eliminated and awakening is therefore faster than with other inhaled agents.

Sevofluorane *(Ultane)* unlike desfluorane, sevofluorane does not cause any coughing or other related side effects, and can therefore be used without intravenous agents for rapid induction. For this reason, sevofluorane is replacing halothane for induction in pediatric patients. Like desfluorane, this agent is rapidly eliminated and allows rapid awakening.

Nitrous oxide is a weak anesthetic and is used with other agents, such as thiopental, to produce surgical anesthesia. It has the fastest induction and recovery and is the safest because it does not slow breathing or blood flow to the brain. However, it diffuses rapidly into air-containing cavities and can result in a collapsed lung *(pneumothorax)* or lower the oxygen contents of tissues *(hypoxia)*.

Anesthesia performed with general anesthetics occurs in four stages which may or may not be observable because they can occur very rapidly. Which stage is the one in which skeletal muscles relax, and the patient's breathing becomes regular?

- Analgesia
- Excitement
- Surgical anesthesia
- Medullary paralysis

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• **Surgical anesthesia**

Four Stages of General Anesthesia:

- **Stage One** → Analgesia: The patient experiences analgesia or a loss of pain sensation but remains conscious and can carry on a conversation. **Note:** The best monitor of the level of analgesia is the **verbal response.**
- **Stage Two** → Excitement: The patient may experience delirium or become violent. Blood pressure rises and becomes irregular, and breathing rate increases. This stage is typically bypassed by administering a barbiturate, such as Methohexital or Thiopental, before the anesthesia.
- **Stage Three** → Surgical Anesthesia: During this stage, the skeletal muscles relax, and the patient's breathing becomes regular. Eye movements slow, then stop, and surgery can begin.
- **Stage Four** → Medullary Paralysis: This stage occurs if the respiratory centers in the medulla oblongata of the brain that control breathing and other vital functions cease to function. Death can result if the patient cannot be revived quickly. This stage should never be reached. Careful control of the amounts of anesthetics administered prevent this occurrence.

Notes:

1. The **medulla** is the last area of the brain to be depressed during general anesthesia. This area is the **most vital** part of the brain and contains the cardiac, the vasomotor, and respiratory centers of the brain.
2. The most reliable sign of **"oxygen want"** while monitoring a patient during **general anesthesia** is an increased pulse rate. Cyanosis may also be present.
3. The emergency most frequently experienced during outpatient general anesthesia is **respiratory obstruction.**
4. The best anesthetic technique used in oral surgery to avoid aspiration of blood or other debris when a patient is under general anesthesia is **endotracheal intubation** with pharyngeal packs.
5. A patient with an **acute respiratory infection** is contraindicated for general anesthesia.
6. The eyes are taped shut prior to draping a patient before surgery to **prevent corneal abrasion.**

An **incisional biopsy** is indicated for which of the following lesions?

- A .5 cm papillary fibroma of the gingiva
- A 2 cm exostosis of the hard palate
- A 2 cm area of Fordyce's disease of the cheek
- A 3 cm hemangioma of the tongue
- A 3 cm area of leukoplakia of the soft palate

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- **A 3 cm area of leukoplakia of the soft palate**

Leukoplakia is a **premalignant** lesion. This means that if left untreated, some of the lesions progress to carcinoma. It is because of this chance of malignant transformation that **all leukoplakias should be biopsied.**

Biopsy Technique and Surgical Principles:

- **Anesthesia:** Block local anesthetic techniques are employed when possible; if not, infiltration may be used but the solution should be injected at least **1 cm away** from the lesion.
- **Tissue stabilization:** Use fingers or clamps.
- **Hemostasis:** Gauze compresses *(avoid high speed suction).*
- **Incision:** Sharp scalpel.
- **Extent of tissue:** Obtain some normal tissue adjacent to lesion if possible.
- **Handling of tissue:** Use a traction suture through the specimen, **not** tissue forceps to avoid specimen trauma.
- **Specimen care:** After removal, the tissue should be immediately placed in **10% formalin solution** that is at least 20 times the volume of the surgical specimen. **Note:** No other solution is acceptable.

The Method of Tissue Removal Varies Among the Type of Biopsies:

1. In a **needle** *(percutaneous)* biopsy, the tissue sample is simply obtained by use of a syringe. A needle is passed into the tissue to be biopsied, and cells are removed through the needle.
2. In an **open** biopsy, an incision is made in the skin, the organ is exposed, and a tissue sample is taken.
3. A **closed** biopsy involves a much smaller incision than open biopsy. The small incision is made to allow insertion of a visualization device, which can guide the physician to the appropriate area to take the sample.

Remember: When the entire tumor is removed, it is called an **excisional biopsy** technique. If only a portion of the tumor is removed, it is called an **incisional biopsy** technique.

While extracting a **mandibular** third molar, you notice that the **distal root tip** is missing. Where is it **most likely** to be found?

- In the infratemporal fossa
- In the submandibular space
- In the mandibular canal
- In the pterygopalatine fossa

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• **In the submandibular space**

Important: To prevent this, **avoid all apical pressures** when removing the roots or root tips of all mandibular molars. If a mandibular molar root tip is displaced inferiorly, it may either be in the mandibular canal or through the lingual cortical plate.

Note: The **submandibular space** usually drains infections from the **mandibular bicuspids** and **molars** because their apices lie **below the** mylohyoid muscle attachment.

The **submandibular space** is a potential space of the neck bounded by the **oral mucosa** and **tongue** anteriorly and medially; the superficial layer of **deep cervical fascia** laterally, and the **hyoid bone** inferiorly. It comprises **two spaces**, the sublingual and submaxillary spaces, divided by the **mylohyoid muscle.**

The **submental space** is the medial part of the submaxillary space. It contains the **submental lymph nodes** that drain the median parts of the **lower lip, tip of the tongue, and the floor of the mouth.** Usually drains infections from the **mandibular incisors** and **canines** because their apices **lie above** the mylohyoid muscle attachment.

The **sublingual space** is the superior part of the submandibular space, containing the sublingual gland and loose connective tissue surrounding the tongue.

Remember: Ludwig's angina is the most commonly encountered neck space infection *(involves the sublingual, submandibular, and submental spaces).*

While attempting to remove a **grossly decayed mandibular molar,** the crown fractures. What is the recommended next step in order to **facilitate the removal** of this tooth?

- Use a larger forcep and luxate remaining portion of tooth to the lingual
- Separate the roots
- Irrigate the area and proceed to remove the rest of the tooth
- Place a sedative filling and reschedule patient

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• **Separate the roots**

***This can be done with a chisel, elevator, or **most easily with a bur.**

Teeth with **two** or **more roots** often need to be sectioned into single entities prior to successful removal. A popular method of sectioning is to make a bur cut between the roots, followed by inserting an elevator in the slot and turning it 90° to cause a break.

Roots can be removed by **closed** technique. The surgeon should begin a surgical removal if the closed technique is not immediately successful.

Teeth **are resistant** to crush **but are not** resistant to shear. **Therefore:**
• Place the beaks of the forceps **opposite** to each other at the **same level** on the tooth.
• The beaks should be applied in a line **parallel** with the long axis of the tooth.

Remember: When luxating a tooth with forceps, the movements should be firm and deliberate, primarily to the facial with secondary movements to the lingual. The **maxillary first bicuspid is least likely to be removed by rotation forces** due to its root structure *(obviously molars are not removed by rotation).*

The **tongue** receives its blood supply from the:

- Vertebral artery
- Lingual artery
- Inferior alveolar artery
- Palatine artery

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• **Lingual artery**
***It also receives blood** from the tonsillar branch of the facial artery and the ascending pharyngeal artery.

The **lingual** artery arises from the **external carotid** artery at the level of the tip of the greater horn of the hyoid bone in the carotid triangle. Branches include dorsal lingual artery, suprahyoid artery, and sublingual artery *(which supplies sublingual gland)*. It terminates as the **deep lingual** artery, which ascends between the genioglossus and inferior longitudinal muscles. **Note:** The **floor of the mouth** also receives its blood supply from the **lingual** artery.

The **vertebral** arteries arise from the subclavian arteries and join to form the basilar artery. The basilar artery is the main blood supply to the brainstem and connects to the Circle of Willis.

The **inferior alveolar** artery is a branch of the maxillary artery that runs inferiorly and through the mandibular foramen to supply the mandibular teeth.

The **palatine** artery is a branch of the maxillary artery, with distribution to the soft palate, gingivae, and bones and mucous membrane of the hard palate.

Remember:
- **Motor innervation** → from the hypoglossal nerve *(CN XII)*.
- **Sensory innervation** → **lingual** *(branch of trigeminal CN V-3)* supplies the anterior two-thirds, **glossopharyngeal** *(CN IX)* supplies the posterior one-third *(including vallate papillae)*, vagus *(CN X)* through the **internal laryngeal nerve** supplies the area near the epiglottis. **Note:** Besides the posterior 1/3 of the tongue the **glossopharyngeal nerve** also supplies sensory innervation to the tonsil, nasopharynx and pharynx areas.
- **Taste** → **facial** *(CN VII)* **via chorda tympani** supplies the anterior two-thirds; **glossopharyngeal** *(CN IX)* supplies the posterior one-third.

Anticholinergic drugs like atropine are given preoperatively to:

- Minimize the amount of analgesia needed postoperatively
- Provide a more rapid induction of anesthesia
- Induce relaxation prior to induction of anesthesia
- Decrease the risk of bradycardia during surgery

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• **Decrease the risk of bradycardia during surgery**

All **anticholinergic drugs** interfere with the **binding of acetylcholine** at its receptor. The most common method for categorizing anticholinergic drugs is to identify the ionization state of nitrogen *(i.e., tertiary or quaternary)* because this affects the drug's ability to penetrate the CNS. Atropine, scopolamine, benztropine, dicyclomine, and trihexyphenidyl are all **tertiary compounds,** while glycopyrrolate, ipratropium, and probanthine are **quaternary.**

Note: As a general rule, tertiary compounds penetrate the CNS **more readily** than do quaternary *(i.e., ionized)* compounds. One exception to this rule is **atropine,** which at normal doses penetrates the CNS poorly.

The principal therapeutic uses of anticholinergic drugs in dentistry are:
• To **decrease** the flow of saliva during dental procedures
• To **decrease** the secretion from respiratory glands during general anesthesia
***Atropine** is the most commonly used anticholinergic drug for these purposes. Scopolamine penetrates the CNS more readily than atropine but is rarely used.

Notes:
1. A patient premedicated with atropine will exhibit **mydriasis** *(dilated pupils).*
2. Atropine is **contraindicated** for nursing mothers and for patients with **glaucoma.**

For a patient with end-stage renal disease *(ESRD)* on dialysis, it is recommended to perform oral surgery:

- The day before dialysis
- The day of dialysis
- The day after dialysis
- Two days before dialysis

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• **The day after dialysis**

End-stage renal disease *(ESRD)* is a condition in which there is a permanent and almost complete loss of kidney function. The kidney functions at less than 10% of its normal capacity. In end-stage renal disease, toxins slowly build up in the body. **Normal kidneys** remove these toxins *(i.e., urea and creatinine)* from the body through urine.

Patients with ESRD:
- Are often on **steroid** therapy
- Are more susceptible to **post-op infections**
- Have an **increased tendency** to bleed

***When **oral surgical procedures** are undertaken on these patients, **meticulous attention** to good surgical technique is necessary to decrease the risks of excessive bleeding and infection.

Some important points to remember when treating patients with renal insufficiency **and** those on hemodialysis:
- Never measure the patient's blood pressure on the arm where the dialysis shunt has been created
- Avoid the use of drugs that are **metabolized** or **excreted by the kidney**
- Do not use **NSAID's** *(they are nephrotoxic)*
- Perform oral surgery the day **after** dialysis
- **Consult physician** for possible prophylactic antibiotics

Which of the following is the most common technique used for **mandibular advancement?**

- The step osteotomy
- Mandibular ramus sagittal split osteotomy
- The vertical ramus osteotomy
- The vertical body osteotomies

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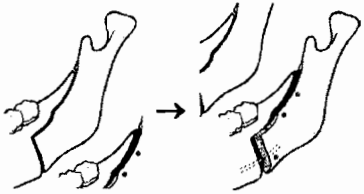
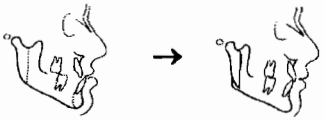
• **Mandibular ramus sagittal split osteotomy**

The mandibular ramus **sagittal split osteotomy** has become one of the most commonly performed mandibular orthognathic procedures. The mandible is split sagittally and can either be used to advance the mandible *(in the case of* **retrognathia***)* or to set back the mandible *(in treating* **prognathia***)*. It is the **standard procedure** used today. **Note:** The **position** of the **condyle** is unchanged during correction of mandibular prognathism or retrognathism.

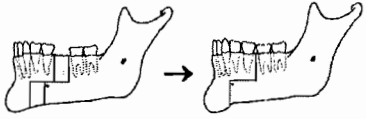


Vertical ramus osteotomy → can be used to set the mandible posteriorly. Used for the correction of **prognathism.**



Vertical body osteotomies → procedures that involve extracting mandibular teeth bilaterally *(usually bicuspids)*. A piece of bone is also removed from the mandible and you slide everything back. Used for **prognathism.**

The **step osteotomy** → may be indicated in cases of mandibular prognathism, retrognathism, asymmetry, and apertognathia. By performing bilateral step-shaped cuts in the body of the mandible, the lower jaw is divided into three separate, independently moveable pieces.is particularly well suited in cases with edentulous spaces



Note: A **LeFort I osteotomy** is most commonly used to correct **maxillary retrognathia.**

The most common direction in which the articular disc in the TMJ can be displaced is:

- Lateral
- Medial
- Posterior
- Anteromedial

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• **Anteromedial**

In a **healthy** temporomandibular joint *(TMJ)*, the articular disc is seated on the condyle and is held in place by the **collateral ligaments** *(also called "discal ligaments")* that are attached to the medial and lateral poles of the condyle. Attached to the anterior portion of the articular disc are muscle fibers from the lateral pterygoid muscle.

When the collateral ligaments become **elongated or torn,** they become loose which allows the lateral pterygoid muscle to pull the articular disc out of place. When this occurs, it is called a **disc displacement.** Because of the anteromedial direction of the lateral pterygoid muscle, the articular disc is usually displaced **anteromedially.**

Note: When the articular disc is displaced anteromedially to the condyle, a **click sound** is usually demonstrated when the mouth is opened and the condyle moves past the thick posterior band of the articular disc. There can also be a clicking sound when the mandible moves to the opposite side as the condyle again moves past the thick posterior band of the articular disc. Often another click will be demonstrated when the mouth is subsequently closed and the condyle moves from the thin central area of the disc and then past the thicker posterior band as the articular disc once again becomes displaced. A **crepitation sound** *(also known as crepitus)* is usually associated with a degenerative process *(osteoarthritis)* of the condyle, the **dull thud** is usually associated with a self-reducing subluxation of the condyle, and **tinnitus** is described as ear ringing.

All of the following characteristics raise the suspicion of malignancy **except:**

- Erythroplasia
- Ulceration
- Duration
- Slow growth
- Bleeding
- Induration
- Fixation

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- **Slow growth**

Characteristics of lesions that raise the suspicion of malignancy:
- **Erythroplasia** → lesion is totally red or speckled red and white
- **Ulceration** → lesion is ulcerated or is an ulcer
- **Duration** → more than two weeks
- **Rapid growth**
- **Bleeding** → bleeds on gentle manipulation
- **Induration** → lesion and surrounding tissue is firm to the touch
- **Fixation** → feels attached to adjacent structures

A red but not ulcerated area on mucous membrane is called **erythroplasia.** The texture may be normal or roughened. Size is variable, some being so small as to virtually escape detection whereas large areas are conspicuous to casual inspection. There are usually no symptoms. Being neither elevated nor depressed, they present as quiet, unpretentious lesions. The border may be sharp or blend imperceptibly into surrounding normal mucosa. It **must constantly** be kept in mind that early carcinoma frequently appears as an area of erythroplasia. There are certain areas of the oral mucosa which seem more prone to develop malignancy. Additionally, oral cancer is more often seen in those over age 40. Because of this, an area of erythroplasia in a cancer prone area in a patient past 40 is highly suspicious for malignancy and should be biopsied on the day it is seen. This is especially true for those lesions whose duration exceeds 2 weeks.

Which nerve is the **largest** of the 12 cranial nerves and is the **principal general sensory nerve to the head,** particularly the face?

- Vagus *(CN X)*
- Glossopharyngeal *(CN IX)*
- Facial *(CN VII)*
- Trigeminal *(CN V)*

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• **Trigeminal *(CN V)***

The **trigeminal nerve** exits the inferolateral pons as a sensory and motor root. **The larger sensory root** enters the trigeminal *(semilunar, gasserian)* ganglion in the middle cranial fossa. The three sensory divisions of the nerve arise from the ganglion and leave the cranial cavity through foramina in the sphenoid bone. The smaller motor root passes under the ganglion and joins the mandibular division as it exits through the foramen ovale. The mandibular division innervates eight muscles.

Somatic sensory cell bodies of the ganglion's sensory fibers enter the:
- **Ophthalmic division** *(V-1)* to supply general sensation to the orbit and skin of face above eyes.
- **Maxillary division** *(V-2)* to supply general sensation to the nasal cavity, maxillary teeth, palate, and skin over maxilla.
- **Mandibular division** *(V-3)* to supply general sensation to the mandible, TMJ, mandibular teeth, floor of mouth, tongue and skin of mandible.

The axons of the neurons enter the pons through the sensory root and terminate in one of the three nuclei of the trigeminal sensory nuclear complex:
- **Mesencephalic nucleus** mediates proprioception *(ex. muscle spindle)*
- **Main sensory nucleus** mediates general sensation *(ex. touch)*
- **Spinal nucleus** mediates pain and temperature from head and neck

Note: Proprioceptive fibers from muscles and the TMJ are found only in the mandibular division. The cell bodies of proprioceptive first order neurons are found in the **mesencephalic nucleus, not the** trigeminal ganglion. The TMJ, as is the case with all joints, receives no motor innervation. The muscles that move the joint receive the motor innervation.

Branchiomeric motor fibers innervate the temporalis, masseter, medial and lateral pterygoid, anterior belly of the digastric, mylohyoid, tensor tympani, and tensor veli palatini *(palati)*.

What is the **first step** when initiating CPR?

- Administer oxygen
- Establish unresponsiveness
- Administer epinephrine
- Place a cool towel on the person's forehead

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• **Establish unresponsiveness** → shake and shout → **"Are You OK"?**

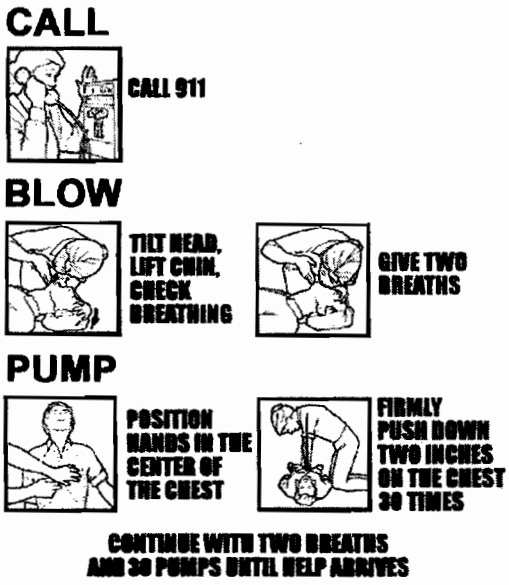
Then:

Important points to remember in CPR:

- If efforts are effective, the **pupils will constrict**
- If too much pressure is incorrectly applied directly over the xyphoid process, **the liver may be injured**
- The result of interruptions in chest compressions while performing CPR is a reduction of the blood flow and **fall in the blood pressure to zero**

Remember, you should **stop CPR only** under the following conditions:

- If another trained person takes over CPR for you
- If EMS personnel arrive and take over care of the victim
- If you are exhausted and unable to continue
- If the scene becomes unsafe



The correct position for ending a vertical releasing incision is:

- Mid-buccal of the tooth
- At a line angle of the tooth
- Mid-lingual of the tooth
- Any of the above

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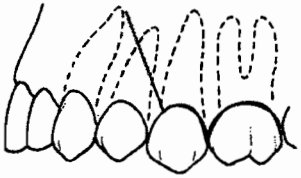
• **At a line angle of the tooth**



Important: The correct position for ending a vertical releasing incision is at a tooth line angle **not over** the buccal surface of a tooth. If it ends over a buccal surface, the edges are difficult to approximate and this may lead to periodontal problems. Incision should never cross bony prominences as this increases the chance for wound dehiscence.

Three types of **incisions** used in oral surgery:
1. **Linear** → straight line incision used for **apicoectomies.**
2. **Releasing** → used when adding a vertical leg to a horizontal creation incision. For **extractions, augmentations,** etc.
3. **Semi-lunar** → curved incision mostly used for **apicoectomies.**

***The **No. 15 blade** is universally used for oral surgical procedures

The term **flap** indicates a section of soft tissue that **(1)** is outlined by a surgical incision, **(2)** carries its own blood supply, **(3)** allows surgical access to underlying tissues, **(4)** can be replaced in the original position, and **(5)** can be maintained with sutures and is expected to heal.

The **basic principles** of oral surgical flap design:
- Flap design should ensure **adequate blood supply**; the base of the flap should be larger than the apex.
- Reflection of the flap should **adequately expose** the operative field.
- Flap design should permit **atraumatic closure** of the wounds.

In patients who have a **LeFort II fracture,** a common finding is **paresthesia** over the distribution of the:

- Infraorbital nerve
- Inferior alveolar nerve
- Mylohyoid nerve
- Hypoglossal nerve

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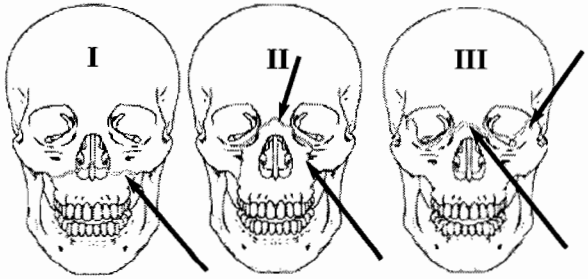
• **Infraorbital nerve**

Le Fort Fractures *(Mid Face Fractures)* → result from severe frontal blows. Frequently associated with intracranial damage, CSF leak.



Types of fractures:

- **Le Fort I** *(also called a horizontal fracture)* → a horizontal fracture through the maxillae just above the maxillary teeth. **Results is an open bite.**
- **Le Fort II** → a fracture in which the maxillae are separated from the facial skeleton with the separated bone being **pyramidal** in shape and including the palate and maxillary teeth. Signs include periorbital edema, ecchymosis, subconjunctival hemorrhage, and nose bleeding.
- **Le Fort III** → a horizontal fracture in which the entire maxilla and one or more facial bones are separated from the upper face. These patients will have **restriction of mandibular movement.**
- **Also** zygomaticomaxillary complex fractures, zygomatic arch fractures, or nasoorbital ethmoid fractures.

Important: The first step in the treatment of these fractures which affect the occlusal relationship is similar to the treatment of mandibular fractures → to re-establish a proper occlusal relationship by placing the maxilla into proper occlusion with the mandible.

Note: Due to the slope of the sphenoid bone comprising the floor of the cranial vault, blows to the maxilla will cause the maxilla to be driven backwards and downwards. **This may result in an open bite or impingement of the airway.**

The American Society of Anesthesiologists would give what **ASA** classification to a patient with **severe disease that is not incapacitating?**

- ASA - I
- ASA - II
- ASA - III
- ASA - IV
- ASA - V

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• **ASA - III**

American Society of Anesthesiologists (ASA)
Classification of Physical Status

ASA - I — A normal healthy patient

ASA - II — A patient with mild systemic disease or significant health risk factor *(such as smoking, excessive alcohol use, and obesity)*

ASA - III — A patient with severe disease that is not incapacitating

ASA - IV — A patient with severe systemic disease that is a constant threat to life

ASA - V — A moribund patient who is not expected to survive without the operation

ASA - VI — A declared brain-dead patient whose organs are being removed for donor purposes

The **sublingual** gland is made up mostly of:

- Serous cells, which secrete a watery fluid, essentially devoid of mucus
- Mucous cells, which produce a very mucus-rich secretion
- Acini composed of both serous and mucus epithelial cells
- None of the above

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- **Mucous cells, which produce a very mucus-rich secretion**

The sublingual glands are located in the floor of the mouth beneath the tongue, close to the midline. The mylohyoid muscle supports the individual sublingual glands inferiorly. They have numerous small ducts *(Rivian ducts)* that open onto the floor of the mouth. Most of the secretory units are **mucous secreting with serous demilunes.**

The **sublingual gland** is innervated by parasympathetic secretomotor fibers from **the facial nerve,** which run in the chorda tympani and in the lingual nerve *(branch of V-3)* and synapse in the **submandibular ganglion.** The **blood supply** comes from the **sublingual artery,** which is a branch of the lingual artery *(which is a branch of the external carotid).*

Important:
- **Lymphatic drainage** from both the sublingual and submandibular glands goes to the submandibular and the deep cervical lymph nodes.
- Sometimes the **numerous sublingual ducts** join to form a single main excretory duct *(Bartholin's duct)* that usually empties into the submandibular duct.

Note: Von Ebner's glands are located around the circumvallate papilla of the tongue. Their main function is to rinse the food away from the papilla after it has been tasted by the taste buds. They are purely serous.

Following a lesion of the hypoglossal nucleus or nerve, there is _____of the muscles of the _____one-half of the tongue.

- Hypertrophy, ipsilateral
- Hypertrophy, contralateral
- Atrophy, ipsilateral
- Atrophy, contralateral

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- **Atrophy, ipsilateral** *(same side)*

Lesions of the hypoglossal nerve:

- **Injury of the hypoglossal nerve** eventually produces paralysis and atrophy of the tongue on the **affected side** with the tongue deviated to the affected side. **Dysarthria** *(inability to articulate)* may also be found.

- **Unilateral lesions** of the hypoglossal nerve result in the deviation of the protruded tongue **towards the affected side.** This is due to the **unopposed action** of the genioglossus muscle on the normally innervated side of the tongue *(the genioglossus pulls the tongue forward)*. Remember, the genioglossus arises laterally in the tongue and inserts on the midline of the mandible.

Important: If the **genioglossus muscle is paralyzed,** the tongue has a tendency to fall back and obstruct the oropharyngeal airway with risk of suffocation.

Postoperative hypotension is **usually** due to the effect of:

- Transfusion reactions
- A fat embolism
- The anesthetic or analgesics on the myocardium
- Liver failure

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• **The anesthetic or analgesics on the myocardium**

***Leading to **myocardial depression.**

Other common causes of postoperative hypotension:
• Intravascular hypovolemia
• Rewarming vasodilation
• Hypothyroidism

The treatment is **narcan** *(a narcotic antagonist)* if hypotension is due to **narcotics.** Use atropine *(an anticholinergic)* if bradycardia is present.

***Possible treatment options include:**
- Elevation of the lower extremities
- Administration of carefully monitored fluid boluses
- Administration of vasopressors *(e.g., ephedrine)*

Note: Postoperative hypertension is most often due to **post-op pain.** Treat with narcotics and sedatives. **Other common causes include:**
• Hypercapnia
• Anxiety
• Overdistension of the bladder
• Hypoxia

The following signs: nausea, pallor, cold perspiration, widely dilated pupils, eyes rolled up, and brief convulsions are indicative of a patient having a _____ **reaction.**

- Somatogenic
- Psychogenic
- Either of the above
- None of the above

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• **Psychogenic**

***A **psychogenic reaction** is caused by psychological factors rather than physical factors *(drugs).*

Vasovagal syncope, **a psychogenic reaction,** is the most commonly experienced complication associated with the use of local anesthetic solutions. The clinical signs closely resemble those of shock. These psychogenic reactions readily respond to placing the patient in a supine position.

The following drugs, when administered one hour prior to the dental appointment, are **safe** and **effective** ways to allay the fears of an apprehensive **adult** dental patient and possibly avoid a psychogenic reaction in the dental chair:

- Diazepam *(Valium)*: 5 -10 mg orally *(PO)*
- Pentobarbital *(Nembutal):* 50 -100 mg orally *(PO)*
- Secobarbital *(Seconal)*: 50 -100 mg orally *(PO)*
- Promethazine *(Phenergan)*: 25 mg orally *(PO)*

***Note dosages and route of administration.**

These drugs are **not recommended unless** you have experience with them and can handle any complications that may happen from their use. **Note:** For a dentist to use "enteral sedation" *(the use of a pharmacological method that produces a minimally-depressed level of consciousness)* some states require special training and registration with the state.

Note: A **somatogenic reaction** is the development of a reaction from an organic pathophysiologic cause.

Malignant hyperthermia *(MH)* is a pharmacogenetic disease of skeletal muscle.
Characteristically patients with this disease:

- Have congenital heart disease
- Suffer from arteriosclerosis
- Have no signs or symptoms except during an anesthetic
- Have sickle-cell anemia

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• **Have no signs or symptoms except during an anesthetic**

Malignant hyperthermia is an autosomal dominant inherited condition. Classic MH most often manifests in the operating room, but it can also occur within the first few hours of recovery from anesthesia. When exposed to inhalational anesthetics, muscle metabolism increases, and a series of signs and symptoms appear, which if left untreated can lead to death. The **earliest findings** are an increased production of carbon dioxide and signs of increased sympathetic nervous system activity.

It is characterized by a **sudden, rapid rise in body temperature** associated with signs of increased muscle metabolism, such as tachycardia, tachypnea, sweating, and cyanosis; increased carbon dioxide production; and usually muscle rigidity. It usually occurs in apparently healthy children and young adults at an average age of 21 years. There is no sex differential. **Important:** Triggers for malignant hyperthermia → **1)** All potent inhalational agents including the newer agents sevoflurane, and desflurane **2)** All depolarizing muscle relaxants *(i.e., Succinylcholine, decamethonium)*.

When MH is diagnosed early and treated promptly, the mortality rate should be near zero. Whenever anesthesia is administered, dantrolene should be readily available as well as a protocol for management of MH *(100% oxygen, cooling procedures, and the correction of acidosis and hyperkalemia)*. Dantrolene is, at the moment, the only known drug that treats MH. It impairs calcium-dependent muscle contraction and controls hypermetabolism manifestations.

Note: People who have malignant hyperthermia are informed of their condition and advised that one-half of their first-degree relatives are likely to have the trait.

Dissociative anesthesia is a unique method of pain control that reduces anxiety and produces a trancelike state in which the person is not asleep, but rather feels separated from his or her body. The primary medication used is:

- Demerol
- Ketamine
- Pentobarbital
- Promethazine hydrochloride

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• **Ketamine**

Dissociative anesthesia is useful in emergency situations, such as an injury. It can also be used for short procedures that are painful, such as changing bandages. This method is safe and lasts only a short time. Because a person does not usually recall the procedure, this method is useful in children. The primary medication used is called **ketamine**. A sedative is often given before ketamine to reduce anxiety. A single dose of ketamine produces a trancelike state for about 10 to 30 minutes and pain control for about 30 to 45 minutes. The individual's eyes stay open during the procedure, but he or she is in a daze and feels no pain.

Note: A person who has had dissociative anesthesia usually does not remember the procedure, especially if a sedative has been given along with the pain medication. Most people feel back to normal within a few hours. As the medication wears off, an individual may have intense dreams and even hallucinations.

Ketamine:
• Increases secretions of the salivary and bronchial glands
• Increases BP, muscle tone and heart rate, **but not** respirations
• Side effects include: hypertension, increased pulse and delirium

Which cranial nerve provides **motor innervation** to the sternocleidomastoid and trapezius muscles?

- Facial *(CN VII)*
- Vestibulocochlear *(CN VIII)*
- Glossopharyngeal *(CN IX)*
- Vagus *(CN X)*
- Accessory *(CN XI)*
- Hypoglossal *(CN XII)*

• **Accessory *(CN XI)***

Summary of Cranial Nerves VII – XII		
NO.	**Nerve**	**Main Functions**
VII	Facial	**Motor** supply to muscles of facial expression; submandibular and sublingual salivary glands; lacrimal gland; mucous glands of nose and mouth — **Sense** of taste from anterior part of tongue; sensory input from outer ear
VIII	Vestibulocochlear	**Vestibular division:** Sense of balance; sense of head position. **Cochlear division:** Sense of hearing.
IX	Glossopharyngeal	**Motor** supply to stylopharyngeus muscle and parotid salivary gland — **Sense** of taste from posterior third of tongue; sensory input from blood pressure receptors in carotid artery
X	Vagus	**Motor** supply to pharynx, larynx, trachea, bronchi, lungs, heart, esophagus, stomach, intestines, liver, pancreas, and kidneys — **Sensory** input from pharynx, larynx, and thoracic and abdominal organs
XI	Accessory	**Motor** supply to sternocleidomastoid and trapezius muscles; muscles of soft palate, pharynx, and larynx
XII	Hypoglossal	**Motor** supply to muscles controlling tongue *(except for palatoglossus muscle)*

All of the following are resorbable sutures **except:**

- Plain gut
- Chromic gut
- Nylon
- Vicryl
- Dexon

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• **Nylon**

Resorbable sutures evoke an **intense** inflammatory reaction. This is the main reason neither plain gut or chromic gut are used for suturing the surface of a skin wound. When suturing an extraction site in the anticoagulated patient, a non-resorbable suture is recommended. Resorbable sutures are accompanied by an inflammatory response, increasing fibrinolytic activity, potentially resulting in clot breakdown.

Type of Suture	Degree of Knot Security	Tensile Strength	Duration of Wound Security	Tissue Reactivity
Absorbable:				
Surgical gut	Poor	Fair	5-7 days	Most
Chromic gut	Fair	Fair	10-14 days	Most
Polyglactin *(Vicryl)*	Good	Good	30 days	Minimal
Polyglycolic acid *(Dexon)*	Best	Good	30 days	Minimal
Polydixanone *(PDS)*	Fair	Best	45-50 days	Least
Polyglyconate *(Maxon)*	Fair	Best	45-50 days	Least
Nonabsorbable:				
Nylon *(Ethilon)*	Good	Good	N/A	Minimal
Polypropylene	Least	Best	N/A	Least
Silk	Best	Least	N/A	Most

Monofilament sutures consist of material made from a single strand. They resist infection by not harboring organisms. **Plain and chromic gut are examples:**

- **Plain gut** - from sheep intestine, susceptible to rapid digestion by proteolytic enzymes, retain strength for 5-7 days.
- **Chromic gut** - "chromitized" to produce more resistance to proteolytic enzymes, retains strength for 9-14 days.

Polyfilament sutures are made of multiple fibers that are either braided or twisted. They generally have good handling properties. The most common examples used in oral surgery are silk, Dexon, and Vicryl.

*****Non-resorbable sutures should be removed in 5-7 days.**

The **major** arterial supply to the temporomandibular joint is derived from the:

- Facial artery
- Angular artery
- Occipital artery
- Superficial temporal artery and the maxillary artery

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• **Superficial temporal artery and the maxillary artery**

The major **arterial** blood supply to the TMJ is derived from the **superficial temporal artery** and from the **maxillary artery** posteriorly, and from smaller masseteric, posterior deep temporal, and lateral pterygoid arteries anteriorly. The **venous drainage** is through a diffuse plexus around the capsule and rich venous channels that drain the retrodiscal tissue.

The **fibrous capsule** of the TMJ is innervated from a large branch of the **auriculotemporal nerve** *(branch of V-3)*. The **anterior** region of the joint is innervated from the **masseteric nerve** *(also a branch of V-3)* and from the **posterior deep temporal nerve** *(also a branch of V-3)*. The sensory innervation of the TMJ is via the trigeminal nerve as well. The nerve fibers primarily follow the vascular supply and terminate as free nerve endings. Thus, the capsule, synovial tissue, and extreme periphery of the disc are innervated. The articular cartilage and the central part of the disc contain **no nerves.** Both myelinated and nonmyelinated nerves are seen in the TMJ. The retrodiscal bilaminar zone has a rich neurovascular supply and is the source of proprioception.

Remember → muscles acting on the joint:
- Masseter
- Pterygoids *(medial and lateral)*
- Temporalis
- Digastric

Thrombocytopenia *(low platelet count)* that is less than_____is an absolute contraindication to elective surgical procedures because of the possibility of significant bleeding.

- 50,000 / mm^3
- 75,000 / mm^3
- 100,000 / mm^3
- 125,000 / mm^3

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• **50,000 / mm^3**

Patients with less than 10,000 - 20,000 platelets have been known to bleed spontaneously. Platelet counts between 50,000 and 100,000 have not been associated with significant bleeding provided platelet function is normal.

Possible etiologies for low platelet counts are:
- Idiopathic thrombocytopenic purpura *(ITP)*
- Disseminated intravascular coagulation *(DIC)*
- Marrow invasion or aplasia
- Hypersplenism
- Drugs
- Cirrhosis
- Transfusions
- Viral infections $\rightarrow$ infectious mononucleosis

Notes:
1. **Normal** platelet count is 150,000 - 450,000
2. **Emergency** procedures may be done with a few as 30,000 platelets if the dentist is working closely with the patient's hematologist and uses excellent techniques of tissue management.

Which of the following is defined as a "productive cough that is present for a period of 3 months in each of 2 consecutive years in the absence of another identifiable cause of excessive sputum production"?

- Congestive heart failure
- Emphysema
- Chronic bronchitis
- Asthma

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...ctive cough, often with wheezing, is a **universal factor** of chronic bronchitis.

COPD is broadly defined and encompasses several clinical and pathologic entities, namely emphysema and chronic bronchitis. Evidence of airflow obstruction that is chronic, progressive, and for the most part fixed, characterizes COPD.

Chronic bronchitis is a very common, debilitating respiratory disease, which has a **strong association** with cigarette smoking. In the early stages of chronic bronchitis, a cough usually occurs in the morning. As the disease progresses, coughing persists throughout the day. This chronic cough is commonly referred to as "smoker's cough."**Cor pulmonale** *(enlargement of the right ventricle of the heart)*, airway narrowing, and obstruction along with squamous metaplasia of the bronchial tree are common results of chronic bronchitis.

The **characteristic pathologic change** in chronic bronchitis is **hyperplasia** of bronchial submucosal glands and bronchial smooth muscle **hypertrophy**, which can be quantified by the **Reid index**, a ratio of glandular layer thickness to bronchial wall thickness.

Important: Patients with chronic bronchitis may be **predisposed to** lung cancer *(bronchogenic carcinoma)*.

Remember: Patients with chronic bronchitis *(or any COPD)* can have difficulty during oral surgery. Many of these patients depend on maintaining an upright posture to breathe adequately. They frequently experience difficulty breathing if placed in an almost supine position or if placed on high-flow nasal oxygen.

ORAL SURGERY & PAIN CONTROL

Misc.

Bleeding that consists of pinpoint dots of blood is called _____. Larger flat areas where blood has collected under the tissue, up to a centimeter in diameter, are called _____. A very large area is called a (an) _____.

- Purpura, petechiae, ecchymosis
- Petechiae, ecchymosis, purpura
- Ecchymosis, purpura, petechiae
- Petechiae, purpura, ecchymosis

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• **Petechiae, purpura, ecchymosis**

Postoperative ecchymosis is a result of trauma to the underlying blood vessels. Blood escapes from the vascular tree and accumulates in the tissues. **It is common after extractions in elderly patients** due to the fragility of the vessel walls. All patients should be warned that it may occur following extractions. **Note:** Sometimes the patient will complain of a diffuse, non-painful, **yellowish** discoloration of the skin. Moist heat often speeds the resolution of postoperative ecchymosis.

Remember: Osteoradionecrosis is the most serious potential complication after extractions from areas previously irradiated. Osteoradionecrosis *(ORN)* is a condition of nonvital bone in a site of radiation injury. ORN can be spontaneous, but it most commonly results from tissue injury. The absence of reserve reparative capacity is a result of the prior radiation injury.

ORAL SURGERY & PAIN CONTROL

Pericoronitis is acute inflammation of the tissue overlying and surrounding a partially erupted or erupting tooth. The most commonly involved tooth is a:

- Maxillary third molar
- Maxillary second molar
- Mandibular third molar
- Mandibular second molar

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...al picture is that of a markedly red, swollen, suppurative lesion. The involved tissue is very tender and often accompanied by pain radiating to the ear, throat, and floor of the mouth. Excruciating pain is produced when the opposing tooth impinges upon the inflamed tissue during mastication. There may be trismus of the masticator muscles on the affected side. Involvement of the cervical nodes, fever, and malaise are common. If this occurs, antibiotic therapy is indicated.

The **principal etiologic factors** in pericoronitis are food debris and bacterial waste products which have accumulated under the soft tissue flap, overlying a partially erupted tooth. This tissue is often traumatized during mastication which further exacerbates the situation.

Satisfactory emergency treatment is as follows:

1. Carefully cleanse beneath the tissue flap using a dental scaler if available. Then flush thoroughly with an irrigating syringe and warm saline.
2. Instruct the patient to rinse with warm saline hourly.
3. Prescribe a soft diet and instruct the patient to refrain from chewing on the affected side of the mouth.
4. Repeat treatment daily until the inflammatory reaction subsides.

Important: The **maxillary third molar** is the most frequent contributing factor to pericoronal infections found around mandibular third molars. Always examine the maxillary third molar, it may be supererupted or malaligned.

Alloplastic grafts are:

- Those where the bone to be grafted to the jaw is taken, or harvested from one's own body
- Taken from human donors
- Inert, man-made synthetic materials
- Harvested from animals

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• **Inert, man-made synthetic materials**

The term alloplastic is synonymous with **synthetic.** This indicates that the material is produced from inorganic sources and contains no animal or human components. Alloplastic materials offer a prepackaged solution to common reconstructive surgical problems without the need for autogenous grafting and donor site morbidity.

For bone replacement a man-made material that mimics natural bone is used. Most often **hydroxyapatite** *(HA)* is used for augmentation of the mandible. Hydroxyapatite is a dense, biocompatible material that can be produced synthetically or obtained from biologic sources such as coral. The **granular** or **particle form** is most commonly used for alveolar ridge augmentation. **Note:** When placed in a subperiosteal environment, HA bonds both physically and chemically to the bone.

Some advantages and disadvantages of restructuring an atrophic ridge with hydroxyapatite granules:

• **Advantages:**
  - It is a simple surgical technique suitable as an office procedure
  - **No donor site is required** to obtain autogenous bone graft material
  - Hydroxyapatite is **totally biocompatible and nonresorbable**

• **Disadvantages:**
  - Migration of the hydroxyapatite granules
  - Poor ridge form *(inadequate height)*
  - **Abnormal color** under the mucosa
  - Mental nerve **neuropathy** - excessive augmentation
  - Cannot participate in phase 1 osteogenesis since no viable osteogenic cells are present

The **nerve to the mylohyoid muscle** is a branch of the:

- Ophthalmic nerve *(CN V-1)*
- Maxillary nerve *(CN V-2)*
- Mandibular nerve *(CN V-3)*
- Facial nerve *(CN VII)*

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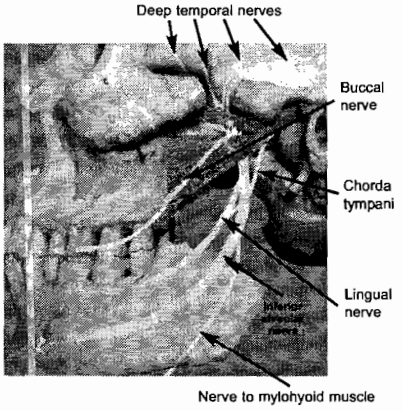
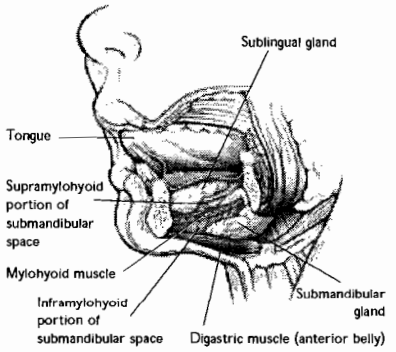
• **Mandibular nerve *(CN V-3)***

Function of mylohyoid → elevates hyoid bone, base of tongue, and floor of mouth. The **sublingual gland** is located **superior** to the mylohyoid muscle.





Notes:

1. **When placing the film** for a periapical view of the mandibular molars, it is the **mylohyoid muscle** that gets in the way if it is not relaxed.
2. When the floor of the mouth is lowered surgically, the **mylohyoid** and **genioglossus** muscles are detached.

When an adult victim has a pulse but is breathless, what is the recommended rate of rescue breathing?

- Once every 2 – 3 seconds
- Once every 5 – 6 seconds
- Once every 8 – 9 seconds
- Once every 10 –12 seconds

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- **Once every 5 – 6 seconds → 10 – 12 breaths / minute**

* When a **child** victim has a pulse but is breathless, the recommended rate of rescue breathing is once **every 3 seconds** *(20 breaths / minute).*
* A victim whose heart and breathing have stopped has the best chance for survival if emergency medical services are activated and CPR is begun within **four minutes.**
* Minimum length of time when assessing for a pulse is **5 seconds.**

CPR Ready Reference			
	Adults	**Children**	**Infants**
Rescue breathing, victim has a pulse, give 1 breath every	5-6 seconds	4 seconds	3 seconds
No pulse, locate compression landmark	The lower half of sternum, two finger widths superior to the xiphoid process	The lower half of sternum, two finger widths superior to the xiphoid process	One finger width below nipple line
Compressions are performed with	2 hands stacked: heal of one on sternum	Heal of one hand on sternum	Two or three fingers on sternum
Rate of compression per minute	100	100	At least 100
Compression depth	1-1/2-2"	1-1-1/2"	1/2-1"
Ratio compressions to breath - 1 rescuer - 2 rescuers	 15:2 15:2	 15:2 15:2	 15:1 15:1
Length of time to deliver each breath	1.5-2 seconds / breath	1-1.5 seconds / breath	1-1.5 seconds / breath

The first stage of wound healing is known as the:

- Maturation stage
- Inflammatory stage
- Proliferative stage

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• **Inflammatory stage**

Stages of wound healing:

1. **Inflammatory stage** *(consists of a vascular and cellular phase)*: neutrophils and lymphocytes predominate. The macrophage seems to be the most important inflammatory cell for wound healing.
2. **Proliferative stage** *(fibroblastic stage → mediated by fibroblasts)*: collagen and new blood vessels are produced.
3. **Maturation stage** *(remodeling stage)*: collagen fibers continue to increase tensile strength.

There are two basic methods of wound healing:

1. **Primary intention** *(also called primary closure or first intention)*. Involves minimal re-epithelialization and collagen formation, allowing the wound to be "sealed" within 24 hours. Healing occurs more rapidly with a lower risk of infection, with less scar formation and less tissue loss than wounds allowed to heal by secondary intention. Examples include: **well-repaired and well-reduced bone fractures.**
2. **Secondary intention** *(also called secondary closure or secondary intention)* involves re-epithelialization via migration from the wound edges, collagen deposition in the connective tissue, contracture, and remodeling. The site fills in with **granulation tissue.** Healing is slower and results in scarring and wound depression. Examples include: **extraction sockets,** poorly reduced fractures, and large ulcers.

Factors that impair wound healing → diabetic patients, patients with protein deficiencies, older patients, infections, foreign material, necrotic tissue, ischemia, and tension on the wound.

Remember: 3% hydrogen peroxide is the agent of choice for the debridement of intraoral wounds.

The **most common** pathognomonic sign of a **mandibular fracture** is:

- Nasal bleeding
- Exophthalmos
- Malocclusion
- Numbness in the infraorbital nerve distribution

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• **Malocclusion**

Other signs and symptoms of a mandibular body or angle fracture include:
• Lower lip numbness
• Mobility, pain, or bleeding at the fracture site

The treatment of mandible fractures can be divided into **open** and **closed** techniques. **Closed** treatment refers to **external fixation devices** and **intermaxillary fixation** *(IMF)* which is based on the principle that when the teeth of a fractured segment are in correct occlusion, then the bone fragments to which they are attached will, in most cases, also be satisfactorily reduced. The mandible must be immobilized for 3-6 weeks for most fractures. The most common technique for IMF is the use of **prefabricated arch bars.**

Open reduction involves direct exposure and reduction of the fracture through a **surgical incision.** When open reduction must be performed, direct surgical access to the area of the fracture must be obtained. This access can be accomplished through a number of surgical approaches, depending on the area of the mandible fractured. Both intraoral and extraoral approaches are possible. In either case a surgical approach should avoid vital structures such as nerves, ducts, and blood vessels and should result in as little scarring as possible. The traditional method of bony stabilization after open reductions has been the placement of direct intraosseous wiring combined with a period of IMF ranging from 3 to 6 weeks.

Notes:

1. Indications for **open reduction** include continued gross displacement of the bony segments and an unfavorable fracture that is likely to result in further displacement of the fractured segments caused by muscle pull, or when teeth are missing in one or more of the fractured segments.This type of reduction is commonly performed **for displaced angle** or **body fractures. Remember:** Condylar neck fractures are usually treated by **closed reduction.**
2. In a fracture involving the **angle of an edentulous mandible,** the proximal segment is usually displaced anteriorly and superiorly.

All of the following are phases of hemostasis **except:**

- Vascular
- Leukocytic
- Platelet
- Coagulation

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- **Leukocytic**

Normal Control of Bleeding

There are **three** phases of hemostasis:

1. **Vascular phase**
   - Vasoconstriction
   - Begins immediately after injury
2. **Platelet phase**
   - Platelets and vessel walls become sticky
   - Mechanical plug of platelets seals off openings of cut vessels
   - Begins seconds after injury
3. **Coagulation phase**
   - Blood lost into surrounding area coagulates through extrinsic and common pathways
   - Blood in vessels in area of injury coagulates through intrinsic and common pathways
   - Slower than other phases

Important: If a patient is taking aspirin, anticoagulants, broad-spectrum antibiotics, alcohol or anticancer medications you should be prepared to take **special measures in order to control the bleeding.** **Note:** Patients with specific systemic diseases will also have a prolonged bleeding time. These include nonalcoholic liver disease, hepatitis, cirrhosis, and hypertension.

Five means of obtaining wound hemostasis:

1. By **assisting natural hemostatic mechanisms** → usually accomplished by placing a cotton sponge with pressure on bleeding vessels or the use of a hemostat directly on the vessel.
2. By the **use of heat** on the cut vessels *(called thermal coagulation)*
3. By **suture ligation** of the vessel
4. By the **placement of a pressure dressing** over the wound → most bleeding from oral surgery can be controlled this way.
5. By using **vasoconstrictive substances** *(epinephrine)* in local anesthetics

Infection within the carotid sheath may damage all of the following structures **except:**

- Common carotid artery
- Sympathetic trunk
- Internal jugular vein
- Internal carotid artery
- Vagus nerve

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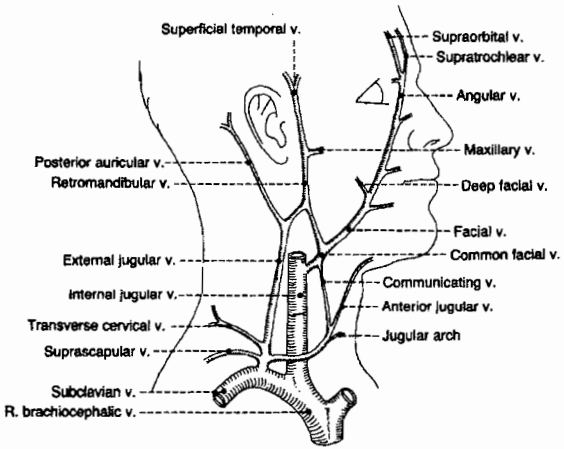
• **Sympathetic trunk**

***The carotid sheath does not contain the sympathetic trunk, which lies posterior to the carotid sheath and anterior to the prevertebral fascia.

The **carotid sheath** is located at the lateral boundary of the retropharyngeal space at the level of the oropharynx on each side of the neck deep to the sternocleidomastoid muscle. It extends from the base of the skull to the first rib and sternum. It contains the carotid arteries, the internal jugular vein, the vagus nerve, and the deep cervical lymph nodes.

The **facial vein** unites with the retromandibular vein below the border of the mandible and empties into the **main venous structure** of the neck, the **internal jugular vein.** The **internal jugular vein** descends through the neck within the **carotid sheath** and unites behind the sternoclavicular joint with the **subclavian vein** to form the **brachiocephalic vein.** The brachiocephalic veins *(right and left)* unite in the superior mediastinum to form the **superior vena cava,** which returns blood to the right atrium of the heart.



The infratemporal fossa communicates with the pterygopalatine fossa through the _____which is a cleft between the lateral pterygoid plate and the maxilla.

- Sphenopalatine foramen
- Pterygoid canal
- Petrotympanic fissure
- Pterygomaxillary fissure

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• **Pterygomaxillary fissure**

The **infratemporal fossa** is an irregular space behind the maxilla. Its roof is formed by the greater wing of the sphenoid. The lateral pterygoid plate of the sphenoid is medial. Laterally, it is limited by the coronoid process and ramus of the mandible. The infratemporal fossa communicates with the pterygopalatine fossa through the **pterygomaxillary fissure** which is a cleft between the lateral pterygoid plate and the maxilla. It communicates with the orbit through the **inferior orbital fissure** which is between the maxilla and the greater wing of the sphenoid. Contents of the **infratemporal fossa** include: • Some muscles of mastication *(lateral and medial pterygoids, lower part of the temporalis)* • The maxillary artery and some of its branches • The pterygoid venous plexus • The mandibular nerve and its branches • Otic ganglion • Chorda tympani

The **pterygopalatine fossa** is a small space behind and below the orbital cavity. It lies between the pterygoid plates of the sphenoid and palatine bone below the apex of the orbit.

Boundaries of fossa:

- Anterior wall → infratemporal surface of the maxilla
- Posterior wall → inferior portion of the greater wing of the sphenoid bone and the roof of pterygoid plates
- Roof → sphenoid bone and the orbital plate of the palatine bone
- Medial wall → perpendicular plate of the palatine bone
- There is **no lateral wall or floor** to the fossa; they are replaced by the pterygomaxillary fissure and descending palatine canal respectively

Note: The maxillary nerve *(V-2)* and the maxillary artery pass through the pterygopalatine fossa.The maxillary nerve enters the pterygopalatine fossa and branches into three major sections: the ganglionic branches, the zygomatic nerve and the posterior superior alveolar nerve. The ganglionic branches travel to the pterygopalatine ganglion, which in turn sends sensory, parasympathetic and sympathetic fibers back to the maxillary nerve.

The optimum site for **IV sedation** for an outpatient is the:

- Median basilic vein
- Median cephalic vein
- Median antebrachial vein
- Axillary vein

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• **Median cephalic vein**

This vein lies in the lateral aspect of the antecubital fossa *(anterior to the elbow)*. Avoid entering the brachial artery. **If the artery is entered,** the following symptoms will appear → immediate burning at the site of the injection, the arm will appear blotchy, and the pulse in the arm will be weak compared to the other arm.

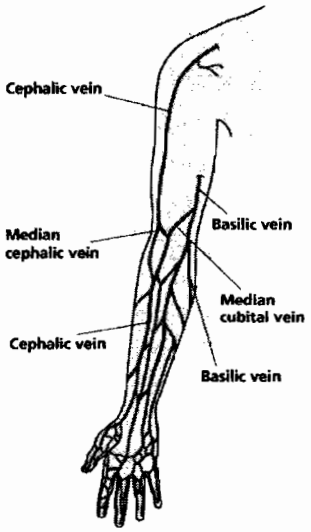
IV Sedation:

- Usually done with a 21 gauge needle
- Popular drug is **Valium** *(Diazepam)*
- The rate of injection of Valium is a 1 ml / minute
  — **1 ml of injectable Valium contains 5 mg of Valium**
- Injection is **discontinued** when the eyelids droop *(ptosis)*

Three common signs **indicating** when the correct level of sedation has been reached when using Valium:

1. Blurring of vision
2. Slurring of speech
3. **50% ptosis** of the eyelids *(this is called Verrill's sign)*

Remember: Valium is **contraindicated** for use in a patient with a history of narrow angle glaucoma.



Unlike other inhalation anesthetics, _____cannot be delivered by standard vaporizers. It requires the use of electrically heated vaporizers.

- Nitrous Oxide
- Desflurane
- Enflurane
- Halothane
- Isoflurane
- Sevoflurane

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• **Desflurane**

Inhalation anesthetics are substances that are brought into the body via the lungs and are distributed with the blood into the different tissues. The main target of inhalation anesthetics *(or so-called volatile anesthetics)* is the brain. Currently used inhalation anesthetics include five volatile liquids enflurane, halothane, isoflurane, sevoflurane, desflurane, and one gas *(nitrous oxide)*. The volatile liquids require a vaporizer for inhalational administration. The desflurane vaporizer has a heating component to allow delivery at room temperature.

Some inhalation agents have an unpleasant odor and may irritate the respiratory tract. This irritation may cause coughing and muscle spasms in the voice box, or larynx *(laryngospasm)*, or in the bronchial tubes in the lungs *(bronchospasm)*. Sevoflurane is less irritating to the airway than the others and is preferred for inducing anesthesia in children.

Important: All the potent inhalation agents are capable of triggering malignant hyperthermia *(MH)*, a rare inherited disorder that is potentially fatal.

The speed of induction of anesthetic effects depends on several factors:

1. **Solubility** → primary factor
2. **Inspired gas partial pressure**
3. **Ventilation rate**
4. **Pulmonary blood flow**
5. **Arteriovenous concentration gradient**

Administration of an inhalation anesthetic *(except nitrous oxide)* is usually preceded by intravenous or intramuscular administration of a short acting sedative hypnotic drug, often a barbiturate *(Thiopental)*. The procedure almost always requires endotracheal intubation.

Which of the following is the **phase of anesthesia** that begins with the administration of anesthetic and continuing until the desired level of patient unresponsiveness is reached?

- Induction
- Maintenance
- Recovery

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• **Induction**

*****Stage I and Stage II of general anesthesia together are referred to as induction.**

The **depth** of general anesthesia *(by inhalation)* varies with the partial pressure *(tension)* of the anesthetic agent in the brain, and the rates of induction and recovery depend upon the rate of change of tension in this tissue *(also blood supply to the lungs, pulmonary ventilation, and the concentration of the anesthetic influence the rate of induction)*. The signs and stages of anesthesia are most likely to be seen with anesthetic that has a **slow rate** of induction.

Notes:

1. **Maintenance** is the process of keeping a patient in surgical anesthesia.
2. **Recovery** is the phase of anesthesia commencing when surgery is complete and the delivery of the anesthetic is terminated and ending when the anesthetic has been eliminated from the body.
3. The behavior of patients under general anesthesia suggests that the most resistant part of the CNS is the **medulla oblongata** *(cardiac, vasomotor, and respiratory centers of the brain)*.
4. The most **controllable** route for administration of a general anesthetic is inhalation.
5. Minimum alveolar concentration (MAC) → alveolar concentration of anesthetic at which 50% of the patients are unresponsive to a standard surgical stimulus.
6. Meyer-Overton theory → anesthesia commences when a chemical substance reaches a certain molar concentration in the hydrophobic phase.
7. Second gas effect → potent agents are administered with nitrous oxide so that the potent agent will be delivered in increased amounts to the alveoli as gas rushes to replace the nitrous oxide absorbed by pulmonary blood.

Which size suture has the **least strength** and the **smallest diameter?**

- 9-0
- 3-0
- 2
- 5

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• **9–0**

Suture size is based on **strength and diameter.** This system uses **"0" as the baseline,** average size suture. As suture diameter **decreases,** "0's" are added or numbers followed by a "0" *(for example, 000 and 3–0 are the same size)*. As suture diameter **increases** above "0," numbers are assigned to the suture material.

Because suture material is foreign to the human body, the **smallest-diameter suture sufficient** to keep the wound closed properly should be used. Most oral and maxillofacial surgical procedures require the use of **3–0** or **4–0 sutures.**

The **TMJ** is a:

- Hinge joint
- Gliding *(sliding) joint*
- Combined hinge and gliding *(sliding)* joint

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- **Combined hinge and gliding *(sliding)* joint**

The TMJ is a **ginglymoarthrodial joint** *(meaning that it glides and rotates)*, permitting both **hinge-like rotation and sliding** *(gliding)* **movements. Note:** Ginglymus means rotation, and arthrodial means freely movable.

Components of the TMJ:

- **Mandibular condyle** *(sometimes called the condyloid process of the mandible)* – the articulating surface or functioning part of the condyle is located on the superior and anterior surfaces of the head of the condyle. This surface is covered with a dense layer of **fibrous connective tissue.**
- **Articular fossa** – this fossa is the anterior three-fourths of the larger mandibular fossa. It is considered to be a **non-functioning portion** of the joint. **Remember:** The mandibular fossa *(glenoid fossa)* is the temporal component of the TMJ; it is bounded in front by the articular eminence, and behind, by the tympanic part of the temporal bone, which separates it from the external auditory meatus.
- **Articular eminence** *(also called the articular tubercle)* – is a ridge that extends mediolaterally just in front of the mandibular fossa. It is considered to be the **functional portion** of the joint. It is lined with a thick dense layer of **fibrous connective tissue.**
- **Articular disc** *(also called the meniscus)* – is a biconcave **fibrocartilaginous disc** interposed between the condyle of the mandible and the mandibular *(glenoid)* fossa of the temporal bone which provides the gliding surface for the mandibular condyle, resulting in smooth joint movement. The central part is avascular and devoid of nerve tissue, only the extreme periphery is slightly innervated.

A history of rheumatic fever, IV drug abuse, or heart murmur should alert the dentist to the possibility of:

- Diabetes mellitus
- AIDS
- Valvular disease
- End stage renal disease

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• **Valvular disease**

Valvular heart disease is commonly caused by damage to the heart muscle as a result of rheumatic fever or a congenital abnormality. Common symptoms of **valvular heart disease** may include the following:
• Weakness on exertion
• Rapid heartbeat
• Chest discomfort
• Fainting spells
• No symptoms at all in some patients
Important: Patients with valvular heart disease are also at risk for bacterial endocarditis.

Rheumatic fever is a sequela of a previous Group A , beta hemolytic streptococcal infection, usually of the upper respiratory tract. The disease involves the heart, joints, central nervous system, skin, and subcutaneous tissues. It is characterized by an exudative and proliferative inflammatory lesion of the connective tissue, especially that of the heart, joints, blood vessels, and subcutaneous tissue.

Heart inflammation *(carditis)* disappears gradually, usually within five months. However, it may permanently damage the heart valves, resulting in **rheumatic heart disease.** The valve between the left atrium and ventricle *(mitral valve)* is **most commonly damaged. Note:** The pulmonary valve is rarely involved.

Remember: A **heart murmur** may have no pathological significance or may be an important clue to the presence of valvular, congenital, or other structural abnormalities of the heart.

Hemophilia A is characterized by a deficiency of:

- Clotting factor IX
- Clotting factor VIII
- Clotting factor XI
- von Willibrand factor

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• **Clotting factor VIII**

Hemophilia A and B are inherited as a **sex-linked recessive trait** by which males are affected and females are carriers. The majority of people afflicted with hemophilia have type A and it presents under the age of 25. The signs, symptoms and clinical manifestations include excessive bleeding from minor cuts, epitaxis, hematomas, and hemarthroses.

Classifications of Hemophilia:
- **Hemophilia A** → considered the classical type, caused by a deficiency of coagulation **factor VIII** *(anti-hemophilic factor)*.
- **Hemophilia B** *(also called Christmas disease)* → due to a deficiency in **factor IX** *(plasma thromboplastin component)*.
- **Hemophilia C** *(also called Rosenthal's syndrome)* → **not sex-linked**, less severe bleeding. Due to a deficiency of **factor XI** *(plasma thromboplastin antecedent)*.

Important → A true hemophiliac is characterized by having the following:
- **Prolonged partial thromboplastin time** *(PTT)*
- Normal prothrombin time *(PT)*
- Normal bleeding time

Note: von Willebrand's disease is inherited as an **autosomal dominant** bleeding disorder, it occurs with equal frequency in both sexes. Due to a deficiency in the von Willebrand factor, which is a large glycoprotein that has binding sites for factor VIII and also facilitates the adhesion of platelets to collagen *(important in the formation of a platelet plug)*.

Important: Patients with severe thrombocytopenia typically present with mucosal bleeds. Patients with hemophilia typically present with deep hemorrhage in weight-bearing joints.

The Caldwell-Luc procedure eliminates blind procedures and facilitates the recovery of large root tips or entire teeth that have been displaced into the maxillary sinus. When performing this procedure an opening is made into the facial wall of the antrum above the:

- Maxillary tuberosity
- Maxillary lateral incisor
- Maxillary premolar roots
- Maxillary third molar

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• **Maxillary premolar roots**

If a **large root fragment** or the entire tooth is displaced into the maxillary sinus *(antrum)*, it should be removed. The usual method is a **Caldwell-Luc approach.** This is a surgical procedure in which an opening is made into the maxillary sinus by way of an incision into the canine fossa **above the level** of the premolar roots. The tooth or root is then removed. Post-operative management includes a figure-eight suture over the socket, sinus precautions, antibiotics, a nasal spray and a systemic decongestant to keep the sinus ostium open and infection free. **Important:** An **oral surgeon** to whom the patient should be referred **should perform** this procedure.

If the root tip is **small** *(2 or 3 mm)*, **noninfected,** and cannot be removed through the small opening in the socket apex, **no additional** surgical procedure should be performed through the socket, and the root tip should be left in the sinus. If the root tip is left in the sinus, measures should be taken similar to those taken when leaving any root tip in place. The patient must be informed of the decision and given proper follow-up instructions.

Remember: The **palatal root of the maxillary first molar** is most often dislodged into the maxillary sinus during an extraction procedure.

Note: If a root tip of a **mandibular third molar** disappears from site while trying to retrieve it, the most likely location for it to be in is the **submandibular space.** Other possible locations would be the inferior alveolar canal or the cancellous bone space.

The universal sign of **laryngeal obstruction** is:

- Mydriasis
- Stridor *(crowing sounds)*
- Sweating
- Tachycardia

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- **Stridor** *(crowing sounds)*

***Stridor** is a high-pitched, noisy respiration, like the blowing of the wind. It demands immediate attention. It is caused by partial obstruction of the airway at the level of the larynx or trachea.

Because total airway obstruction usually occurs during inspiration, there is usually adequate oxygen left in the cerebral blood to permit up to 2 minutes of consciousness. If the obstruction is not recognized and managed and oxygen delivered to the victim's lungs, blood, and brain, permanent neurologic damage occurs within 3 to 5 minutes.

Non-invasive Procedures for Obstructed Airway

- Back blows, manual thrusts, Heimlich maneuver, chest thrust, and finger sweep

Invasive Procedures for Obstructed Airways

***These procedures should only be performed by **persons trained** in these techniques and **if proper equipment** is available.

- **Tracheotomy** → Is used more for long-term airway maintenance and not for emergency airways
- **Cricothyrotomy** → Is a procedure for establishing an emergency airway where other methods are unsuitable or impossible. The access site is the cricothyroid membrane of the trachea, located on the anterior neck, between the cricoid and thyroid cartilages.

Important: A cricothyrotomy may be lifesaving in an **anaphylactic reaction** in which a patient shows signs of laryngeal obstruction. If a patient shows signs of **laryngeal obstruction,** that is, stridor *(crowing sounds)*, epinephrine should be given and oxygen administered. If a patient **loses consciousness** and appears to be unable to breathe, an emergency cricothyrotomy may be required to bypass the laryngeal obstruction.

What is the best way to palpate the **posterior aspect** of the mandibular condyle?

- Intraorally
- Externally over the posterior surface of the condyle with the mouth open
- Through the external auditory meatus
- Any of the above

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- **Externally over the posterior surface of the condyle with the mouth open**

The **temporomandibular joint** should be evaluated for tenderness and noise. When checking for joint noises *(clicking and crepitus),* the joint is palpated laterally *(in front of the external auditory meatus)* while the patient opens and closes the mandible.

Tenderness can be assessed by palpating the lateral aspects of the joints when the mouth is closed and during opening of the mouth. The joint should also be palpated for tenderness while the patient opens maximally, and the fingertip should be positioned slightly posterior to the condyle to apply force to determine if there is inflammation of the retrodiscal tissue.

Note: By placing fingertips in the patient's external auditory meatus, this technique can produce false joint sounds during mandibular function because of pressure against the thin ear canal cartilage.

Remember: (1) The **posterior aspect** of the condyle is rounded and convex, whereas the **anteroinferior aspect** is concave. **(2)** The condyles are not symmetrical nor identical.

Although there are several types of malignant oral cancers, more than 90% of all diagnosed oral cancers are:

- Squamous cell carcinoma
- Hodgkin's disease
- Keratoacanthomas
- Salivary gland tumors

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• **Squamous cell carcinoma**

Squamous cell carcinoma *(SCC)* is the **most common malignant oral tumor**, representing a little over 90% of all oral malignancies. It is 9 to 10 times more frequent in males than in females and, although seen in all ages, its highest incidence is after the fourth decade.

SCC of the tongue is the **most common** intraoral malignancy. The most common location is the **posterior lateral border,** followed by the posterior one-third or base of the tongue. It is uncommon on the dorsum or tip of the tongue. These lesions usually **metastasize early** and the prognosis **is not as good as lip lesions.**

The floor of the mouth is the **second most common** intraoral location of SCC. It is seen predominantly in older men, especially those who are chronic alcoholics and smokers. These lesions metastasize early and the **prognosis is very poor.**

Remember: The **treatment of choice** for oral cancer is surgery.

Note: Squamous cell carcinoma usually affects the **lower lip** and rarely the upper lip. This occurrence has been attributed to greater exposure of the lower lip to sunlight. Lip carcinoma commonly presents as an ulcer. In many cases, a keratin crust covers the ulcer. The rest of the lip vermilion may show actinic changes. They are usually discovered early and only a small percentage show lymph node metastasis. **Prognosis is very good.**

Wharton's duct:

- Enters the oral cavity through buccal mucosa opposite the mandibular second bicuspid
- Enters the oral cavity through buccal mucosa opposite the maxillary second molar
- Enters the oral cavity near the sulculus terminalis
- Enters the floor of the mouth near the lingual frenula

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• **Enters the floor of the mouth near the lingual frenula**

The submandibular glands *(formerly called the submaxillary glands)* are located in the submandibular triangle *(digastric triangle)*. The **submandibular duct** *(Wharton's duct)* emerges from the anterior end of the deep part of the gland. It passes forward along the side of the tongue, beneath the mucous membrane of the floor of the mouth. It opens into the mouth on the summit of a small papilla, which is situated at the side of the frenulum of the tongue. **Clinically,** it is important to remember that the submandibular duct and the deep part of the gland can be readily palpated through the mucous membrane of the floor of the mouth alongside the tongue. Saliva can usually be seen emerging from the orifice of the duct.

The submandibular glands are innervated by parasympathetic secretomotor fibers from the **facial nerve,** which run in the chorda tympani and in the lingual nerve *(branch of V-3)* and synapse in the **submandibular ganglion. Note: This is the same as the sublingual glands. The blood supply comes from the facial artery,** *(which is a branch of the external carotid artery)*.

Important: During its course, Wharton's duct is closely related to the lingual nerve which eventually crosses over it. This is important because if you incise the mucous membranes of the floor of the mouth, depending on where you cut, you may expose the lingual nerve, Wharton's duct, and the sublingual gland.

Notes:

1. To expose the duct **intraorally,** only mucous membrane needs to be cut through.
2. Lymphadenopathy is the **most common** cause of swelling of the tissues in the submandibular triangle.

If a **small communication** is made with the maxillary sinus during extraction of a maxillary second molar, what treatment is recommended?

- The sinus communication should be closed with a flap procedure
- No additional surgical treatment is necessary
- A figure-eight suture should be placed over the tooth socket
- A continuous suture should be placed under tension over the tooth socket

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• **No additional surgical treatment is necessary** → allow the blood clot to form

Post-operative instructions to patient:
• Avoid nose blowing for 7 days
• Open mouth when sneezing
• Avoid vigorous rinsing
• Soft diet for 3 days

If a sinus communication should occur the following **medications** may be prescribed for one week:
1. Afrin → local *(nasal)* decongestant
2. Antibiotics *(Amoxicillin)*
3. Actifed → systemic decongestant

Notes:
1. If the opening is of **moderate size** *(2-6 mm)*, a figure-eight suture should be place over the tooth socket.
2. If the opening is **large** *(7 mm or larger)*, the opening should be closed with a flap procedure.

Important: If an entire tooth or a large fragment of one is displaced into the sinus, it should be removed. If the tooth fragment is irretrievable through the socket, it should be retrieved through a **Caldwell-Luc approach ASAP.** However, only perform this if you know what you are doing. If not, **refer patient** to an oral surgeon.

Remember: The integrity of the floor of the maxillary sinus is at greatest risk with surgery involving the removal of a **single remaining maxillary molar.** The fear here is possible **ankylosis.**

Which **two** major forces are used for routine tooth extractions?

- Rotation
- Pulling
- Pushing
- Luxation

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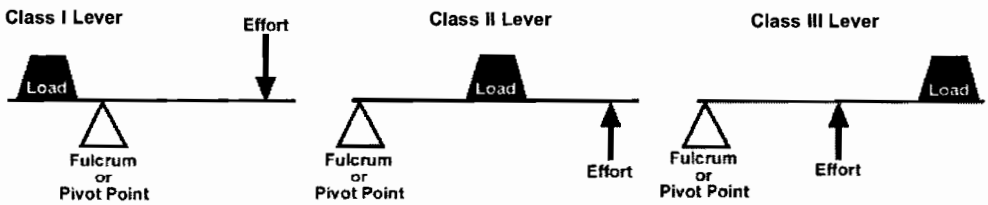
- **Rotation**
- **Luxation**

Luxation is the loosening of the tooth in the socket by progressive severing of the periodontal ligament fibers. Patience and controlled force are needed, **not** brute strength. The force should be applied as low down the root as possible when extracting teeth. You should support the jaw with your other hand and have a thumb and finger on either side of the tooth being extracted. **Note:** Rotation forces can be used on single rooted teeth. Teeth are extracted by **luxation forces** perpendicular to the long axis of the tooth, not by pulling along the long axis. The fulcrum is as **close to the apex** of the tooth as possible.

Remember: The beak of the extraction forcep is designed so that most of the pressure exerted during an extraction is transmitted to the **root** of the tooth.

Important: When using dental elevators, one should always have respect for the forces generated. Due to the large amount of leverage, dental elevators can generate tremendous forces during normal use, and have potential to cause iatrogenic damage.

Note: A **Class II lever** is used during tooth extractions → see pictures below



The **process of fracture healing** can occur in:

- One way → by direct or primary bone healing which occurs without callus formation
- One way → by indirect or secondary bone healing which occurs with a callus precursor stage
- Two ways → by direct or primary bone healing which occurs without callus formation and indirect or secondary bone healing which occurs with a callus precursor stage

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- **Two ways → by direct or primary bone healing which occurs without callus formation and indirect or secondary bone healing which occurs with a callus precursor stage**

Primary bone healing involves a direct attempt by the cortex to re-establish itself after interruption. Bone on one side of the cortex must unite with bone on the other side of the cortex to re-establish mechanical continuity. Under these conditions, bone-resorbing cells on one side of the fracture show a tunnelling resorptive response, whereby they re-establish new haversian systems by providing pathways for the penetration of blood vessels.

Secondary bone healing involves the classical stages of fracture healing.

The healing of bone can be divided into three overlapping phases:

1. **Hemorrhage** *(early inflammatory)* → occurs first and is associated with clot organization and proliferation of blood vessels. This nonspecific phase occurs during the first 10 days.
2. **Callus formation** *(repair)* → a primary callus is formed in the next 10 to 20 days. A secondary callus forms in 20 to 60 days.
3. **Functional reconstruction** *(late remodeling)* mechanical forces are important in this phase. The haversian systems are lined up according to stress lines. Excess bone is removed. The shape of the bone is molded to conform with functional usage so that bone may be added to one surface and removed from another. It takes 2 to 3 years to completely reform a fracture.

Remember:
Endosteal proliferation → occurs **within** a bone.
Periosteal proliferation → occurs **within** the connective tissue covering all bones *(periosteum)*.

Which artery supplies the maxillary molar teeth?

- Inferior alveolar artery
- Pterygoid portion of the maxillary artery
- Posterior superior alveolar artery
- Facial artery

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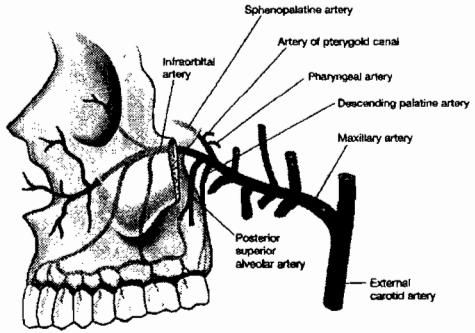
• **Posterior superior alveolar artery**

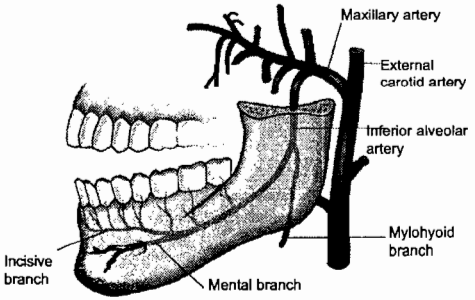
The **external carotid artery** supplies most of the head and neck, **except for** the brain *(the brain gets its blood supply from the internal carotid and the vertebral arteries)*. The external carotid passes through the parotid salivary gland and **terminates** as the maxillary and superficial temporal arteries. The superficial artery supplies the scalp. The **maxillary artery** supplies the muscles of mastication, the maxillary and mandibular teeth, the palate, and almost all of the nasal cavity.

The **mandibular teeth** receive blood from the inferior alveolar artery, which is a branch of the maxillary artery. The **maxillary teeth** also receive blood from branches of the maxillary artery as follows:

• **Posterior teeth** → from the posterior superior alveolar artery.
• **Anterior teeth** → from the anterior and middle superior alveolar arteries.

Remember: The **venous return** of both dental arches is the **pterygoid plexus** of veins.





A 52-year-old woman requests removal of a painful mandibular second molar. She tells you that she has **not rested for two days** and nights because of the pain. Her medical history is unremarkable, except that she takes **20 mg of Prednisone daily** for erythema multiforme. **How do you treat this patient?**

- Have patient **discontinue** the Prednisone for two days **prior** to the extraction
- Give **steroid supplementation** and remove the tooth with local anesthesia and sedation
- Instruct the patient to take **3 grams of amoxicillin one hour prior to extraction**
- **No special treatment is necessary** prior to extraction

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- **Give steroid supplementation and remove the tooth with local anesthesia and sedation**

Note: The fear here is that the patient may not have sufficient adrenal cortex secretion *(adrenal insufficiency)* to withstand the stress of an extraction without taking additional steroids. *(This holds true for any patient who has been treated for any disease with steroid therapy).*

Patients taking chronic daily doses of steroids *(total dose greater than 10-20 mg/day of prednisone)* should be considered for steroid supplementation, **in consultation with their physician.** Patients who have taken acute doses of prednisone greater than 10-20 mg/day within the past two weeks should also be considered for steroid supplementation.

For oral surgery on these patients:

- If currently taking steroids → double daily dose on the day of surgery, if pain is significant continue for first post-operative day
- If less than 2 weeks after steroid therapy discontinued → double daily maintenance dose on the day of surgery
- If more than 2 weeks after steroids stopped → none needed

Prednisone is a glucocorticoid prescribed as an anti-inflammatory agent.

Remember: Erythema multiforme is a hypersensitivity syndrome characterized by polymorphous eruption of skin and mucous membranes. Macules, papules, nodules, vesicles, or bullae and target or *("bull's-eye-shaped")* lesions are seen. A severe form of this condition is known as **Stevens-Johnson syndrome.** These patients may be receiving moderate doses of systemic corticosteroids and therefore may be unable to withstand the stress of an extraction. Consultation with their physician is absolutely necessary **before** treating these patients.

Local anesthetics stop axonal conduction by blocking:

- Calcium channels
- Sodium channels
- Potassium channels
- All of the above

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- **Sodium channels**

Local anesthetics produce anesthesia by inhibiting excitation of nerve endings or by blocking conduction in peripheral nerves. This is achieved by anesthetics **reversibly binding to and inactivating sodium channels.** Sodium influx through these channels is necessary for the depolarization of nerve cell membranes and subsequent propagation of impulses along the course of the nerve. When a nerve loses depolarization and capacity to propagate an impulse, the individual loses sensation in the area supplied by the nerve.

Local anesthetics **reversibly block** nerve impulse conduction and produce **reversible** loss of sensation at their administration site. The side of action of local anesthetics is at the **lipoprotein sheath** of the nerves.

Important: Small, myelinated nerve fibers which conduct pain and temperature sensations, are **affected first,** followed by touch, proprioception, and skeletal muscle tone.

Potassium, calcium, and **chloride conductances** remain unchanged.

Nitrous oxide works on the:

- Peripheral Nervous System *(PNS)*
- Central Nervous System *(CNS)*
- Autonomic Nervous System *(ANS)*

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• **Central Nervous System** ***(CNS)***

Nitrous oxide is the only **inorganic gas** used by the anesthesiologist. Room air contains 21% oxygen; **you must make sure that the patient receives at least this much oxygen.** Maximal safe concentrations of nitrous oxide *(70% nitrous / 30% oxygen)* produce analgesia and amnesia.

Nitrous oxide is carried in the bloodstream in physical solution. There is no metabolism or degradation of nitrous oxide in the body. **It is excreted solely via the lungs, unchanged.** High blood levels of nitrous oxide can be achieved quite quickly. **It is non-toxic to body tissues.** The only toxicity with the use of nitrous oxide is the lack of oxygen that could result from the operator's error. The gag reflex is only slightly obtunded with nitrous oxide analgesia. It is believed that nitrous oxide has its main effects on the **reticular activating system** and the **limbic system.**

Nitrous oxide is a weak anesthetic. It is used to supplement inhalation agents. It is the only inhalation anesthetic with **sympathomimetic** activity. It should **not be used in doses higher** than 70 % combined with 30% oxygen. It is known to diffuse into air containing spaces and to increase the pressure in such cavities. 100% oxygen should be administered during awakening in order to **avoid diffusion hypoxia.**

Remember:

- The **first symptom** of nitrous oxide analgesia is **tingling of the hands.**
- **Nausea** is the most common side effect of nitrous oxide analgesia.
- The correct total liter flow of nitrous oxide/oxygen is determined by the amount necessary to keep the reservoir bag **1/3 to 2/3 full.**

Local anesthetics are **most effective** in tissues that have a pH:

- Below 7
- Above 7
- Below 3
- Makes **no difference** what the pH of the tissue is

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• **Above 7**

Physiologic activity of local anesthetics is a function of their lipid solubility, diffusibility, affinity for protein binding, percent ionization at physiologic pH, and vasodilating properties.

Lipid solubility is an important characteristic. Potency is directly related to lipid solubility, because 90% of the nerve cell membrane is composed of lipid. Increased lipid solubility leads to faster nerve penetration and blockade of sodium channels.

Diffusibility of the local anesthetic through tissue other than nerve tissue also influences the speed of action onset.

Protein binding is related to the duration of action. The more firmly the local anesthetic binds to the protein of the sodium channel, the longer the duration of action.

Local anesthetics exist in **ionized** *(cation)* and **non-ionized** *(base)* **forms,** the proportions of which vary with the pH of the environment. The **non-ionized** *(base)* portion is the form that is capable of diffusing across nerve membranes and blocking sodium channels. Anesthetics with presence of greater non-ionized portions have a faster onset of action. Local anesthetics differ in respect to the pH at which the ionized and non-ionized forms are present at equilibrium, but this pH is generally in the range of 7.6 - 8.9. The more closely the equilibrium pH for a given anesthetic approximates the physiologic pH of tissues *(i.e., 7.35 - 7.45)*, the more rapid the onset of action.

A **decrease in pH** shifts equilibrium toward the ionized form, delaying onset of action. This explains why local anesthetics are slower in onset of action and less effective in the **presence of inflammation,** which creates a more acidic environment with lower pH. This limits the formation of **free base** *(the reaction shifts to the left)*.

The **initial effect of toxicity** upon the brain for local anesthetics is usually stimulation and then depression. However, it is also possible that the excitatory phase of the reaction may be extremely brief or may not occur at all. This is true especially with:

- Lidocaine
- Tetracaine
- Etidocaine
- Bupivocaine

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• **Lidocaine**

Local anesthetics are the most commonly used drugs in dentistry. The number of adverse reactions reported, particularly toxic reactions, are extraordinarily negligible.

Lidocaine usually has a sedative effect on the brain *(CNS)*. Initially, lidocaine toxicity depresses brain function in the form of **drowsiness** and slurred speech. It can progress to unconsciousness and even coma. **Important:** The initial effect of toxicity upon the brain for the other local anesthetics is usually **stimulation** *(the first sign of which is nervousness or increased anxiety)* and then depression.

In general, a mild toxic reaction manifests as agitation, talkativeness, and increased vital parameters *(blood pressure, heart rate, and respiration)*. If the clinical manifestations **do not** progress beyond these signs with retention of consciousness, **no definitive therapy is necessary.** The lidocaine will undergo redistribution and biotransformation, and the blood level will fall below the toxic level in a short time.

Treatment of a **sustained** convulsive reaction to a local anesthetic includes oxygen and Diazepam IV. If proper equipment and adequately trained staff **are not** available, **do not** attempt injections.

Some possible **side effects** of systemic absorption *(not necessarily toxic levels)* of lidocaine include tonic-clonic convulsions, respiratory depression, and decreased cardiac output.

In general, **barbiturates** have the ability to:

- Produce long-term cortical depression
- Provide sedation but no analgesia
- Provide sedation and analgesia
- Be sedative but not hypnotic

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- **Provide sedation but no analgesia**

Properties of barbiturates:

- CNS depressants → CNS depression with barbiturates is additive with alcohol and opioids
- Have no significant analgesic effect even at doses that produce general anesthesia
- Have anticonvulsant effects

Mechanism of action of barbiturates:

- Exert their effect by enhancing GABA receptor binding and prolonging the opening of chloride channels

Barbiturates:

- Well absorbed orally, distributed widely throughout the body
- Metabolized in the liver to inactive metabolites that are excreted in the urine

Therapeutic uses of barbiturates:

- Anesthesia → influenced by duration of action. Thiopental is an ultra-short acting barbiturate used IV to induce surgical anesthesia. **Note:** After IV administration, the **last tissue to become saturated** as a result of redistribution is fat *(as compared to liver, brain, and muscle tissue)*
- Anticonvulsant → phenobarbital used in long-term management of tonic-clonic seizures, status epilepticus and eclampsia
- Anxiety → can be used as mild sedatives to relieve anxiety and insomnia

Drug interactions → CNS depressants, alcohol, and opioid analgesics enhance the CNS depression of barbiturates.

Important: Barbiturates can lead to excessive sedation and cause anesthesia, coma and even death. Barbiturate overdoses may occur because the effective dose of the drug is not too far away from the lethal dose.

For impacted mandibular third molars, place the following in their correct order from the least difficult to most difficult to remove.

- Vertical
- Horizontal
- Distoangular
- Mesioangular

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- **Mesioangular**
- **Horizontal**
- **Vertical**
- **Distoangular**

Important: This is the **exact opposite of impacted maxillary third molars,** where the mesioangular impactions are the **most difficult** and the vertical and distoangular impactions are the **easiest to remove.**

***The mesioangular impaction is also the **most common** and comprises about 43% of all impacted teeth.

Once sufficient amounts of bone have been removed from around the impacted tooth, the tooth is usually sectioned. Sectioning allows portions of the tooth to be removed separately with elevators through the opening provided by bone removal. **Note:** Bone is rarely, if ever, removed on the lingual aspect of the mandible because of the likelihood of damaging the lingual nerve.

Sectioning of the tooth is done for the following reasons:

- Allows for minimal bone removal
- Allows for minimal force needed to remove tooth
- Shortens the entire surgical procedure

***Tooth sectioning can be performed with either a bur or chisel; **however,** the bur is used by most surgeons.

The **most common cause** of Cushing syndrome is:

- Ectopic ACTH Syndrome
- Pituitary Adenomas
- Adrenal Tumors
- Iatrogenic

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- **Pituitary Adenomas**

Cushing syndrome is a hormonal disorder caused by prolonged exposure of the body's tissues to high levels of the hormone cortisol. Sometimes called "hypercortisolism," it is relatively rare and most commonly affects adults aged 20 to 50. The female-to-male incidence ratio is approximately 5:1.

It is caused by prolonged exposure to elevated levels of either endogenous or exogenous glucocorticoids. Individuals can develop moon facies, facial plethora, supraclavicular fat pads, buffalo hump, truncal obesity, and purple striae. They often complain of proximal muscle weakness, easy bruising, weight gain, hirsutism, and, in children, growth retardation.

Exposure to excess glucocorticoids results in multiple medical problems, including hypertension, obesity, osteoporosis, fractures, impaired immune function, impaired wound healing, glucose intolerance, and psychosis. Exogenous steroids suppress the normal endogenous secretion of glucocorticoids, with full recovery taking as long as a year after cessation of glucocorticoid administration. Thus, patients who are on or who have taken steroids are at risk for developing an adrenal crisis.

Important: The patient's cardiovascular status must be evaluated and treated if necessary **prior to surgery.**

Management of an **acute asthmatic episode** occurring during oral surgery includes all *of the following* **except:**

- Terminate all dental treatment
- Position the patient in an erect or semi-erect position
- Patient should administer their own bronchodilator using an inhaler
- Administer nitroglycerin
- Administer oxygen
- Monitor vital signs

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• **Administer nitroglycerin**

***Nitroglycerin** is given in the management of a patient having **chest discomfort** → possible anginal attack.

Note: In most severe asthmatic attacks or when the patient's bronchodilator is ineffective, **epinephrine** *(0.3 ml of a 1:1,000 dilution)* can be injected IM or SC.

Asthma is a syndrome consisting of dyspnea, cough, and wheezing caused by bronchospasm, which results from a hyperirritability of the tracheobronchial tree. **There are two types: allergic asthma** *(most common form)* and **idiosyncratic** asthma.

Avoid the use of the following drugs: aspirin, NSAID's, barbiturates, narcotics, and erythromycin *(if patient is taking theophylline).*

Important: There are no contraindications for the use of nitrous oxide sedation in asthmatic patients. Because anxiety is a stimulus for an asthmatic attack, nitrous oxide sedation is actually beneficial for these patients. **If patient is taking steroids,** consult physician for the possible need for corticosteroid augmentation.

Note: The inhalation of a selective beta$_2$-agonist *(terbutaline, albuterol)* is the preferred treatment for an **acute asthmatic attack.**

Status asthmaticus is the most severe clinical form of asthma, usually requiring hospitalization, that does not respond adequately to ordinary therapeutic measures. If not managed properly, **chronic partial airway obstruction** may lead to death from **respiratory acidosis** *(which is produced by hypoxemia and hypercapnea).*

Which statement is true concerning Type 2 diabetes?

- Type 2 Diabetes is less common than Type 1 Diabetes
- Type 2 Diabetes usually develops in young persons
- The typical Type 2 Diabetes patient is skinny although there are exceptions
- Type 2 Diabetes is associated with insulin resistance rather than the lack of insulin like seen in Type 1 Diabetes

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- **Type 2 Diabetes is associated with insulin resistance rather than the lack of insulin like seen in Type 1 Diabetes**

Diabetes is the most common pancreatic endocrine disorder. It is a **metabolic disease involving mostly carbohydrates** *(glucose)* **and lipids.** It is caused by absolute deficiency of insulin *(Type 1)* or resistance of insulin's action in the peripheral tissues *(Type 2)*. The classic triad of symptoms includes polydipsia, polyuria, and polyphagia.

Comparison of Type 1 and Type 2 Diabetes Mellitus		
Characteristic	**Type 1 Diabetes**	**Type 2 Diabetes**
Level of insulin	None	May be normal or exceed normal
Typical age of onset	Childhood	Adulthood
Percentage of diabetes	10-20%	80-90%
Basic defect	Destruction of B cells	Reduced sensitivity of insulin's target cells
Associated with obesity	No	Usually
Speed of development of symptoms	Rapid	Slow
Development of ketosis	Common if untreated	Rare
Treatment	Insulin injections, dietary management	Dietary control and weight reduction; occasionally oral hypoglycemic drugs

ORAL SURGERY & PAIN CONTROL

Grafts

Match the term on the left with the correct description on the right.

- Autograft
- Allograft
- Xenograft
- Isograft

Tissue removed from an animal donor and surgically transplanted to a human

Tissue surgically removed from one area of a person's body, such as the iliac crest, and transplanted in another site on the same person

Tissue surgically transplanted from an individual of the same species who is genetically related to the recipient

Tissue surgically transplanted from one individual to a genetically non-identical individual of the same species

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- **Autograft** → tissue surgically removed from one area of a person's body, such as the iliac crest, and transplanted in another site on the same person
- **Allograft** → tissue surgically transplanted from one individual to a genetically non-identical individual of the same species
- **Xenograft** → tissue removed from an animal donor and surgically transplanted to a human
- **Isograft** → tissue surgically transplanted from an individual of the same species who is genetically related to the recipient

Classification of grafts *(or implants):*

- **Autogenous grafts** *(also called autografts)* are composed of tissues taken from the **same individual. Most frequently used in oral surgery.**
- **Allogeneic grafts** *(also called allografts)* are composed of tissues taken from an individual of the **same species** who is **not genetically related** to the patient *(usually cadaver bone)*.
- **Isogeneic grafts** *(also called isografts)* are composed of tissues taken from an individual of the same species who **is genetically related** to the recipient.
- **Xenogeneic implants** *(also called xenografts or heterografts)* are composed of tissues taken from a donor of another species, for example, animal bone grafted to man. **Rarely used in oral surgery.**

Note: Rejection of the graft is most common when **allografts** or **xenografts** of bone and cartilage are used in oral surgery. **Autogenous** grafts, although frequently presenting surgical and technical problems, **do not** as a rule involve rejection *(or immunological complications).*

The ideal graft should:

- Be replaced by the host bone
- Withstand mechanical forces
- Produce **no** immunologic response *(or rejection)*
- Actively assist osteogenic *(bone-forming)* processes of the host. The greatest osteogenic potential occurs with an **autogenous cancellous graft and hemopoietic marrow.**

All of the following are systemic **contraindications** to elective surgery **except:**

- Blood dyscrasias *(i.e., hemophilia, leukemia)*
- Controlled diabetes mellitus
- Addison's disease or any steroid deficiency
- Fever of unexplained origin
- Nephritis
- Any debilitating disease
- Cardiac disease

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- **Controlled diabetes mellitus**

***Uncontrolled diabetes mellitus** is a systemic contraindication to elective surgery.

Important: Patients with these systemic conditions can be treated, but you need to consult with the patient's physician before treatment. In most cases, these patients are best treated in the hospital by an oral surgeon.

Note: Cardiac disease, such as coronary artery disease, uncontrolled hypertension, and cardiac decompensation can complicate exodontia. **Usually a postinfarction patient is not subjected to oral surgery within six months of his infarction.** However, emergency procedures can be performed, provided the patient's physician has been consulted.

While there are many reasons for **autotransplanting** teeth, tooth loss as a result of dental caries is the most common indication, especially when:

- Maxillary central incisors are involved
- Mandibular first molars are involved
- Mandibular canines are involved
- Maxillary third molars are involved

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• **Mandibular first molars are involved**

First molars erupt early and are often heavily restored. **Autotransplantation** in this situation involves the removal of a third molar which may then be transferred to the site of an unrestorable first molar. Other conditions in which transplantation can be considered include tooth agenesis *(especially of premolars and lateral incisors)*, traumatic tooth loss, atopic eruption of canines, root resorption, large endodontic lesions, cervical root fractures, localized juvenile periodontitis as well as other pathologies.

Patient selection is very important for the success of autotransplantation. Candidates must be in good health, able to follow post-operative instructions, and available for follow-up visits. They should also demonstrate an acceptable level of oral hygiene and be amenable to regular dental care. **Most importantly,** the patients must have a suitable recipient site and donor tooth.

The **most important criteria** for success involving the recipient site is **adequacy of bone support.** There must be sufficient alveolar bone support in all dimensions with adequate attached keratinized tissue to allow for stabilization of the transplanted tooth.

The **donor tooth** should be positioned such that extraction will be as atraumatic as possible. Abnormal root morphology, which makes tooth removal exceedingly difficult and may involve tooth sectioning, is contraindicated for this surgery. Teeth with either open or closed apices may be donors; however, the **most predictable results** are obtained with teeth having between one-half to two-thirds completed root development. **Note:** The most likely cause of failure will be a chronic, **progressive external root resorption.**

Important: An **allogeneic** tooth transplant refers to a situation in which a tooth from one individual is placed in another individual. The almost **universal sequelae** of an allogeneic tooth transplant is **ankylosis and progressive root resorption. Remember:** The change in continuity of the occlusal plane observed **after ankylosis of a toot**h is caused by the continued eruption of the other non-ankylosed teeth and growth of the alveolar process.

One of the primary differences between ester-type and amide-type local anesthetics is the:

- Rate of absorption
- Extent of distribution
- Site of metabolism
- Duration of action

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• **Site of metabolism**

Amide type local anesthetics undergo biotransformation in the **liver** by microsomal enzymes but some is excreted unchanged *(10-20%)*. **Ester** type local anesthetics undergo rapid biotransformation in the **blood plasma.** The major portion of this inactivation process occurs within the blood through hydrolysis to paraaminobenzoic acid by the enzyme **pseudocholinesterase.** Patients with pseudocholinesterase inactivity are unable to detoxify ester type agents at a normal rate. **Amide** type anesthetics are **recommended** in these patients. An **easy way** to identify amide local anesthetics is to remember that the drug name contains an **i plus - caine** *(lidocaine, mepivacaine, and bupivacaine)*. Esters such as procaine, benzocaine, and tetracaine contain no i.

Allergic reactions to amide type local anesthetics are rare but may occur as a result of hypersensitivity to the local anesthetic agent itself or due to an **allergy to methylparaben** or other preservatives used in many solutions. These reactions are characterized by cutaneous lesions of delayed onset or urticaria, edema, and other manifestations of allergy.

Important: For those patients allergic to both ester and amide type local anesthetics, **Diphenhydramine** is a safe and effective alternative.

Amides	Duration of Action
Lidocaine *(Xylocaine)*	Med. *(30-60 min)*
Mepivacaine (*Carbocaine)*	Med. *(45-90 min)*
Bupivacaine *(Marcaine)*	Long *(120-240 min)*
Etidocaine *(Duranest)*	Long *(120-180 min)*
Prilocaine *(Citanest)*	Med. *(30-90 min)*
Esters	**Duration of Action**
Procaine *(Novacain)*	Short
Chlorprocaine *(Nesacaine)*	Short
Tetracaine *(Pontocaine)*	N/A
Benzocaine	N/A

A fasting blood glucose level above _____or a non-fasting glucose level greater than _____is indicative of diabetes.

- 100 mg/dl, 140 mg/dl
- 80 mg/dl, 100 mg/dl
- 50 mg/dl, 75 mg/dl
- 140 mg/dl, 200 mg/dl

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• **140 mg/dl, 200 mg/dl**

Glucose is regulated primarily by the liver in response to hormones released from structures such as the pancreas *(insulin, glucagon)*, adrenal medulla *(epinephrine)*, and the adrenal cortex *(cortisol)*. Blood glucose is used most commonly to diagnose diabetes mellitus and to explain the altered mental state.

An **increase** in glucose levels may indicate diabetes mellitus *(type 1 or 2)*, acute pancreatitis, hyperthyroidism, Cushing syndrome, acromegaly, or advancing age.

A **decrease** in glucose levels may indicate oral hypoglycemics or exogenous insulin, pancreatitis, starvation, liver disease, sepsis, or hypothyroidism.

Blood Chemistry / Normal Laboratory Values	
Arterial Blood Gases:	**Urine**
HCO_3 = 21 to 28 mEq/L	pH = 6.0 (4.7–8.0)
pCO2 = 35 to 45 mmHg	Specific gravity = 1.005 to 1.025
pH = 7.35 to 7.44	
pO_2 = 83 to 108 mmHg	

Blood Chemistry / Normal Laboratory Values	
Calcium = 9 to11 mg/dl	Protein = 5.5 to 8.0 g/dl
Carbon dioxide = 21 to 30 mEq/L	Potassium = 3.5 to 5.0 mEq/L
Chloride = 98 to 106 mEq/L	Phosphorus = 3 to 4.5 mg/dl, 1-1.5 mEq/L
Cholesterol Total = 180 to 240 mg/dl Esters = 100 to 180 mg/dl	Phosphatase Acid = 0.2 to 1.8 international units Alkaline = 21 to 91 international units
Creatinine = 1 to 2.0 mg/dl	Sodium = 132 to 142 mEq/L
Glucose = 70 to 120 mg/dl	Urea nitrogen = 10 to 20 mg/dl
Osmolality = 280 to 300 mOsm/L	

The treatment of mandible fractures can be divided into **open** and **closed** techniques. _____refers to **external fixation devices and intermaxillary fixation** *(IMF)* which is based on the principle that when the teeth of a fractured segment are in correct occlusion, then the bone fragments to which they are attached will, in most cases, also be satisfactorily reduced.

- Open reduction
- Closed reduction

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• **Closed reduction**

***It is called closed reduction because it **does not involve** direct opening, exposure, and manipulation of the fractured area. The mandible must be immobilzed for 3-6 weeks for most fractures.

Forms of immobilization of a fracture:

1. A **barton bandage** is the **simplest form** of immobilization. Primarily a **first aid measure** until definitive therapy can be instituted.
2. **Intermaxillary fixation** *(IMF)* is establishing a proper occlusal relationship **by wiring the teeth together.** This method will **successfully treat** most fractures of the mandible.The main methods for such fixation are wiring, **arch bars**, and splints. This is the **classical way** to immobilize the fracture after **closed reduction.**
3. **External skeletal fixation** is used in most cases in which the management of a fractured bone segment is **not satisfactorily accomplished by intermaxillary fixation.** It involves the placement of screws or pins through the skin on each side of the fracture and a cold cure acrylic bar which holds the screws in proper relationship with the fracture in the reduced position. It is cumbersome and esthetically displeasing.
4. **Direct intraosseous wiring** combined with a period of **IMF** is the traditional method of bone stabilization **after** open reduction. This method of stabilization can be accomplished through a variety of different wiring techniques. The wire is placed through holes on either side of the fracture and immobilization is accomplished by tightening the wires.

Zygomatic arch fractures can be nicely demonstrated by which radiographic view?

- Water's view
- Lateral skull view
- Posteroanterior skull view
- Submental vertex view

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• **Submental vertex view → See figure #2 below**

This type of fracture **(See figure #1)** may not cause any problem other than perhaps a slight sinking of the cheekbone area. There may be some encroachment and impairment in closure of the jaw if it comes down and entraps the coronoid process of the mandible.

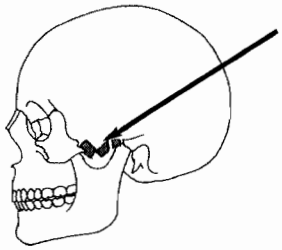

Figure #1

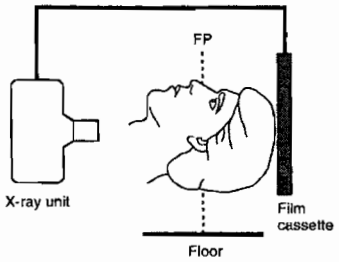

Figure #2

Important: Fractures of the facial bones, particularly the **zygomatic complex** may on rare occasions be complicated by damage to the contents of the **superior orbital fissure.**

Other possible complications of the zygomatic complex fracture include:
• **Paresthesia** *(most common)* → usually subsides
• The antrum *(sinus)* may be filled with a **hematoma,** which usually evacuates itself
• Ocular muscle balance **may be impaired** because of fracture of the orbital process
Note: Fracture of the **infraorbital rim** presents with the following symptoms:
• Numbness of the following areas on the **affected side** → upper lip, cheek, and nose.
Remember: The **Water's view** is best to evaluate orbital rim areas.

Currently, the most popular used implants are:

- Blade form implants
- Subperiosteal implants
- Transosseus implants
- Root form implants

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• **Root form implants**

Dental implants are divided into three categories based on their relationship to the oral tissues:

1. **Endosseous implants** are implants that are **surgically inserted into the jawbone.** They are the **most frequently used implants today.** They are further subdivided into **root form** and **blade** *(plate)* **form.**
2. **Subperiosteal implants** are frameworks specifically fabricated to fit on top of supporting areas in the mandible or maxilla under the mucoperiosteum. This type of implant **"rides on"** bone.
3. **Transosseous implants** are implants that are similar to endosseous implants in that they are surgically inserted into the jawbone. **However,** these implants actually penetrate the entire jaw so that they actually emerge opposite the entry site, usually at the bottom of the chin. **Note:** Their **primary indication** is in the very atrophic mandible where root form implants may further compromise the strength of the jaw.

Remember: Osseointegrated implants are anchored directly to living bone. This determination is made by **radiographic and light microscopic analysis.** Only endosseous and transosseous implants are considered true osseointegrated implants.

Root form implants → cylindrical in shape, can be smooth, threaded, perforated, and solid or hollow, vented, coated, or textured. They are available in various widths *(3.2 mm to 7 mm)* and lengths *(10 mm to 18 mm)*. Typically made of titanium. Treatment with root form implants is divided into three phases; surgical, healing and prosthetic. **Note:** These implants are the **most popular.**

Blade implants *(also known as plate form implants)* → are flatter in appearance and are utilized when there is insufficient width of bone but adequate depth is present. They are available in single and two stage forms. Typically made of titanium as well.

Two basic types of implant placement:

1. **Submerged** → requires a second surgical procedure *(two-stage)* to uncover the fixture.
2. **Nonsubmerged** → does not require a second surgical procedure *(one-stage).*

How many milligrams of **epinephrine** are in each cartridge *(1.8 cc)* of **2% lidocaine** with 1:100,000 epinephrine?

- 0.018 mg
- 18 mg
- 0.036 mg
- 36 mg

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- **0.018 mg** → 1.8 cc x .01 mg epinephrine = 0.018 mg

Important:

- **1 cc of 2% lidocaine with epinephrine 1:100,000** contains the following:
  - 20 mg of lidocaine
  - 0.01 mg of epinephrine
  - 6 mg of NaCL
  - 0.5 mg of sodium-metabisulfate → preservative to stabilize epinephrine
  - 1 mg of methylparaben *(a preservative)*
  - NaOH to stabilize pH

- **1.8 cc of 2% lidocaine** *(which is a carpule)* **with epinephrine 1:100,000** contains the following:
  - 36 mg of lidocaine → 1.8 x 20 mg
  - .018 mg of epinephrine → 1.8 x .01 mg
  - 10.8 mg of NaCl → 1.8 x 6 mg
  - .90 mg of sodium-metabisulfate → 1.8 x 0.5
  - 1.8 mg of methylparaben → 1.8 x 1 mg
  - NaOH to stabilize pH

Which lymph nodes directly receive lymph from the **tip of the tongue?**

- Submental lymph nodes
- Submandibular lymph nodes
- Parotid lymph nodes

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• **Submental lymph nodes**

All lymph from the head and neck region moves to a chain of **superficial cervical lymph** nodes found in the dermis of the neck. After these nodes, lymph is passed to **deep cervical lymph** nodes located along the carotid sheath just lateral to the trachea. From this terminal group, lymph flows into the **right or left jugular** lymph trunks. These vessels drain into either the **thoracic duct** or the **right lymphatic duct.**

Some regional groups of lymph nodes:

Parotid lymph nodes → receive lymph from a strip of scalp above the parotid salivary gland, from the anterior wall of the external auditory meatus, and from the lateral parts of the eyelids and middle ear. The **efferent lymph vessels** drain into the deep cervical lymph nodes.

Submandibular lymph nodes → receive lymph from the front of the scalp, the nose and adjacent cheek; the upper lip and lower lip *(except the center part)*; the paranasal sinuses; the **maxillary and mandibular teeth** *(except the mandibular incisors)*; the **anterior two-thirds of the tongue** *(except the tip)*; the floor of the mouth and vestibule; and the gingiva. The **efferent lymph vessels** drain into the deep cervical lymph nodes.

Submental lymph nodes → receive lymph from the **tip of the tongue**, the floor of the mouth beneath the tip of the tongue, the **mandibular incisor teeth and associated gingiva,** the center part of the lower lip, and the skin over the chin. The **efferent lymph vessels** drain into the submandibular and deep cervical lymph nodes.

Which of the following refers to the **amount of your blood** that is occupied by red blood cells?

- Erythrocyte sedimentation rate
- Specific gravity of the blood
- Hematocrit
- Partial thromboplastin time

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• **Hematocrit** → it's like chocolate milk: how much of it is **chocolate** and how much of it is milk.

The **CBC** and **urinalysis** are two tests that should be done **before** deciding whether to use a **general anesthetic** for surgical procedures on a patient. **Note:** If a bleeding problem is suspected the **PTT** *(partial thromboplastin time)* should also be evaluated.

Complete blood count (CBC) includes:

- **Hematocrit** *(the volume percentage of red blood cells in whole blood)*
  - Normal **men** = 40 - 54 percent
  - Normal **women** = 37 - 47 percent
- **Total leukocytes** *(WBC's)*
  - Normal = 5,000 - 10,000 / mm^3
  - **Dental infection** = 15,000 - 20,000 / mm^3

Important: The **minimal acceptable value** for the hematocrit is **30 percent** for elective surgery.

- **Hemoglobin**
  - Normal **men** = 14 - 18 g / dL
  - Normal **women** = 12 - 16 g / dL
- **Total erythrocytes** *(RBC's)*
  - Normal **men** = 5.0 (4.5 - 6.0) x 10^6 / mm^3
  - Normal **women** = 4.5 (4.3 - 5.5) x 10^6 / mm^3

Normal values for coagulation:

- Template bleeding time = 1 to 9 minutes
- Prothrombin time *(PT)* = 11 to 16 seconds *(compared to normal control)*
- Partial thromboplastin time *(PTT)* = activated, 32 - 46 seconds *(compared to normal control)*
- Platelets = 140,000 to 440,000 / ml

Important: Warfarin *(Coumadin)* is the most widely prescribed anticoagulant taken orally. It acts by interfering with vitamin K which is vital for blood clotting and the manufacture of prothrombin in the body. PT and PTT will be **increased** by warfarin.

Urine values:

- pH = 6.0 *(4.7-8.0)*
- Specific gravity = 1.005 to 1.025

How long should one **wait** before obtaining a biopsy of an oral ulcer?

- 4 days
- 7 days
- 14 days
- 30 days

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• **14 days** → 2 weeks

Almost all oral ulcers **caused by trauma** will heal within 14 days. Therefore, **any ulcer** that is present for 2 weeks or more **should be biopsied.**

Biopsy is also indicated in the following instances:

- Pigmented lesions *(black / brown)*
- When tissue is associated with **paresthesia,** this is often an **ominous sign.**
- If a lesion suddenly **enlarges,** it should be biopsied.

Note: Always aspirate a central bone lesion to rule out a vascular lesion. If a lesion seems compressible, pulsatible, blue, or a bruit is heard, beware of a vascular lesion and biopsy only under a controlled hospital setting.

***A **stethoscope** is used to listen for a **bruit.**

Sedation with nitrous oxide should be **avoided** in patients with:

- Asthma
- COPD
- ESRD
- Diabetes

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• **COPD** → mainly emphysema and chronic bronchitis

Administration of volatile anesthetics *(desflurane, enflurane, halothane, isoflurane, and sevoflurane)* is **not a concern** for COPD patients. All volatile anesthetics are bronchodilators and, therefore, are beneficial to patients with COPD. Sedation with nitrous oxide **should be avoided** in patients with COPD.

There are **no contraindications** for the use of nitrous oxide sedation in asthmatic patients. Because anxiety is a stimulus for the asthmatic attack, nitrous oxide sedation is actually beneficial for these patients.

Nausea and vomiting are the most common adverse effects, occurring in 1% to 10% of patients. Fasting is not required for patients undergoing nitrous oxide sedation. The practitioner, however, may recommend that only a light meal be consumed in the 2 hours prior to the administration of nitrous oxide. **Diffusion hypoxia** can occur as a result of rapid release of nitrous oxide from the blood stream into the alveoli, thereby diluting the concentration of oxygen. This may lead to headache and disorientation and can be avoided by **administering 100% oxygen** after nitrous oxide has been discontinued.

Remember: The most common **complication** associated with nitrous oxide sedation is a behavioral problem *(laughing, giddy)*.

Note: Some literature states that **nitrous oxide** is acceptable for the pregnant patient, **however,** from a risk management point it may be prudent not to use nitrous oxide on any pregnant patient.

All of the following are possible etiologies of respiratory acidosis **except:**

- COPD
- Asthma
- Cardiac arrest
- Severe pulmonary edema or pneumonia
- Anxiety or pain
- Injury to the airway or chest wall
- Cerebrovascular accident
- Drugs *(narcotics, sedatives)*
- Muscular dystrophy or myasthenia gravis

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• **Anxiety or pain** → are possible etiologies of respiratory alkalosis

An **abnormality** in one or more of the pH control mechanisms can cause one of two major disturbances in acid-base balance.

1. **Acidosis:** is a condition in which the blood has too much acid *(or too little base)*, frequently resulting in a decrease in blood pH. When the pH of the blood falls **below normal**, which is around 7.3, the central nervous system becomes **so depressed** that the person first experiences disorientation and later may become comatose. **Note:** The normal blood bicarbonate-carbonic acid ratio is 20:1. A 10:1 ratio indicates **uncompensated** acidosis. **Severe acidosis** always occurs during CPR.
2. **Alkalosis:** is a condition in which the blood has too much base *(or too little acid)*, occasionally resulting in an **increase** in blood pH. The **major effect** on the body is overexcitability of the nervous system. This may result in tetany *(tonic spasm)*.

Depending on the cause of the condition, acidosis or alkalosis may be **respiratory** or **metabolic:**

Metabolic acidosis is excessive blood acidity characterized by an inappropriately **low level** of bicarbonate in the blood. Possible etiologies: diabetic or starvation ketoacidosis, lactic acidosis, uremia, severe dehydration, and certain drugs *(aspirin, methanol, ethylene glycol)*.

Respiratory acidosis is excessive blood acidity caused by a buildup of carbon dioxide in the blood as a result of poor lung function or slow breathing *(decrease in respiratory rate)*. Possible etiologies are listed on the front of the card. **Treatment of acidosis** is ingestion of **sodium bicarbonate.**

Metabolic alkalosis is a condition in which the blood is alkaline because of an inappropriately **high level of bicarbonate.** Possible etiologies: vomiting, diuretics, cystic fibrosis, Cushing syndrome, hyperaldosteronism, exogenous steroids, or hypoparathyroidism.

Respiratory alkalosis is a condition in which the blood is alkaline because rapid or deep breathing results in a low blood carbon dioxide level. Possible etiologies: anxiety, pain, fever, pulmonary embolus, low levels of oxygen in the blood *(high altitude)*, head injury, hypoxia, pregnancy, hyperthyroidism, hepatic insufficiency and aspirin overdose. **Note:** It is much less common than respiratory acidosis. **Treatment of alkalosis** is ingestion of **ammonium chloride.**

Which of the following statements concerning a maxillary torus is true?

- Occurs more commonly in males
- Should be removed → excised in one piece
- Most often occurs in the midline of the hard palate
- Occurs most often in people over 70

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- **Most often occurs in the midline of the hard palate**

Here it is called the **torus palatinus.** They usually appear before the age of 30 and affect females more frequently than males.

Maxillary tori present few problems when the maxillary dentition is present and only occasionally interfere with speech or become ulcerated from frequent trauma to the palate.

Indications for the **removal** include a large, lobulated torus with a thin mucoperiosteal cover extending posteriorly to the vibrating line of the palate that **prevents seating of a denture** and also **prevents a posterior seal** at the fovea palatini. Other indications for the removal of maxillary tori are, chronic irritation, interference with speech, rapid growth and in patients that have a cancer-phobia.

Technique for removal:
- A **stent** should be fabricated prior to removal of a palatal torus
- A double-Y incision should be made over the midline of the torus
- After careful elevation of the flaps, the torus should be scored multiple times in the anterior, posterior, and transverse dimensions
- An **osteotome** can be used to remove each of these small portions
- A large bur or bone file is used to smooth the area
- After thorough irrigation, the wound is closed loosely with horizontal mattress sutures
- The stent is placed to prevent hematoma formation and to support the flap

Important: The maxillary torus **should not be excised en masse** to prevent entry into the nose *(the palatine bone will come out with torus).*

Other than mandibular and maxillary third molars, what is the next most commonly impacted permanent tooth?

- Maxillary canines
- Maxillary lateral incisors
- Mandibular first molars
- Mandibular premolars

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- **Maxillary canines**

Classifications of impactions → one system employs a description of the **angulation of the long axis** of the impacted third molar with respect to the long axis of the second molar:
- Mesioangular *(43% of all impacted teeth)*
- Distoangular *(6% of all impacted teeth)*
- Vertical *(38% of all impacted teeth)*
- Horizontal *(3% of all impacted teeth)*

***In addition,** teeth can also be angled in a buccal or lingual direction.
Note: Most mandibular third molars are angled toward the lingual direction.

Impactions are also classified based on the **relationship to bone and tissue:**
- **Soft tissue impacted** → impacted by soft tissue only
- **Partial bony impaction** → crown is partially covered by bone
- **Full bony impaction** → tooth completely covered by bone

The most common site for a **supernumerary tooth** is in the maxillary incisor area. When it occurs here, it is called a **mesiodens.** These teeth are usually small, peg-shaped, and do not resemble the teeth normal to the site. Treatment is surgical removal.

The **maxillary first molar** is innervated by the:

- **Anterior** superior alveolar and **middle** superior alveolar nerves
- **Middle** superior alveolar and **posterior** superior alveolar nerves
- **Posterior** superior alveolar and **inferior** alveolar nerves
- **Middle** superior alveolar nerves and **palatine** nerves

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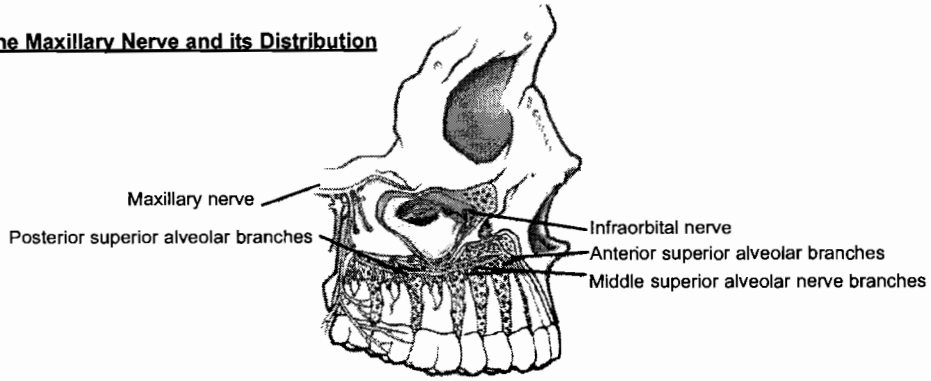
- **Middle superior alveolar and posterior superior alveolar nerves**

- The **posterior superior alveolar** *(PSA)* nerve block, otherwise known as the tuberosity block or the zygomatic block, is used to achieve anesthesia of the maxillary molar teeth up to the first molar with the exception of its mesiobuccal root.
- The **middle superior alveolar** *(MSA)* nerve block is useful for procedures where the maxillary premolar teeth or the mesiobuccal root of the first molar require anesthesia.
- The **anterior superior alveolar** *(ASA)* nerve block or infraorbital nerve block is a useful technique for achieving anesthesia of the maxillary central and lateral incisors and canine as well as the surrounding soft tissue on the buccal aspect.

Remember: In order to **extract** the maxillary first molar, you must numb both the **PSA** and **MSA** nerves as well as the greater *(anterior)* palatine nerve for palatal anesthesia *(soft tissue)*.

The Maxillary Nerve and its Distribution



Scopolamine can be used for which of the following?

- Treatment of minor pain or fever
- Preoperative medication to produce amnesia and decrease salivary and respiratory secretions
- Treatment of muscle spasm associated with acute TMJ pain
- Management of mild to severe hypertension

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- **Preoperative medication to produce amnesia and decrease salivary and respiratory secretions**

Mechanism of action → blocks the action of acetylcholine ***(anticholinergic agent)*** at parasympathetic sites in smooth muscle, secretory glands and the CNS; increases cardiac output, dries secretions, antagonizes histamine and serotonin.

Scopolamine is very effective for the **prevention of motion sickness** and this indication represents the most common clinical use. Because scopolamine depresses the central nervous system, it is used as a sedative prior to anesthesia and as an antispasmodic in certain disorders characterized by restlessness and agitation, e.g., delirium, tremors, psychosis, mania, and Parkinsonism.

When you **premedicate** a patient with Scopolamine, the following effects will be prolonged *(similar to premedication with Morphine)*:
- Amnesia
- Psychic sedation
- Decreased salivation

Notes:

1. **Reduction of secretions** occurs by competitive blockade of acetylcholine and other cholinergic stimuli at cholinergic receptors sites on salivary and bronchial glands.
2. Antagonism of acetylcholine on the sphincter and ciliary body in the eye, produces mydriasis *(dilation of pupils)*.

A patient who fractured his jaw in an automobile accident received an IM injection of 60 mg of meperidine. He subsequently developed a severe reaction characterized by tachycardia, hypertension, hyperpyrexia, and seizures. When questioned, the uninjured father revealed that the patient has been taking a drug for a psychiatric condition. Which of the following psychiatric drugs is **most likely** to be responsible for this untoward reaction with meperidine?

- Alprazolam
- Paroxetine
- Fluoxetine
- Phenelzine

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• **Phenelzine**

Monoamine oxidase *(MAO)* **inhibitors** are a class of drugs used for depression and Parkinson's Disease. Examples of MAO inhibitors include isocarboxazid *(brand name Marplan)*, phenelzine *(Nardil)*, tranylcypromine *(Parnate)*, and selegiline *(Eldepryl)*.

Mechanism of action → thought to act by increasing endogenous concentrations of norepinephrine, dopamine, and serotonin through inhibition of the enzyme *(monoamine oxidase)* responsible for the breakdown of these neurotransmitters.

Meperidine *(Demerol)* is a **potent narcotic analgesic** prescribed for the relief of moderate to severe pain, and as a cough suppressant. It is probably the most widely used narcotic in American hospitals. It compares favorably with morphine, the standard for narcotic analgesics. Meperidine is the **most abused drug** by health professionals.
Note: It produces slight euphoria but no miosis.

Important: Concomitant administration of meperidine and MAO inhibitors has resulted in life-threatening hyperpyrexic reactions that may culminate in seizures or coma.

Remember: Morphine is the standard drug to which all analgesic drugs are compared. It causes **euphoria,** analgesia, and drowsiness along with miosis and **respiratory depression.**

A surgical procedure used to recontour the supporting bone structures in preparation of a complete or partial denture is called a (*an*):

- Closed reduction
- Operculectomy
- Alveoloplasty
- Gingivoplasty

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• **Alveoloplasty**

An alveoloplasty is the surgical preparation of the alveolar ridges *(i.e., removing undercuts)* for the reception of dentures or shaping and smoothing the socket margins after extractions of teeth with subsequent suturing to insure optimal healing.

The **objectives** of this recontouring should be to provide the best possible tissue contour for prosthesis support, while maintaining as much bone and soft tissue as possible.

Remember:

1. A **closed reduction** is the closing of the space between fractured bone **without** cutting through the soft tissue or surrounding bone.
2. A **gingivoplasty** is a surgical procedure to **reshape the gingivae** to create a normal, functional form.
3. An **operculectomy** is the removal of the operculum, which is the flap of tissue over an unerupted or partially erupted tooth.

Cavernous sinus thrombosis may occur as a consequence of the hematogenous spread of a maxillary odontogenic infection via the venous drainage of the maxilla. The causative agent is generally:

- Staphylococcus aureus
- Streptococci
- Pneumococci
- Fungi

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• **Staphylococcus aureus**

A **cavernous sinus thrombosis** is a blood clot within the cavernous sinus, a large channel of venous blood in a cavity bordered by the sphenoid bone and the temporal bone of the skull. The cavernous sinus is an important structure because of its location and its contents which include the oculomotor nerve, the trochlear nerve, branches of the trigeminal nerve *(the ophthalmic and maxillary nerves)*, and the abducens nerve.

Infections of the face can cause a septic thrombosis of the cavernous sinus. Furunculosis and infected hair follicles in the nose are frequent causes. Extractions of maxillary anterior teeth in the presence of an acute infection and especially the curettage of the socket under such circumstances can cause this condition. The infected thrombus ascends in the veins against the usual venous flow. It usually occurs in the **ophthalmic vein.** This is possible because of the **absence of valves** in the angular, facial, and ophthalmic veins.

Patients present with:
• Proptosis *(exophthalmos)*
• Orbital swelling
• Neurologic signs
• Fever

The infection **is life-threatening** and requires prompt and aggressive treatment, consisting of elimination of the source of infection, drainage, parenteral antibiotic therapy, and neurosurgical consultation.

Which of the following techniques is best for a wide based frenectomy?

- Diamond excision
- V-Y advancement
- Z-plasty

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• **V-Y advancement**

When a **frenum** is positioned in such a way as to interfere with the normal alignment of teeth or results in pulling away of the gingiva from the tooth surface causing recession it is often removed using a surgical process known as a **frenectomy.**

There are **three surgical techniques** that are used for a frenectomy:

- **Diamond excision**
- **Z-plasty**

} are effective when the mucosal and fibrous tissue band is **relatively narrow.** These techniques **relax the pull** of the frenum.

- **V-Y advancement** is often preferred when the frenal attachment has a wide base. This technique is good for lengthening tissue and usually results in less scarring.

Note: Local anesthetic infiltration is usually sufficient for surgical treatment of frenal attachments. Care must be taken to **avoid excessive infiltration** directly in the frenum area since it may **obscure the anatomy** that must be visualized at the time of excision.

When a patient **attempts protrusion,** the mandible deviates markedly to the **right.** Which muscle is unable to contract?

- Buccinator muscle
- Temporalis muscle
- Right lateral pterygoid muscle
- Left lateral pterygoid muscle

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- **Right lateral pterygoid muscle** → with **lateral pterygoid injury,** the mandible will deviate **toward the side** of injury.

The mandible will also deviate **toward** the side of injury with:
- **Ankylosis** of the condyle → the most common cause of TMJ ankylosis is **trauma**
- A **unilateral** condylar fracture

The mandible will deviate **away** from the affected side with:
- Condylar **hyperplasia** → malocclusion is also a common occurrence with this injury

Remember: The lateral pterygoids *(right and left)* acting together are the **prime protractors** of the mandible. **Important:** In addition to opening and protruding, the lateral pterygoids move the mandible from **side to side.** For **right lateral** excursive movements, the **left lateral** pterygoid muscle is the prime mover and vice versa.

A patient who sustained a **subcondylar fracture** *(unilateral condylar fracture)* on the **left** side would be unable to deviate the mandible to the **right** *(as stated above the mandible will deviate to the side of injury with a unilateral condylar fracture, this patient would not be able to deviate the mandible to the right)* This is normally treated by a **closed procedure involving intermaxillary fixation.** This procedure immobilizes the concomitant fractures and corrects the displacement of the jaws associated with the condylar fracture thereby correcting the shift of the midline toward the side of the fractured condyle and the slight premature posterior occlusion on that side.

When would you place a suture over a **single extraction** socket?

- Routinely
- Never
- If the patient requests it
- When there is severe bleeding from the gingiva or if the gingival cuff is torn or loose

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- **When there is severe bleeding from the gingiva or if the gingival cuff is torn or loose**

Normal post-extraction procedure:

- All loose bone spicules and portions of the tooth, restoration, or calculus are removed from the socket as well as from the buccal and lingual gutters and the tongue.
- The socket must be **compressed by the fingers** to reestablish the normal width present before the buccal plate was surgically expanded. **Note:** The natural recontouring of the residual ridge occurs primarily by resorption of the labial-buccal cortical bone.
- **Sutures** are usually not placed unless the papillae have been excised.
- The socket is covered with a gauze sponge that has been folded and moistened slightly at its center with cold water.
- The patient is instructed to bite down on the pressure dressing for 30-60 minutes.
- A printed instruction sheet is given to the patient.
- A prescription for pain is given if the need is anticipated.

If bleeding persists for some time following an extraction, it may be helpful to instruct the patient to bite on a tea bag. The **tannic acid** in the tea bag will help promote hemostasis.

Remember: The most common cause of **post-extraction bleeding** is the failure of the patient to follow **post-extraction instructions.**

At what point should the EMS be activated with adult victims?

- After one minute of CPR
- After two minutes of CPR
- After three minutes of CPR
- Immediately when an adult is found to be unresponsive

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• **Immediately when an adult is found to be unresponsive**

***For an **infant or child victim** the EMS should be activated **after one minute of CPR**

Cardiopulmonary Resuscitation

A = Airway

- Place victim flat on his / her back on a hard surface.
- Shake victim at the shoulders and shout "are you okay?"
- If **no response,** call emergency medical system → **911** then,
- **Head-tilt/chin-lift** → open victim's airway by tilting their head back with one hand while lifting up their chin with your other hand.

B = Breathing

- Position your cheek close to victim's nose and mouth, look toward victim's chest, and
- **Look, listen, and feel** for breathing *(5-10 seconds)*
- If not breathing, pinch victim's nose closed and give 2 full breaths into victim's mouth
- If breaths won't go in, reposition head and try again to give breaths. If still blocked, perform abdominal thrusts *(Heimlich maneuver)*

C = Circulation

- Check for carotid pulse by feeling for 5-10 seconds at side of victim's neck.
- If there is a pulse but victim is **not breathing,** give **rescue breathing** at rate of **1 breath every 5-6 seconds** or **10-12 breaths per minute**
- If there is **no pulse,** begin chest compressions as follows:
  - Place heel of one hand on lower part of victim's sternum. With your other hand directly on top of first hand, depress sternum 1.5 to 2 inches.
  - Perform **15 compressions** to every **2 breaths** *(rate: 100 per minute)*.
  - Check for return of pulse every minute.

***Continue uninterrupted until advanced life support is available.**

Which of the following teeth could be removed **without pain** after administration of an **inferior alveolar** and **lingual** nerve block?

- **All anterior teeth** on the side of the injection
- **Canine** and **first premolar** on the side of the injection
- **All teeth** in that quadrant on the side of the injection
- Both **premolars** and **first molar** on the side of the injection

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• **Canine and first premolar on the side of the injection**

You need to give a **long buccal injection** in order to extract the molars and second bicuspid. For operative procedures, a long buccal injection **may not** be needed for these teeth. **Note:** The lingual nerve is usually anesthetized by depositing a few minims of solution while advancing the needle laterally and posteriorly parallel to the occlusal plane during an inferior alveolar block (*See picture below).* **Incisors may need local** infiltration for extractions.

Note: An injection into the parotid gland *(capsule)* when attempting to administer an inferior nerve block may cause a **Bell's palsy facial expression** → paralysis of the forehead muscles, the eyelid and of the upper and lower lips on the same side of the face that the injection was given.

Remember: The bone of the maxilla is **more porous** than that of the mandible, therefore, it can be infiltrated anywhere.

Which of the following TMJ ligaments prevents posterior and inferior displacement?

- Sphenomandibular ligament
- Temporomandibular ligament
- Stylomandibular ligament

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• **Temporomandibular ligament**

The **temporomandibular ligament** *(also called the lateral ligament)* runs from the articular eminence to the mandibular condyle. It provides lateral reinforcement for the capsule. This ligament **prevents posterior and inferior displacement** of the condyle *(it is the **main stabilizing ligament** of the TMJ)*. **Note:** This ligament keeps the head of the condyle in the mandibular fossa if the condyle is fractured.

The **sphenomandibular and stylomandibular ligaments** are considered to be **accessory ligaments.** The former is attached to the lingula of the mandible and the latter at the angle of the mandible. These ligaments are responsible **for limitation** of mandibular movements *(limit excessive opening)*.
Note: The **sphenomandibular ligament** is most often damaged in an inferior alveolar nerve block.

Collateral ligaments *(also referred to as "discal ligaments")* are ligaments that arise from the periphery of the disc, and are attached to the medial and lateral poles of the condyle respectively and stabilize the disc on the top of the condyle. These ligaments restrict **movement of the disc away** from the condyle during function.

Trismus caused by an inferior alveolar injection is the result of injecting below the mandibular foramen into the:

- Buccinator muscle
- Masseter muscle
- Temporalis muscle
- Medial pterygoid muscle
- Lateral pterygoid muscle

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• **Medial pterygoid muscle**

Limited jaw opening, or trismus, is a relatively common complication following local anesthetic administration. It can be caused by spasm of the muscles of mastication, which in turn may be a result of needle insertion into or through one of them. The most common muscle to be the source of trismus is the **medial pterygoid,** which can be penetrated during an inferior alveolar nerve block if the injection is performed well below the mandibular foramen. Rarely, the temporalis may be penetrated before it attaches onto the coronoid process if the needle is inserted too far laterally. Even more rarely, the lateral pterygoid muscle may be penetrated if a block is administered too far superiorly.

The main symptom of trismus is the **limitation of movement** of the mandible, which is often associated with pain. Symptoms will arise from one to six days following an injection. The duration of symptoms and their severity are both variable.

Management of trismus:
• Apply hot, moist towels to the site for approximately 20 minutes every hour
• Use analgesics as required
• The patient should gradually open and close mouth as a means of physiotherapy

Following an inferior alveolar nerve block injection or a mental block injection, a prickly or tingling sensation *(paresthesia)*, even complete numbness in the lower lip, may result and persist for a considerable time. This is usually considered to be **due to direct trauma** or piercing of the nerve trunk by the needle. This happens more often in the case of the mental block injection. The symptoms of paresthesia gradually diminish *(may last from two weeks to six months)*, and recovery is usually complete.

Remember: The most common cause of **paresthesia of the lower lip** is the removal of a **mandibular third molar** *(especially horizontally impacted ones)*.

The most common cause of loss of consciousness in the dental office is:

- Anaphylaxis
- Syncope
- Heart attack
- Seizure

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• **Syncope** → fainting

Syncope is the **most common** adverse reaction associated with administration of local anesthesia. **Remember:** It often occurs when upright, though can occur when sitting. It will never occur when lying. The patient may complain of feeling generalized **warmth with nausea** and **palpitations.**

The **initial event** in a vasovagal syncope episode is the stress-induced release of increased amounts of catecholamines that causes the following: a decrease in peripheral vascular resistance, tachycardia, and sweating.

As blood pools in the periphery, a drop in blood pressure appears, with a corresponding decrease in cerebral blood flow. The patient will then complain of feeling **dizzy or weak.** Compensatory mechanisms attempt to maintain adequate blood pressure, but they soon fatigue, which leads to **vagally mediated bradycardia.** Once the blood pressure drops below levels necessary to sustain consciousness, syncope occurs.

Place the patient in a **supine position** with the feet elevated *(Trendelenburg position),* monitor vital signs, tight clothing should be loosened and a cold compress placed on the forehead. Oxygen 3 - 4 L / minute should be given via nasal cannula.

Important: The single most important drug to use in any medical emergency, including chronic obstructive pulmonary disease, is oxygen.

Note: The primary airway hazard for an **unconscious** dental patient in a supine position is tongue obstruction. **Remember:** Head tilt / chin lift.

Which of the following will produce **neurolept** anesthesia?

- Neuroleptic agent + narcotic analgesic
- Neuroleptic agent + nitrous oxide
- Neuroleptic agent + narcotic analgesic + nitrous oxide
- Narcotic analgesic + nitrous oxide

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• **Neuroleptic + narcotic analgesic + nitrous oxide**

Neurolept anesthesia is a state of **neurolept analgesia** and **unconsciousness,** produced by the combined administration of a narcotic analgesic and a neuroleptic agent, together with the inhalation of nitrous oxide and oxygen.

Neurolept analgesia only produces an unconscious state **if** nitrous oxide is also administered *(see below).*

Neuroleptic agent + narcotic analgesic = **neurolept analgesia**
(Droperidol) *(Fentanyl)* *(conscious)*

Under the influence of this combination, the patient is sedated and demonstrates psychic indifference to the environment yet **remains conscious** and can respond to questions and commands.

Neurolept + nitrous oxide = **neurolept anesthesia**
analgesia in oxygen *(unconscious)*

Induction of anesthesia is slow, but consciousness returns quickly after the inhalation of nitrous oxide is stopped.

The maximum allowable dose for 2% lidocaine with 1:000,000 epinephrine is:

- 2.0 mg lidocaine / per lb.
- 3.2 mg lidocaine / per lb.
- 6.0 mg lidocaine / per lb.
- 7.0 mg lidocaine / per lb.

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• **3.2 mg lidocaine / per lb.** *(or 7 mg / kg)*

For example: 150 lb. *(or 70 kg)* patient x **3.2 mg** = 480 mg *(maximum allowable dosage)* ÷ **36 mg** *(amount of Lidocaine in 2% carpule)* = approximately 13.3 carpules *(approximately 13)*

Note: 1 kg = 2.2 pounds

For carbocaine **without epinephrine,** the maximum allowable dose is 3.0 mg / lb.

Remember:
The maximum allowable dose of **epinephrine** that can be administered to a **cardiac-risk patient** is 0.04 mg or 0.20 mg of levonordefrin. In terms of local anesthetics this would be equivalent to:

Maximum Allowable Vasoconstrictor for the Cardiac Patient			
Vasoconstrictor Concentration and Type	**Vasoconstrictor *(MG/ML)***	**Standard Dental Cartridge *(MG/1.8 CC)***	**Maximum Allowed Cartridges**
1:20,000 levonordefrin	0.5	0.09	2
1:50,000 epinephrine	0.02	0.036	1
1:100,000 epinephrine	0.01	0.018	2
1:200,000 epinepfrine	0.005	0.009	4

All of the following are cardinal signs of a localized osteitis *(dry socket)* **except:**

- Pain is worse than before extraction
- Throbbing pain *(often radiating)*
- Bilateral lymphadenopathy
- Fetid odor
- Bad taste
- Poorly healed extraction site

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- **Bilateral lymphadenopathy**

The etiology of dry socket is not absolutely clear but is thought to develop because of increased fibrinolytic activity causing accelerated lysis of the blood clot. It is **most common** following extraction of the **mandibular molars.** Smoking, premature mouth rinsing, hot liquids, surgical trauma, and oral contraceptives all have been implicated in the development of a dry socket. **Note:** Careful technique and minimal trauma reduce the frequency of patients developing dry socket.

Treatment for dry socket:

- Flush out debris with slightly warmed saline solution → **gently !!!**
- Place a sedative dressing in socket *(eugenol).* The dressing should be removed within 48 hours and replaced until the patient becomes asymptomatic. **Note:** (1) The gauze provides an attachment for the obtundent paste so it stays in the socket (2) **Eugenol** is the active component in most sedative dressings.
- Analgesics should be prescribed if necessary.

***Antibiotics are generally not indicated

Notes:

1. Dry socket is the **most common complication** seen after the surgical removal of a mandibular molar.
2. Curetting a dry socket can cause the condition to worsen because healing will be further delayed, any natural healing already taking place will be destroyed, and there is a risk of causing the localized inflammatory process to be spread to the adjacent sound bone.

The **ideal time** to remove impacted third molars is:

- When the root is **fully** formed
- When the root is **approximately two-thirds** formed
- Makes **no difference** how much of the root is formed
- When the root is approximately **one-third** formed

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- **When the root is approximately two-thirds formed**

  — Patient would be around the age of 17-21.
  — **At this time,** the bone is more flexible and the roots are not formed well enough to have developed curves and rarely fracture during extraction.

When the root is **fully formed,** the possibility increases for abnormal root morphology and for fracture of the root tips during extraction.

If the root development is **insufficient** *(one-third or less)*, the tooth is more difficult to remove because it tends to roll in its crypt like a ball in a socket, **which prevents** easy elevation.

Notes:

1. Patients who are **young tolerate surgery very well.** Postoperative complications are usually minimal.
2. Older individuals have the **most postoperative difficulties.** The bone is more dense and usually the patient responds more slowly to the entire process *(anesthesia and surgery).*

A person who has been on **suppressive doses of steroids** will:

- **Never regain** full adrenal cortical function
- Take as much as a **year** to regain full adrenal cortical function
- Take as little as a **week** to regain full adrenal cortical function
- Take usually a **couple of days** to regain full adrenal cortical function

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- **Take as much as a year to regain full adrenal cortical function**

The following guidelines may help determine if a patient's adrenal function is suppressed, **however,** if any **doubt exists,** consult the patient's physician **before** performing surgery.

Some Guidelines:

- People on **small doses** *(5 mg Prednisone / day)* will have suppression when they have been on the regimen for a month.
- People taking equivalence of 100 mg cortisol / day *(20-30 mg Prednisone / day)* will have abnormal cortical function in a **week.**
- **Short-term therapy** *(1-3 days)* of even high dose steroids **will not** alter adrenal cortical function.
- A person who has been on **suppressive doses of steroids** will take as much as a **year** to regain full adrenal cortical function.

Remember: Stress or fatigue may cause an **adrenal crisis** in a patient that has suppressed adrenal function. Patients may experience lightheadedness or dizziness, weakness, sweating, abdominal pain, nausea and vomiting, or even loss of consciousness.

In adrenal crisis, an intravenous or intramuscular injection of hydrocortisone must be given immediately. Supportive treatment of low blood pressure with intravenous fluids is usually necessary. Hospitalization is required for adequate treatment and monitoring.

COPD *(Chronic Obstructive Pulmonary Disease)* is an umbrella term used to describe lung disease associated with **airflow obstruction.** Most generally, emphysema, chronic bronchitis and chronic asthma either alone or in combinations fall into this category. Patients with which disorder have a **barrel-chested appearance?**

- Chronic asthma
- Chronic bronchitis
- Emphysema

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• **Emphysema**

***This results from the fact that in these patients the distal air spaces become enlarged, and the lungs become hyperinflated.

There is continuing debate as to whether this term also includes acute asthma, however as a general rule, it is not included as, even though it does have obstructive components to it, it is in part reversible, and is more generally considered a restrictive lung disease. In most cases, bronchitis and emphysema occur together. **Note:** Secondary pulmonary hypertension is most often caused by COPD.

Chronic obstructive pulmonary disease:

- Chronic asthma → dyspnea and wheezing expiration caused by episodic narrowing of the airways.
- Chronic bronchitis → is the inflammation and eventual scarring of the lining of the bronchial tubes. When the bronchi are inflamed and / or infected, less air is able to flow to and from the lungs and a heavy mucus or phlegm is coughed up. The condition is defined by the presence of a mucus-producing cough most days of the month, three months of a year for two successive years without other underlying disease to explain the cough.
- Emphysema → often coexists with chronic bronchitis, it begins with the destruction of air sacs *(alveoli)* in the lungs where oxygen from the air is exchanged for carbon dioxide in the blood. The walls of the air sacs are thin and fragile. Damage to the air sacs is irreversible and results in permanent "holes" in the tissues of the lower lungs. As air sacs are destroyed, the lungs are able to transfer less and less oxygen to the bloodstream, causing shortness of breath. The lungs also lose their elasticity, which is important to keep airways open. The patient experiences great difficulty exhaling.

Important:

1. Drugs with **antiplatelet activity** *(aspirin)* should be prescribed to COPD patients **with caution.** Hemoptysis has been reported after the use of aspirin in patients with COPD.
2. COPD patients taking theophylline **should not be** prescribed erythromycin. Erythromycin increases the metabolism of theophylline and may cause toxicity.

Which of the following occurs when mucus or a foreign object obstructs airflow in a main-stem brochus causing collapse of the affected lung tissue into an airless state?

- Bronchiectasis
- Atelectasis
- Pneumothorax
- Pneumonia

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• **Atelectasis**

Atelectasis typically occurs 36 hours postoperatively and presents with mild dyspnea, low-grade fever, and hypoxia. **Note:** Prolonged atelectasis can lead to **pneumonia.**

The **treatment** of postoperative atelectasis is aimed at expansion of the lung, and, for most patients, incentive spirometry *(encouraging the patient to take long, slow, deep breaths)* is adequate. However, in patients with severe atelectasis, endotracheal suction and even bronchoscopy may be warranted.

Pneumothorax occurs when air leaks into the pleural space causing the lung to recoil from the chest wall. In an awake patient, a pneumothorax typically presents with dyspnea, chest pain, absence of breath sounds on the affected side, and evidence of pneumothorax on a chest x-ray. Tracheal deviation may be present.

The objective of **treatment** for a pneumothorax is to remove the air from the pleural space, allowing the lung to re-expand. In an emergency, a small needle *(such as a standard intravenous needle)* may be placed into the chest cavity through the ribs to relieve the excessive pressure. The definitive treatment is a chest tube, a large plastic tube that is inserted through the chest wall between the ribs to remove the air completely.

Notes:

1. Pneumonitis *(inflammation of the lung)* and atelectasis are two of the most common causes of fever in a patient who has had **general anesthesia.**
2. The most common **post-op complication** of outpatient general anesthesia is nausea.

Alloplastic chin implants and sliding genioplasty represent the two currently accepted methods of chin augmentation. Which procedure has been shown to have the best long-term results for enlargement of the prominence of the chin?

- Alloplastic chin implants
- Sliding genioplasty

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• **Sliding genioplasty** → also called a horizontal sliding osteotomy

Genioplasty is a type of cosmetic surgery that is used to improve the appearance of a person's chin. This can take the form of chin height reduction or chin rounding by osteotomy, or chin augmentation using implants.

A particular process, known as a **sliding genioplasty,** involves removing a horseshoe-shaped piece of the chin bone and sliding it either backwards or forwards, finally fixing it in place using titanium screws.

Problems that are frequently encountered when using **alloplastic materials** for genioplasty:

1. **Migration from** the position in which they were placed at the time of surgery
2. **Erosion of the chin prominence** contiguous with the implant
3. **Unpleasant sensation** in the implant region when exposed to cold temperatures

Remember: Alloplastic grafts are inert, man-made synthetic materials. The modern artificial joint replacement procedures use metal alloplastic grafts. For **bone replacement** a man-made material that mimics natural bone is used. Most often this is a form of calcium phosphate *(i.e., tricalcium phosphate, calcium carbonate,* or **hydroxyapatite***).*

Incision for drainage *(I & D)* in an area of acute infection should only be performed **after:**

- A culture for antibiotic sensitivity has been performed
- Localization of the infection
- A sinus tract is formed
- The patients fever has cleared up

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• **Localization of the infection**

Physiologically, it is at this time that nature has constructed a barrier around the abscess, walling it off from the circulation and making it possible to palpate the presence of purulent material within the abscess cavity *(known as fluctuance).*

Note: After you incise and drain the fluctuant mass, it may be prudent to do a **culture** for antibiotic sensitivity. This should always be done if after incision and drainage the swelling **does not** subside despite large doses of antibiotics.

Prior to actual abscess formation, however, the infection is capable of producing a cellulitis in the soft tissues of the region involved. The palpable tissues take on a condition known as **induration** *(they appear hard, dense, and brawny).* Treatment during this period should be directed towards **localizing** the infection. Early employment of antibiotics may be extremely important in a severe and life-threatening infection. Localization of the infection may be aided by using warm compresses and warm mouth rinses at frequent intervals.

The most commonly used **allogeneic bone** is:

- Freeze-dried
- Demineralized freeze-dried bone
- Fresh frozen

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- **Freeze-dried**

Allogeneic bone is nonvital, osseous tissue harvested from one individual and transferred to another of the same species. Three forms of allogeneic bone include: fresh frozen, freeze-dried, and demineralized freeze-dried bone. Fresh frozen bone, however, is rarely used due to the concern related to transmission of disease.

Freeze-dried bone is osteoconductive, however, it has no osteogenic or osteoinductive capabilities. Freeze-dried allogeneic grafts are usually placed in conjunction with autogenous grafts.

Demineralized freeze-dried allogeneic bone lacks mechanical strength, but has osteoconductive and osteoinductive capabilities. Demineralizing the freeze-dried bone exposes the bone morphogenetic proteins which has been shown to induce bone formation.

The **advantages** of allogeneic bone grafts are that it doesn't require another site of preparation in the host and that a similar bone or a bone of similar shape to that being replaced can be obtained.

The three processes by which **bone can be repaired or regenerated** are:

- **Osteogenesis** (*osteogenic potential)* is the ability to form new bone in a graft by transplanting viable osteoblasts.
- **Osteoconduction** is the ability of the graft to allow vascular and cellular invasion by the host site.
- **Osteoinduction** is the ability of the graft to stimulate differentiation of mesenchymal cells into osteoblasts at the recipient site.

All of the following **may prevent** a patient from developing a vasovagal syncopal reaction **after** the use of a local anesthetic **except:**

- **Slowly injecting** the anesthetic solution
- Watching the patient's **color change** during the injection
- Using a **topical anesthetic** prior to administration of the local anesthetic
- Injecting the anesthetic solution **as quickly as possible**
- Using a low concentration of **vasoconstrictor**
- **Premedicating** extremely anxious patients
- Sympathetic, **but** confident handling of the patient

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• **Injecting the anesthetic solution as quickly as possible**

Vasovagal *(neurocardiogenic)* **syncopal** reactions are not uncommon in the setting of dental procedures. Characteristically, hypotension from vasovagal syncope is accompanied by bradycardia, sweating, and **pallor.** Symptoms such as tightness in the throat and dyspnea are also common. **Note:** Prevention of vasovagal syncopal reactions **involves** proper patient preparation.

Remember: Any signs of an impending syncopal episode should be quickly treated by placing the patient in a **supine position** with the feet elevated *(Trendelenburg position),* monitoring vital signs, loosening tight clothing and placing a cold compress on the forehead. Oxygen 3 - 4 L / minute should also be given via nasal cannula.

Important: The most common **early sign** of syncope is **pallor.**

The most common cause of a **transient loss of consciousness** in the dental office is **vasovagal syncope.** This generally is due to a series of cardiovascular events triggered by the emotional stress brought on by the anticipation of or delivery of dental care.

ORAL SURGERY & PAIN CONTROL

In **children,** the mandible and maxilla are more elastic because of a high cancellous-to-cortical bone ratio. As a result, more _____ fractures are seen.

- Simple
- Greenstick
- Compound
- Comminuted

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Greenstick

The following categories classify mandibular fractures by describing the condition of the bone fragments at the fracture site and possible communication with the external environment:

- **Simple** → divides a single bone into two distinct parts with no external communication. These are **closed fractures** with no lacerations of the oral mucosa or facial tissues.
- **Compound** → fracture communicates with the outside environment *(open fracture)*. This may occur by laceration of the oral tissues exposing the bone fragments, fracture of the maxilla into the sinuses, or by way of skin lacerations that would expose the fracture segments. **Infection is common.**
- **Comminuted** → are multiple fractures of a single bone. They may be **simple** or **compound.**
- **Greenstick** → fracture that extends only through the cortical portion of the bone without complete fracture of the bone. Greenstick fractures are **closed fractures** involving incomplete fractures with flexible bone. **Most often seen in children.**

Remember: The most common complication of an open fracture is **infection.**

In an **adult,** the location of facial fractures is influenced by both the resistance of the bone to fracture and how prominent its position on the facial skeleton is. Adult facial fractures are most commonly seen in the nasal bones followed by the zygoma, mandible, and maxilla. In **children,** early growth in the cranium and orbits predisposes young children to frontal bone and orbital fractures.

What determines whether muscles will **displace fractured segments** from their original position?

- Attachment of the muscle
- Type of fracture
- Direction of muscle fibers
- Line of fracture

The **line of fracture** will determine whether muscles will be able to **displace the fractured segments** from their original position:

- If the fracture line **prevents** the displacement of the fracture by muscle pull, it is termed a **favorable fracture.**
- If the fracture line **results** in a muscle pull displacing the fracture segment, it is termed an **unfavorable fracture.**

The following **radiographic views** are often helpful to evaluate mandibular fractures:

- Posteroanterior view
- Lateral oblique view
- Towne view
- Panoramic view

Remember: The **control of airway** is vital to any treatment of a patient with facial fractures.

Muscles involved in **displacing mandibular fractures** include the medial and lateral pterygoid, temporalis, masseter, digastric, geniohyoid, genioglossus, and mylohyoid. The lateral pterygoid displaces the condyle anteriorly and medially because of its insertion on the pterygoid fovea. Muscles attached to the ramus *(i.e., temporalis, masseter, and medial pterygoid)* result in superior and medial displacement of the proximal segment. As fractures progress anteriorly toward the cuspid region, the digastric, geniohyoid, genioglossus, and mylohyoid exert a posterior-inferior force on the distal segment.

Which of the following factors would have the greatest **negative** influence on a dental implant's success?

- Hypertension
- Patient over 70 years of age
- Smoking
- Alcohol
- Post by-pass surgical patient

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• **Smoking**

***Because smoking affects the healing of bone and overlying tissue, it should be considered a relative contraindication to implant placement.

Any toothless area can be considered for dental implants. Determining whether implants are an option and the type of implants to use include: the patient's requirements and expectations, the amount of additional work needed *(i.e., bone grafting)*, the dentist's skill level, and the long-term prognosis.

Some indications for implant placement:
• Fixed restoration of single or multiple teeth in a partially edentulous jaw
• Retention of a removable prosthesis in a partially edentulous jaw
• Retention of a prosthesis in a completely edentulous jaw
• Retention of a fixed prosthesis in a completely edentulous maxilla or mandible

Important: In patients with uncontrolled systemic diseases such as diabetes, immunocompromised patients, and patients with bleeding disorders, implant placement should be considered with **extreme caution.**

Remember:
1. Implants placed in the maxillary anterior region have the **highest failure rate.**
2. **Mobility** of the implant is regarded as the **most common sign** of implant failure.
3. A dental implant supported prosthesis should fit **passively** on the dental implant.
4. The **minimal interdental** tooth distance recommended for one dental implant is **10 mm.**
5. The **maximum amount of taper** to allow for proper draw on an overdenture attachment such as an "O" ring is **15 degrees.**

All of the following local anesthetics are amides **except:**

- Prilocaine
- Bupivacaine
- Lidocaine
- Procaine
- Mepivacaine
- Etidocaine

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• **Procaine** → is the prototype ester local anesthetic used

An **easy way** to identify amide local anesthetics is to remember that the drug name contains an **i plus - caine** *(lidocaine, mepivacaine, and bupivacaine).* Esters such as procaine, benzocaine, and tetracaine contain no i.

PABA Esters	Nonester Group: Amides
• Procaine *(Novocaine)*	• Lidocaine *(Xylocaine)* → most **commonly** used
• Tetracaine *(Pontocaine)*	• Prilocaine *(Citanest)*
• Propoxycaine *(Ravocaine)*	• Mepivacaine *(Carbocaine)*
• Benzocaine *(Monocaine)*	• Bupivacaine *(Marcaine)*
• Cocaine	• Etidocaine *(Duranest)*

Amides are safe, versatile, and effective local anesthetics. If hypersensitivity to a drug in this group precludes its use, one of the ester-compound local anesthetics may provide analgesia without adverse effect.

Esters are potent local anesthetics slightly different in chemical structure from the amide group. **Tetracaine is most commonly used.** Allergic reactions are **far more common with esters.**

Important: The local anesthetics lidocaine, prilocaine and etidocaine are recommended for the pregnant and lactating patient.

Remember: The drug of choice in management of an acute allergic reaction involving bronchospasm *(an acute narrowing of the respiratory airway)* and hypotension is **epinephrine.**
Note: Allergic reactions to local anesthetic are usually caused by an **antigen-antibody** reaction.

The parotid gland receives its parasympathetic secretomotor innervation from the:

- Glossopharyngeal nerve
- Trigeminal nerve
- Vagus nerve
- Facial nerve

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• **Glossopharyngeal nerve**

The nerve fibers form the lesser superficial petrosal nerve which leaves the skull through the foramen ovale together with the mandibular nerve *(V-3)*. These preganglionic fibers synapse in the **otic ganglion,** then join the **auriculotemporal nerve** *(a branch of V-3)* to be distributed to the gland. Sympathetic fibers arrive at the gland through its blood supply.

The parotid gland is the **largest** of the salivary glands and is a **purely serous** gland. The parotids are located below and just anterior to the ear. They are divided into deep and superficial lobes with the **stylomandibular tunnel** *(which encloses the facial nerve)* being the dividing line. Therefore, a portion of the parotid lies superficial to the mandibular ramus, and another portion lies deep.

The parotid gland is drained by **Stenson's duct,** which pierces the buccinator muscle and crosses the masseter muscle where it opens into the vestibule of the mouth **opposite the maxillary second molar.**

The **external carotid artery** and its terminal branches within the gland, namely the superficial temporal and the maxillary arteries, supply the parotid gland. The **lymphatic drainage** of the parotid gland is through the parotid nodes to the **superior deep jugular lymph nodes.**

Notes:

1. **Mumps** is a viral disease of the parotid gland. **Parotitis** is the inflammation of the parotid gland.
2. **Von Ebner's glands** are the only **other** adult salivary glands which are purely serous.

All of the following statements are true concerning Methohexital *(Brevital)* **except:**

- It is somewhat less lipid soluble and less ionized at physiologic pH than thiopental *(Pentothal)*
- An induction dose of 1-2 mg / kg produces loss of consciousness in less than 20 seconds and recovery in 4-5 minutes
- The elimination half-life is 3 hours, which allows a clearance rate that is 3 - 4 times faster than that of thiopental *(Pentothal)*
- It is classified as an intermediate-acting barbiturate

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- **It is classified as an intermediate-acting barbiturate**

Barbiturates exhibit a dose-dependent CNS depression with hypnosis and amnesia. They are very lipid soluble, which results in a rapid onset of action. They are used most often for induction of anesthesia because they produce unconsciousness in less than 30 seconds.

Barbiturates:

- Ultrashort acting → Methohexital *(Brevital)*, thiopental *(Pentothal)*, and thiamylal *(Surital)*
- Short and intermediate acting → Amobarbital *(Amytal)*, pentobarbital *(Nembutal)*, secobarbital *(Seconal)*, and Butabarbital *(Fioricet, Fiorinal)*
- Long acting → Phenobarbital *(Luminal)*

Most commonly used barbiturates for **induction of anesthesia:**

- Thiopental *(Pentothal)* → Usually prepared as a 2.5% solution. An induction dose of 3-5 mg / kg produces a loss of consciousness within 30 seconds and recovery in 5-10 minutes. Because the elimination half-life is 6-12 hours, patients may experience a slow recovery. When injected intravenously, it can be irritating.
- Methohexital *(Brevital)* → in addition to the true statements on the front of the card → is metabolized in the liver and excreted by the kidney. Brevital **induction** is characterized by being **rapid** and there is also **rapid recovery.** The side effect most often seen is hiccoughs. This is believed to be caused by rapid injection of the Brevital.

Notes:

1. The **most effective agent** in the initial treatment of **respiratory depression** due to the over dose of barbiturates is **oxygen under positive pressure.**
2. A primary advantage of IV sedation is the **ability to titrate individualized dosage.**

ORAL SURGERY & PAIN CONTROL — Disord / Cond

Match the term on the left with the correct meaning on the right.

• Apnea	Below normal CO_2 in arterial blood
• Hypercapnea	Abnormally deep and rapid breathing
• Hypocapnea	Increased pulmonary ventilation in excess of metabolic requirements
• Dyspnea	Permanent cessation of breathing *(unless corrected)*
• Respiratory arrest	Transient cessation or absence of breathing
• Hyperapnea	Excess CO_2 in arterial blood
• Hyperventilation	Underventilation in relation to metabolic requirements
• Hypoventilation	The unpleasant sensation of difficulty in breathing

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- Apnea → Transient cessation or absence of breathing
- Hypercapnea → Excess CO_2 in arterial blood
- Hypocapnea → Below normal CO_2 in arterial blood
- Dyspnea → The unpleasant sensation of difficulty in breathing
- Hyperapnea → Abnormally deep and rapid breathing
- Respiratory arrest → Permanent cessation of breathing *(unless corrected)*
- Hyperventilation → Increased pulmonary ventilation in excess of metabolic requirements
- Hypoventilation → Underventilation in relation to metabolic requirements

Notes:

1. **Hyperventilation** results in the loss of carbon dioxide *(CO_2)* from the blood *(hypocapnea)*, thereby causing a decrease in blood pressure and sometimes fainting.
2. **Hypoventilation** results in an increased level of carbon dioxide *(CO_2)* in the blood *(hypercapnea)*.

How will a **larger than normal** functional residual capacity affect nitrous oxide sedation?

- Nitrous oxide sedation will happen **much quicker**
- Nitrous oxide sedation will **take longer**
- Functional residual capacity **does not effect** nitrous oxide sedation

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- **Nitrous oxide sedation will take longer**

The **functional residual capacity** is the amount of air remaining in the lungs at the end of the normal expiration. **Note:** This air is used to provide air to the alveoli, which will aerate the blood evenly between breaths.

Note: Pulmonary volumes and capacity are about 20 to 25% less in females than in males and are **greater in large** and **athletic persons.** Nitrous oxide sedation will vary accordingly.

Respiratory air volumes during rest and exercise are of physical and clinical interest and they can be measured using a spirometer. The **main volumes** of interest are:

- **Tidal Volume** (TV) → amount of air breathed in and out during quiet breathing
- **Expiratory Reserve Volume** (ERV) → amount of air forced out of the lungs in a maximal expiration, over and above that expired in normal breathing
- **Inspiratory Reserve Volume** (IRV) → amount of air inhaled in a maximal inspiration, over and above that inhaled in normal breathing
- **Vital Capacity** (VC) → TV + ERV + IRV
- **Residual Volume** (RV) → volume of air that remains in the lungs at all times *(can't be measured by spirometry)*
- **Total Lung Capacity** (TLC) → VC + RV

Congestive heart failure *(CHF)* results from impaired pumping ability by the heart. A ventricular ejection fraction below _____ is indicative of CHF.

- 50%
- 65%
- 80%
- 90%

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- **50%**

Causes of CHF include MI, ischemic heart disease, poorly controlled hypertension, structural heart defects, and cardiomyopathy. **Note:** Usually the **left ventricle fails first,** soon followed by right-sided failure.

Clinical signs and symptoms of CHF → fatigue or weakness *(often the earliest symptom of CHF)*, shortness of breath with or without activity, orthopnea, or difficulty breathing while lying flat, rapid or irregular pulse, edema or swelling of legs, feet and ankles, a chronic dry or frothy cough that may be blood-tinged or resemble foam, nocturia, or an increase in urination at night. **Note:** The most common sign of left-sided heart failure is pulmonary edema, whereas right-sided heart failure causes pedal edema or abdominal swelling.

Classes of drugs used in the treatment of CHF:
- **ACE inhibitors** *(e.g., captopril, lisinopril)* and **vasodilators,** which dilate the blood vessels and reduce the heart's workload.
- **Beta blockers** *(e.g., carvedilol, bisoprolol, metoprolol, atenolol)*, which reduce arrhythmias and improve the left ventricle's mechanical efficiency.
- **Inotropes** *(e.g., digoxin)*, which increases the strength of cardiac contractions and reduces symptoms.
- **Diuretics** *(e.g., lasix, aldactone, zaroxolyn)*, which eliminate water and sodium through the kidneys and reduce edema and shortness of breath.

Patient treatment and dental management considerations:
- **Prolonged rest**
- Administration of **oxygen**
- Digitalis *(patients are* **prone** *to nausea and vomiting)*
- Diuretics / vasodilators *(patients are* **prone** *to orthostatic hypotension;* **avoid excessive epinephrine***)*
- Dicumarol *(patients may have* **bleeding problem***)*

In preparing the edentulous mandible for dentures, each of the following may be safely excised **except:**

- A labial frenum
- A lingual frenum
- The mylohyoid ridge
- The genial tubercles
- An exostosis

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• **The genial tubercles**

The genial tubercles are **situated** on the lingual surface of the mandible at a point about midway between the superior and inferior borders. There are four of them, two of which are situated on each side and adjacent to the symphysis. Although usually relatively small, they may be fairly large and extend outward from the surface as spinous processes. These tubercles are the **area of muscle attachment** for the suprahyoid muscles.

Important: If the genial tubercles were **removed**, the tongue would be **flaccid**.

Notes:

1. When removing the **mylohyoid ridge,** be careful to **protect the lingual nerve.**
2. When removing a **mandibular exostosis** *(mandibular torus)* it is recommended that an envelope flap design, which has no vertical components, be used.

During extraction of a maxillary third molar, you realize the **tuberosity** has also been extracted. What is the proper treatment in this case?

- Remove the tuberosity from the tooth and reimplant the tuberosity
- Smooth the sharp edges of the remaining bone and suture the remaining soft tissue
- No special treatment is necessary
- None of the above

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- **Smooth the sharp edges of the remaining bone and suture the remaining soft tissue**

A fracture of the maxillary tuberosity **most commonly results from** extraction of an **erupted maxillary third molar** — or a second molar if it happens to be the last tooth in the arch.

If the tuberosity is fractured **but intact,** it should be manually **repositioned** and stabilized with sutures.

The complications most often seen after extraction of an **freestanding, isolated maxillary molar** are:
- Fracture of the **tuberosity**
- Alveolar process fracture

Important →→→ "Beware of the lone molar" → it is often ankylosed to the bone.
Remember: The ankylosed tooth emits an atypical, sharp sound on percussion.

Key point to remember: Tuberosity fractures may occur and should be treated at the time of surgery. If the operator is unable to do this he / she must arrange an **immediate referral.**

The **pterygomandibular raphe** serves as an insertion point for the:

- Superior pharyngeal constrictor and buccinator muscles
- Temporalis and masseter muscles
- Buccinator and masseter muscles
- Medial and lateral pterygoid muscles

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• **Superior pharyngeal constrictor and buccinator muscles**

On each side, the **pterygomandibular raphe** passes between the tip of the hamulus of the pterygoid bone and the internal surface of the mandible at a point just postero-superior to the posterior limit of the mylohyoid ridge. It is formed by the union of the tendinous ends of the superior constrictor of the pharynx and the buccinator muscle. **Note:** As the mandible moves relative to the hamulus, the length of the raphe is passively increased.

The arrow points to the pterygomandibular raphe.

Position of needle for inferior alveolar nerve block

Notes:

1. The **buccinator muscle** is pierced by the needle when performing an inferior alveolar nerve block.
2. The deep tendon of the temporalis muscle and the superior pharyngeal constrictor muscle form a **V-shaped landmark** for an inferior alveolar nerve block.
3. When draining purulent exudate from an **abscess of the pterygomandibular space** from an intraoral approach, the **buccinator** muscle is most likely to be incised.

Which of the following barbiturates is **not** ultrashort-acting?

- Thiamylal *(Surital)*
- Thiopental *(Pentothal)*
- Amobarbital *(Amytal)*
- Methohexital *(Brevital)*

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• **Amobarbital *(Amytal)***

Barbiturates depress neuronal activity in the midbrain reticular formation, facilitating and prolonging the inhibitory effects of GABA and glycine.

Ultrashort-acting barbiturates:
- Thiopental *(Pentothal)*
- Thiamylal *(Surital)*
- Methohexital *(Brevital)*

Short-acting barbiturates:
- Pentobarbital *(Nembutal)*
- Secobarbital *(Seconal)*

Intermediate-acting barbiturates:
- Amobarbital *(Amytal)*
- Butabarbital *(Fioricet, Fiorinal)*

Long-acting barbiturates:
- Phenobarbital → generally not used in oral surgery

Important: Barbiturates are **contraindicated** in patients with **respiratory disease** or those who are pregnant.
Note: Physical dependence is likely to develop with barbiturates if abused. The dependence has a strong psychological as well as physical basis. Sudden withdrawal from high doses can be **fatal.**

Which of the following is a relatively safe antipyretic drug with **no** anti-inflammatory action?

- Aspirin
- Ibuprofen
- Acetaminophen
- Naproxen

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- **Acetaminophen**

Acetaminophen *(Tylenol)* is the only over-the-counter non-anti-inflammatory analgesic commonly available in the USA. It is a weak cyclooxygenase inhibitor in peripheral tissues, thus accounting for its lack of anti-inflammatory effect. It may be a more effective inhibitor of prostaglandin synthesis in the CNS, resulting in analgesic and antipyretic action. The combination of acetaminophen and propoxyphene (called **Darvocet-N** or **Wygesic**) is used to treat moderate to severe pain due to dental procedures. **Note: Propoxyphene** (*Darvon)* is an oral synthetic opioid analgesic structurally similar to methadone. Darvon compound-65 is a combination of aspirin, caffeine, and propoxyphene.

Notes:

1. **Acetaminophen does not** effect clotting time as does aspirin → it does not have significant antiplatelet effects. It is effective for the same indications as intermediate-dose aspirin. It is therefore useful as an aspirin substitute, especially in children with viral infections *(who are at a risk for Reye's syndrome if they take aspirin)*.
2. **Aspirin** is an anti-inflammatory, antipyretic and analgesic that is used to relieve headaches, toothaches, minor aches and pains, and to reduce fever. The GI tract rapidly absorbs it.
3. **Talwin compound** combines the strong analgesic properties of **pentazocine** and the analgesic, anti-inflammatory, and fever-reducing properties of aspirin. It is used for the relief of moderate pain. It **does not** produce euphoria.
4. The most appropriate time to administer the initial dose of an analgesic to control postoperative pain is **before** the effect of the local anesthetic wears off.
5. The following analgesics **should be avoided** in patients with renal disease: aspirin, acetaminophen, NSAID's, meperidine and morphine.

Which of the following tests should be **routinely performed in the preoperative workup** for a patient that is being admitted to a hospital for surgery?

- A complete blood count *(CBC)*
- A total white blood cell count
- An assessment of the circulating platelets
- A urinalysis
- All of the above

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- **All of the above**

Routine Admission Tests

- A **complete blood count** that includes an evaluation of the **hemoglobin** and **hematocrit** indices

- A **total white blood cell count** with a differential count

- A gross and microscopic **urinalysis**

***Anyone scheduled for general anesthesia should have a **chest x-ray** and patients over 40 years old should also have an **E.K.G.**

Factors to be considered in the decision to hospitalize a patient for an **elective procedure:**

- Medical problems compromising treatment *(diabetes, hemophilia, etc.)*

- Difficulty and extent of surgery

- Consideration of the individual patient *(emotionally disturbed, handicapped, etc.)*

- Cost of hospitalization *(time and money)*

Why is a conventional handpiece that expels forced air **contraindicated** when performing dentoalveolar surgery?

- Too much bone will be removed
- These handpieces can cause tissue emphysema or an air embolus which can be fatal
- These handpieces are not high-powered enough to remove bone
- All of the above

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• **These handpieces can cause tissue emphysema or an air embolus which can be fatal**

Very important: Most high-speed turbine drills used in routine restorative dentistry are **totally unacceptable** for oral surgery. The air exhausted from these drills goes into the wound and may be forced deeper into tissue planes and produce **tissue emphysema,** a potentially dangerous situation.

Rongeur forceps are the most commonly used **instruments** for removing bone. However, the technique that most oral surgeons use when removing bone is the **bur and handpiece.**

Irrigation of the surgical wound during and after the cutting of bone cannot be emphasized enough. Copious amounts of coolant spray are crucial in minimizing osseous necrosis caused by heat generated from the bur. Irrigation serves also to cleanse the crypt and areas beneath the flap of bony debris, tooth fragments, and blood. Distilled water is **not used for irrigation** because it is a hypotonic solution and will enter cells down the osmotic gradient causing cell lysis and rapid death of bone cells.

Note: An acute infected tissue emphysema is usually caused by the **indiscreet use** of:

1. **Air-pressure syringes** → In drying out a root canal with a compressed air syringe, septic material may be forced through the apical foramen into the cancellous portion of the alveolar process and ultimately out through the nutrient foramina into adjacent soft tissues, resulting in formation of a septic cellulitis and tissue emphysema.
2. **Atomizing spray bottles activated by compressed air** → A similar condition can be induced by the use of a compressed-air spray bottle for irrigation of wounds, particularly in the retromolar region. It is safer to use a hand-activated syringe when irrigating wounds or dry ing root canals since it is unlikely that a tissue emphysema would be produced under these circumstances.

Which cardiac condition **would not** require preoperative antibiotic prophylaxis for the prevention of bacterial endocarditis?

- Prosthetic heart valve
- Complex cyanotic congenital heart disease
- Prior coronary artery bypass graft
- Surgically constructed systemic pulmonary shunts or conduits
- Mitral valve prolapse with regurgitations and / or thickened leaflets

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- **Prior coronary artery bypass graft**

Cardiac Conditions Stratification for Risk of Endocarditis	
Endocarditis Prophylaxis Recommended	**Endocarditis Prophylaxis Not Recommended**
High Risk	**Negligible Risk**
Prosthetic heart valves	Isolated secundum atrial septal defect
Surgically constructed systemic pulmonary shunts or conduits	Surgical repair of atrial septal defect, ventricular septal defect, or patent ductus arteriosus *(without residual beyond 6 months)*
Complex cyanotic congenital heart disease	Prior coronary artery bypass graft
Prior bacterial endocarditis	Mitral valve prolapse without regurgitation
Moderate Risk	Physiologic, functional, or innocent heart murmurs
Most other congenital cardiac malformations	Previous Kawasaki disease without valvular dysfunction
Acquired valvular dysfunction	Previous rheumatic fever without valvular dysfunction
Hypertrophic cardiomyopathy	Cardiac pacemakers and implanted defibrillators
Mitral valve prolapse with regurgitations and/or thickened leaflets	

Which artery originates at the level of the tip of the greater horn of the hyoid bone and **does not** accompany the corresponding nerve throughout its course?

- Submental artery
- Inferior alveolar artery
- External carotid artery
- Lingual artery
- Ascending pharyngeal artery

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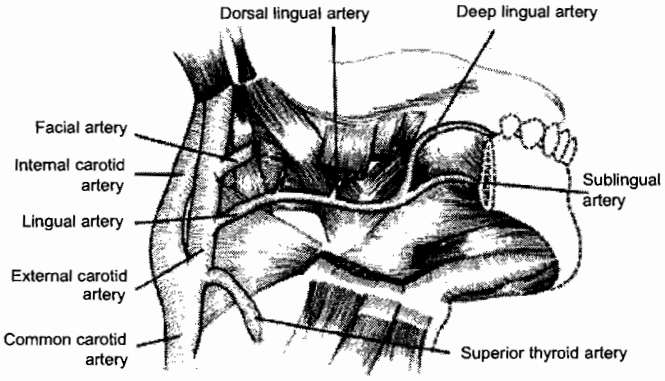
• **Lingual artery**

The lingual artery arises from the **external carotid artery** at the level of the tip of the greater horn of the hyoid bone *(below the facial artery)* in the carotid triangle and passes deep to the hyoglossus muscle to supply the tongue. It **does not** accompany the corresponding nerve throughout its course. The lingual artery supplies structures of the floor of the mouth and the posterior and inferior surface of the tongue.

Branches of the lingual artery:

- **Suprahyoid artery** – supplies the suprahyoid region
- **Dorsal lingual artery** – supplies the dorsum of the tongue *(remember the dorsum is the top part that can be seen easily)*
- **Sublingual artery** – supplies the floor of the mouth and sublingual gland
- **Deep lingual artery** – terminal branch, supplies the anterior two-thirds of the tongue



Important: The **inferior alveolar nerve** and **artery** along with the **lingual nerve** are found in the **pterygomandibular space** between the medial pterygoid muscle and the ramus of the mandible. The inferior alveolar nerve passes lateral to the sphenomandibular ligament.

Note: The lateral pterygoid muscle forms the roof of the **pterygomandibular space.**

The **highest** incidence of fractures occurs in:

- Infants
- Females between the ages 25 and 35
- Young males between the ages of 15 and 24
- Older males between the ages of 65 and 75

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- **Young males between ages 15 and 24** → these fractures are usually the result of trauma

Four reasons that a fracture **does not** heal:

1. **Ischemia** → the navicular bone of the wrist, the femoral neck, and the lower third of the tibia **are all poorly vascularized** and therefore are subject to **ischemic necrosis after a fracture.**
2. **Excessive mobility** → healing is prevented and **pseudoarthrosis** or a **pseudo-joint** may occur.
3. **Interposition of soft tissue** → occurs between the fractured ends.
4. **Infection** → compound fractures have a tendency to become **infected.**

***Important → a **fat embolism** is most often a **sequela of fractures.**

<u>Inappropriate healing *(three types):*</u>

- **Delayed-union** → satisfactory healing which requires greater than the normal six week period. May be caused by infection, interposition of soft tissue or muscle between the fracture segments.
- **Non-union** → failure of the fracture segments to unite properly. May be caused by infection, improper immobilization, or interposition of soft tissue.
- **Mal-union** → can be either delayed or complete union in an improper position. May be caused by improper immobilization or imperfect reduction.

Which of the following is the **primary direction of luxation** for extracting maxillary **deciduous** molars?

- Buccal
- Palatal
- Mesial
- Distal

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• **Palatal**

***As opposed to the **buccal** direction in adults. This is because the deciduous molars are more palatally positioned and the palatal root is strong and less prone to fracture.

In general, the removal of deciduous teeth is not difficult. It is facilitated by the elasticity of young bone and the resorption of the root structure. **Do not** use the **"cowhorn"** forceps for extraction of lower primary molars because the sharp beaks of these forceps could cause damage to the unerupted permanent premolar teeth.

Notes:

1. If the **preoperative radiograph** shows that the permanent premolar is wedged tightly between the bell-shaped roots of the primary tooth, the best treatment is to section the crown of the primary molar and remove the two portions separately. This will help in not disturbing the permanent tooth.
2. **After extraction of mandibular teeth** on a child in which mandibular block was given, **always advise** child not to bite on his / her lip while he or she is numb. Inform parents as well to watch the child so this does not occur.

All of the following are ways of eliminating dead space **except:**

- **Close the wound** in layers to minimize the postoperative void
- **Apply pressure** dressings
- **Use drains** to remove any bleeding that accumulates
- **Allow the void** to fill with blood so that a blood clot will form

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• **Allow the void to fill with blood so that a blood clot will form**

Dead space in a wound is any area that **remains devoid of tissue** after closure of the wound. It is created by either removing tissues in the depths of a wound or by not reapproximating tissue planes during closure. Dead space in a wound usually fills in with blood which creates a **hematoma** with a high potential for infection. This is more likely to happen in closed wound incisions or in an open wound that has closed over at the top too quickly, leaving "dead space" open underneath. Some of these may resolve themselves, but most need to have the fluid drained and the "dead space" needs to be closed, either by deep suturing or by re-opening the top of the wound and packing until it heals from the bottom up.

Ways in which you can **eliminate** dead space:

- **Close the wound** in layers to minimize the postoperative void
- **Apply pressure** dressings
- **Use drains** to remove any bleeding that accumulates
- **Place packing** into the void until bleeding has stopped

The treatment of choice for a recurrent ranula is:

- Marsupialization
- Decompression
- Enucleation
- The Partsch operation

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• **Marsupialization**

Marsupialization, decompression, and the Partsch operation all refer to **creating a surgical window** in the wall of the cyst. The cyst is uncovered or **"deroofed"** and the cystic lining made continuous with the oral cavity or surrounding structures. The cyst sac is opened and emptied.

Marsupialization is the treatment of choice for:
- Ranula → **Note:** For a **recurrent ranula** treatment would also include the excision of the sublingual gland.
- When a cyst is large and close to vital structures

Enucleation is the total removal of a cystic lesion. It is the treatment of choice for:
- Congenital cysts
- Mucoceles
- Most odontogenic cysts

***Enucleation is the treatment of choice whenever possible.

Whether a bone cyst or other cysts are completely enucleated or treated by marsupialization **depends on** the size and location to vital structures.

The **maxillary sinus** opens into the:

- Frontonasal duct
- Bulla ethmoidalis
- Hiatus semilunaris
- Nasolacrimal duct

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- **Hiatus semilunaris** → which is a groove in the middle meatus of the lateral nasal cavity. It also contains the openings of the frontonasal duct and the anterior ethmoid air cells.

Located within the frontal, ethmoid, maxillary, and sphenoid bones are a series of mucous membrane-lined air spaces called the **paranasal sinuses.** The sinuses lighten the skull and enhance the resonance of the voice. The maxillary sinuses develop after the permanent teeth have erupted. Growth continues through adulthood.

Each sinus —or group of sinuses in the case of the ethmoids —is represented on the right and left side of the head. These tend to cause symptoms of pain in different locations of the face. **Maxillary sinusitis** causes pain in the mid-face *(below the eyes)*, cheek, or maxillary posterior teeth. **Ethmoid sinus** infection triggers pain between the eyes, near the bridge of the nose. **Frontal sinusitis** usually causes forehead pain. Pain behind the eyes or at the back of the head may indicate **sphenoid sinusitis.**

Clinical signs of **acute** maxillary sinusitis:
- **Severe pain,** constant and localized
- Tenderness to **percussion** of the **maxillary posterior teeth**
- A mucopurulent **exudate**
- Any unusual motion or jarring **accentuates the pain**
- **Tenderness** over the anterior sinus wall

Commonly prescribed **antibiotics** for the treatment of maxillary sinusitis include ampicillin, amoxicillin and clavulanic acid, cefaclor, cepfuroxime axetil, and trimethoprim-sulfmethoxazole.

Note: The maxillary sinus is innervated by the maxillary division of the trigeminal nerve *(CN V-2)*. Specifically, the ASA, PSA, and MSA nerves as well as the infraorbital nerve.

Which structure **associated with the TMJ** is fibrous, saddle-shaped, and separates the condyle and the temporal bone?

- Articular fossa
- Articular disc *(meniscus)*
- Articular eminence
- None of the above

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• **Articular disc** ***(meniscus)***

The TMJ is the articulation between the condyle of the mandible and the squamous portion of the temporal bone. The condyle is elliptically shaped with its long axis oriented mediolaterally. The articular surface of the temporal bone is composed of the concave **articular fossa** *(which is the anterior three-fourths of the larger mandibular or glenoid fossa)* and the convex **articular eminence** *(tubercle)*.

The most **superficial layer** of the articular surfaces of the TMJ *(condyle and fossa)* is made of **dense fibrous connective tissue** and not hyaline cartilage. Underneath this superficial layer is a layer of fibrocartilaginous tissue that offers resistance against both compressive and lateral forces. Articular surfaces of most diarthrodial joints are covered by hyaline cartilage, but the TMJ is an **atypical** diarthrodial joint in that its articular surfaces are covered with a **dense fibrous connective tissue.**

The **articular disc** *(meniscus)* is composed of **dense fibrous connective tissue,** and it is positioned in between the condyle and the fossa, thereby dividing the joint into superior and inferior joint spaces.

The **articular disc** *(meniscus)* varies in thickness; the thinner central intermediate zone separates the **thicker portions,** which are the **anterior and posterior bands.** The posterior band of the articular disc is the thickest of the two bands, and it is attached with the posterior loose connective tissues called the **retrodiscal tissues** *(bilaminar zone; posterior attachment)*. The less thick anterior band of the articular disc is contiguous with the capsular ligament, the condyle, and the superior belly of the lateral pterygoid muscle.

Note: The **retrodiscal tissue** is highly vascularized and innervated, whereas the articular disc for the most part is not. Only the extreme periphery of the articular disc is slightly innervated.

Following a local anesthetic injection, anesthetic effects will disappear and reappear in a definite order. Which sensation disappears first?

- Touch
- Warm
- Deep pressure
- Pain
- Cold
- Motor

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• **Pain**

Peripheral nerve functions are not affected equally by local anesthetics. Loss of sympathetic function usually occurs first, followed by loss of pain sensation, temperature, touch, deep pressure, and lastly, motor function. This phenomenon is called **differential blockade.** Differential blockade may be due to the size of the nerve, the presence or absence of myelin, and firing frequency.

Size of nerve → local anesthetics preferentially block **small fibers** because the distance over which such fibers can passively propagate an electrical impulse is shorter. During the onset of local anesthesia, when short sections of nerve are blocked, the **small-diameter fibers are the first to fail to conduct.**

Presence or absence of myelin → myelinated fibers tend to become blocked before unmyelinated fibers of the same diameter.

Firing frequency → sensory fibers, especially **pain fibers,** have a **high firing rate** and a relatively long action potential duration *(up to 5 msec)*. Motor fibers fire at a slower rate and have shorter action potential duration *(< 0.5 msec)*. A delta and C fibers are small diameter fibers that participate in high-frequency pain transmission. Therefore, they are **blocked sooner** with lower concentrations of local anesthetics than are A alpha *(motor)* fibers to skeletal muscle.

Clinically, the general order of **loss of function** is as follows:
• Pain
• Temperature *(cold then warm)*
• Touch
• Deep pressure
• Skeletal muscle tone *(motor)*

Note: Nerves regain function in **reverse order.**

Laryngospasm is an uncontrolled / involuntary muscular contraction *(spasm)* of the laryngeal cords. It is a well known, infrequent but serious post-surgical complication. In the operating room it is treated by administering:

- Nitrous oxide
- Oxygen
- Epinephrine
- Enflurane

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• **Oxygen**

A patient under general anesthesia **loses the laryngeal reflex.** If blood and saliva collect near the vocal cords, this stimulates the patient to go into spasm *(laryngospasm)* and the vocal cords will close. When this happens, air cannot pass through and hence the problem. The two most important steps in the initial management of a laryngospasm are applying **oxygen** under positive pressure and administering **succinylcholine.**

Note: Succinylcholine is a **skeletal muscle relaxant** that is used when performing endotracheal intubation and endoscopy procedures.

Laryngospasm is frequently cited as an adverse effect of **ketamine,** but it is rarely observed. Frequently, deep, heavy, loud respirations mistaken for laryngospasm are actually due to airway positioning. Such breathing is managed simply by repositioning the patient's head. True laryngospasm during ketamine sedation is usually caused by stimulation of the vocal cords by instrumentation or secretions.

All of the following are reasons that **vasoconstrictors** are included in local anesthetics **except:**

- They **prolong** the duration of action of the local anesthetic
- They **reduce** the chance of an allergic reaction to the local anesthetic
- They **reduce** the toxicity because less local anesthetic is necessary
- They **reduce** the rate of vascular absorption by causing vasoconstriction
- They help to make the anesthesia **more profound** by increasing the concentrations of the local anesthetic at the nerve membrane

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• **They reduce the chance of an allergic reaction to the local anesthetic**

***This is **false.**
The **most important reason** for the use of vasoconstrictors is to **prolong** the duration of action of the local anesthetic.

Some Vasoconstrictors Used in Local Anesthetics		
Agent	**Available Concentrations**	**Local Anesthetics Which Contain These Agents**
Epinephrine	1:50,000 1:100,000 1:200,000 1:200,000	Lidocaine 2% Lidocaine 2% Prilocaine 4% Marcaine 0.5%
Levonardefrin *(Neo-Cobefrin)*	1:20,000	Mepivovaine 2%
Norepinephrine *(Levophed)*	1:30,000	Procaine 2%

Remember: Vasoconstrictors act at **alpha receptors** to produce constriction of arterioles. **Cocaine** acts as an intrinsic vasoconstrictor. It *(cocaine)* increases the pressor activity of both epinephrine and norepinephrine.

After receiving an injection of a local anesthetic containing 2% lidocaine with 1:100,000 epinephrine, the patient **loses consciousness.** Which of the following is the most probable cause?

- Acute toxicity
- Allergic response
- Syncope
- Hyperventilation syndrome

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• **Syncope**

*****Caused by transient cerebral hypoxia**

Proper management of syncope:

• Place patient in **supine position** with feet slightly elevated *(Trendelenburg position)*

• Establish airway *(head tilt / chin lift)*
  – Administer 100% oxygen via face mask. 0_2 is indicated for treatment all types of syncope **except** hyperventilation syndrome.

• Monitor **vital signs** and **support** patient
  – **Pupils** may **dilate** from brain not getting oxygen.

• Maintain your composure. **Apply cool, wet towel to patient's forehead.**

• Follow-up treatment
  – Determine factors causing unconsciousness.

Remember: Hyperventilation in an anxious dental patient leads to **carpodedal spasm** *(a spasm of the hand, thumbs, foot, or toes).*

Concerning the clinical uses of benzodiazepines, which one of the following statements is **false?**

- Diazepam is used for muscle spasticity in patients with cerebral palsy
- Intravenous diazepam is useful in status epilepticus
- Clonazepam is approved for use in the management of obsessive-compulsive disorders
- Alprazolam has selective anxiolytic effects in patients who suffer from agoraphobia

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• **Clonazepam is approved for use in the management of obsessive-compulsive disorders**

***None of the benzodiazepines have shown significant therapeutic benefit in the management of obsessive-compulsive disorders. Drugs effective for this condition increase the activity of serotonergic systems in the brain, for example fluoxetine, sertraline, and paroxetine.

Benzodiazepines such as **diazepam** *(Valium)*, **chlordiazepoxide** *(Librium)*, **lorazepam** *(Ativan)*, **clonazepam** *(Rivotril)*, **flurazepam** *(Dalmane)*, **temazepam** *(Restoril)*, **triazolam** *(Halcion)*, **alprazolam** *(Xanax)*, and **midazolam** *(Versed)* are medications that are frequently prescribed for the symptomatic treatment of anxiety and sleep disorders. They produce their effects via specific receptors involving GABA. Benzodiazepines are the **most effective** oral sedative drugs used in dentistry.

Benzodiazepines, particularly **diazepam and chlordiazepoxide** are frequently prescribed for **preoperative sedation.** These drugs are tranquilizers and are used to produce conscious sedation in anxious patients. Diazepam is **more potent** than chlordiazepoxide. These drugs **do not produce** hangovers like barbiturates and other sedative drugs. **Note:** Do **not use** these drugs during pregnancy.

Important: Be careful when administering any sedative drug to a patient who is taking a **phenothiazine drug** *(i.e., Chlorpromazine, Fluphenazine, Prochlorperazine)*. These drugs will potentiate the action of sedative drugs.

Notes:

1. **Chloral hydrate** is a sedative and hypnotic that is widely used for **pediatric** sedation.
2. Emotional stress **decreases** the rate of absorption of a drug when given orally.

Which of the following **is not** a contraindication to tooth extraction?

- ANUG
- Acute pericornitis
- Acute dentoalveolar abscess
- Irradiated jaws

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• **Acute dentoalveolar abscess**

An acute dentoalveolar abscess **should not** be a contraindication to extraction. It has been shown that these infections can resolve very quickly when the affected tooth is removed. **However,** it may be difficult to extract such a tooth, either because the patient is unable to open sufficiently wide enough or because adequate local anesthesia cannot be obtained.

Local and Systemic Contraindications to Tooth Extractions	
Local	**Systemic**
Acute infection with uncontrolled cellulitis	Uncontrolled diabetes mellitus
Acute pericornitis	Uncontrolled cardiac disease and dysrythmias
Acute infectious stomatitis	Severe bleeding disorders
Malignant disease	Uncontrolled leukemias and lymphomas
Irradiated jaws	Debilitating diseases
ANUG	Patients who are taking certain medications *(e.g., immunosuppressants, corticosteroids, and cancer chemotherapeutic agents)*

How much **hydrocortisone** is secreted by the adrenal cortex **daily?**

- About 1 mg
- About 100 mg
- About 20 mg
- About 200 mg

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- **About 20 mg**

During stress the cortex can increase the output to **200 mg daily.**

Remember: Patients taking steroids or people with disease of the adrenals will have decreased ability to produce more glucocorticoids *(hydrocortisone)* in times of stress *(extractions).* **The reason for this is as follows:**

Secretion of glucocorticoids is stimulated by **ACTH,** a hormone produced in the anterior pituitary. The pituitary responds to stress by **increasing ACTH** output and therefore glucocorticoid production increases. A relative lack of glucocorticoids will also increase output of ACTH. An **overabundance** of circulating systemic steroids will inhibit production of ACTH. Patients on large doses of steroids **repress ACTH production** which leads to atrophy of adrenal cortex.

The major concern for the dental practitioner treating the patient with insulin-dependent diabetes mellitus is:

- Hyperventilation
- Vasovagal syncope
- Hypoglycemia
- Hypercapnea

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• **Hypoglycemia**

Diabetes Mellitus is a complex disorder involving mostly carbohydrates *(glucose)* and lipids owing primarily to a relative or complete lack of insulin secretion by the beta cells of the pancreas.

People with **well-controlled diabetes** are no more susceptible to infections than people without diabetes, **but** they have more difficulty containing infections *(this is caused by altered leukocyte function)*.

Patients who **take insulin daily** and check their urine regularly for the sugar and ketones *(controlled diabetics)* usually can be treated in the normal manner **without** additional drugs or diet alterations.

Important: If any doubt exists as to the patient's medical status, consultation with the patient's physician is indicated. **Do not assume anything.**

Symptoms of hypoglycemia: tachycardia, palpitations, sweating, tremulousness, nausea, and hunger. The symptoms may progress to coma and convulsions without intervention.

Notes:

1. The treatment of choice for hypoglycemia in an **unconscious** diabetic patient → EMS should be contacted. Then 1 mg of glucagon can be injected IM, or 50 ml of 50% glucose solution can be given by rapid IV infusion. The glucagon injection should restore the patient to a conscious state within 15 minutes; then some form of oral sugar can be given.
2. The treatment of choice for hypoglycemia in a **conscious** diabetic is the administration of an **oral carbohydrate** *(packets of table sugar, orange juice, cola beverages, candy bars, etc.)*

All of the following are symptoms of dehydration **except:**

- Oliguria
- Rise in body temperature
- Increase in heart rate and cardiac output
- Increase in blood pressure
- Severe cell dysfunction

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• **Increase in blood pressure** → actually there is a **decrease** in blood pressure

Dehydration is the loss of water and important blood salts like potassium (K^+) and sodium (Na^+). Vital organs like the kidneys, brain, and heart can't function without a certain minimum amount of water and salt. Causes include decreased intake *(lack of water)* and / or increased output *(vomiting, diarrhea, loss of blood, drainage from burns, diabetes mellitus, diuretic use, or a lack of ADH owing to diabetes insipidus).*

Initially, a patient suffering from dehydration will clinically demonstrate only dryness of the skin and mucous membranes.

However, as dehydration progresses, the **turgor** *(or fullness)* of the skin is lost. If dehydration persists, **oliguria** *(reduced urine output)* occurs as a compensation for the fluid loss. More severe degrees of fluid loss are accompanied by a shift of water from the intracellular space to the extracellular space, a process that causes **severe cell dysfunction,** particularly in the **brain.** Systemic blood pressure falls with continuous dehydration, and declining perfusion eventually leads to death.

Fluids in several forms should be continually urged on the patient. In **severely dehydrated individuals,** they must get to the hospital right away. **IV fluids** will quickly reverse dehydration, and is often **life saving in young children and infants.**

Which of the following is the gold standard for bone regenerative grafting materials for several reasons including the capability to support osteogenesis and having osteoinductive and osteoconductive properties?

- Xenogenic bone
- Allogenic bone
- Autogenous bone
- Alloplastic bone

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• **Autogenous bone**

Autogenous bone is bone from the **same person** *(from one part of the body to another, requires donor site)*. Autogenous grafts *(also called an autograft)* are usually employed to restore large areas of lost mandibular bones following oncological surgery or trauma. Of all the facial bones resected in oncological surgery, the mandible is the **most frequently** removed.

Three forms of **free bone grafts** include:

- **Cortical** grafts are able to withstand early mechanical forces, however, they require more time to revascularize. Common donor sites: cranial vault, Iliac crest, ribs, mandibular symphysis, external oblique ridge.
- **Cancellous** grafts: advantage of cancellous grafts include an apparent increase in healing rate. The most abundant supply can be harvested from the anterior or posterior iliac crest. The only apparent disadvantage of autogenous cancellous grafts is their inability to provide mechanical stability.
- **Corticocancellous** grafts: the advantages of the corticocancellous graft are their ability to provide some mechanical stability and provide some increase in osteogenesis. Corticocancellous grafts do not have the ability to increase osteogenesis as much as cancellous grafts due to the layer of relatively nonporous cortical bone. Donor sites include the ribs, ilium, and skull.

The bone marrow for grafting defects in the mandible and maxilla is generally obtained from the **iliac crest.** Also used for **ridge augmentation.**

Notes:

1. A **costochondrial rib** graft may be employed with the cartilaginous portion simulating the TMJ and condyle. When used for ridge augmentation there is a great deal of **shrinkage.**
2. **Bone plates, biphasic pins, titanium mesh, and intraosseous wires** are used in the fixation of bone grafts. Sutures **are not** generally used.

Osteomyelitis is generally classified into two major groups: suppurative and non-suppurative. **Non-suppurative** osteomyelitis is classified into all of the following **except:**

- Chronic sclerosing *(focal and diffuse)* osteomyelitis
- Garre's sclerosing osteomyelitis
- Acute osteomyelitis
- Actinomycotic osteomyelitis

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• **Acute osteomyelitis**

Osteomyelitis is an inflammatory process within medullary *(trabecular)* bone that involves the marrow spaces. Osteomyelitis is generally classified into two major groups: suppurative and non-suppurative. **Suppurative** osteomyelitis is classified into acute, chronic, or infantile osteomyelitis. **Non-suppurative** osteomyelitis is classified into chronic sclerosing *(focal and diffuse),* Garre's osteomyelitis, and actinomycotic osteomyelitis.

Osteomyelitis can affect both adults and children. It is most often caused by **Staphylococcus aureus.** In **adults,** osteomyelitis often affects the vertebrae and the pelvis. In **children,** osteomyelitis usually affects the adjacent ends of long bones. They include the femur and tibia in the legs and the humerus and radius in the arms.

Note: Garre's osteomyelitis is a distinctive type of non-suppurative osteomyelitis associated with periosteal thickening and peripheral reactive bone formation resulting from mild irritation or infections. The condition is seen exclusively in children or young adults. The mandible is more often affected than the maxilla. Clinically it manifests as a bony, hard, non-tender swelling which is slowly progressive and associated with a painful carious tooth.

Important: Acute osteomyelitis occurs more frequently in the **mandible** as opposed to the maxilla. The primary reason for this is that the blood supply to the **maxilla** is much richer and is derived from a number of different arteries, while the **mandible** tends to draw its primary supply from the inferior alveolar artery. The dense overlying cortical bone of the mandible prevents penetration of periosteal blood vessels, thus the mandibular cancellous bone is more likely to become ischemic and therefore infected. **Important point → a reduced blood supply will predispose bone to osteomyelitis.**

An **endosseous** dental implant has between the bone and implant a:

- Periodontal ligament
- Peri-implant ligament
- Epithelial ligament
- A bone-implant interface

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• **A bone-implant interface**

The histologic definition of **osseointegration** is best described by the following: The direct connection between living bone and a load-bearing endosseous implant at the light microscopic level. Only endosseous and transosseous implants are considered true osseointegrated implants.

Criteria for **success** of a dental implant:
- Clinical immobility under load-bearing conditions
- Symptom free
- Minimal loss of crestal bone
- No peri-implant radiolucency
- Success rate of 85% after 5 years and 80% after 10 years

Notes:

1. In order for an implant to be successful you need **adequate transfer of force** and **biocompatibility.**
2. Handpieces for preparation of dental implant receptor sites are **lowspeed / high torque.**
3. The **preferred floss** to use to clean dental implant abutments is superfloss or yarn.
4. In the event an endosseous dental implant is mobile, the proper procedure is to remove the failing implant, debride the socket, and consider placing a bone graft with a resorbable membrane.
5. You need a minimum of **10 mm** of bone height to place an endosseous *(root form)* dental implant.
6. The minimum required distance from the apex of a mandibular posterior implant to the superior aspect of the inferior alveolar canal is **2 mm.**
7. **Titanium and titanium alloy** are the most common materials used today for two-stage endosseous implants.

All of the following statements concerning injections are true **except:**

- An intravascular injection can cause dizziness or blurred vision
- Injections should always be made slowly with aspiration to avoid intravascular injection
- The presence of a vasoconstrictor prevents an intravasular injection or systemic absorption
- The depression of respiration is a manifestation of the toxic effect of the local anesthesia

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• **The presence of a vasoconstrictor prevents an intravasular injection or systemic absorption**

Vasoconstrictors *(i.e., epinephrine and levonordefrin)* are added to local anesthetics because of their vasoconstrictive properties. Vasoconstriction at the site of injection is beneficial because it limits the uptake of the anesthetic by the vasculature, thereby **increasing the duration** of the anesthetic and **diminishing systemic effects.**

Important: To minimize the likelihood of intravascular injection, **aspiration** should be performed before the local anesthetic solution is injected. If blood is aspirated, the needle must be repositioned until no return of blood can be elicited by aspiration. Note, however, that the absence of blood in the syringe does not assure that intravascular injection will be avoided.

Adverse reactions following the administration of a local anesthetic are, in general, dose-related and may result from high plasma levels caused by excessive dosage, rapid absorption or **unintentional intravascular injection.**

CNS manifestations are excitatory and / or depressant and may be characterized by lightheadedness, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, tinnitus, blurred or double vision, vomiting, sensations of heat, cold or numbness, twitching, tremors, convulsions, unconsciousness, respiratory depression, and arrest. The excitatory manifestations may be **very brief or may not occur at all,** in which case the first manifestation of toxicity may be **drowsiness** merging into unconsciousness and respiratory arrest.

Cardiovascular manifestations are usually **depressant** and are characterized by bradycardia, hypotension, and cardiovascular collapse, which may lead to cardiac arrest. **Note:** In local anesthesia, the **depression of respiration** is a manifestation of the toxic effects of the solution.

Hypercalcemia is an abnormally high level of calcium in the blood, usually more than 10.5 milligrams per deciliter of blood. Many different conditions can cause hypercalcemia; the most common are:

- Hyperparathyroidism and cancer
- Diabetes and hypothyroidism
- Ischemic heart disease and hypertension
- Grave's disease and hypopituitarism

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• **Hyperparathyroidism and cancer**

Calcium levels are regulated by parathyroid hormone *(increased hormone causes bone resorption)* which in turn increases calcium levels. Calcium is also regulated to some extent by the kidney tubules and GI mucosa *(lowering pH will cause increased calcium absorption)*. **Low serum calcium levels** will result in hyperirritability of nerves and muscles.

Phosphorus concentration is also regulated by parathyroid hormone. Increased hormone causes the kidneys to increase the rate of phosphate excretion which causes a decrease in plasma phosphate concentration.

People with cancer often have hypercalcemia. In fact, it is the most common life-threatening metabolic disorder associated with cancer. Ten to twenty percent of all persons with cancer have hypercalcemia. Cancers of the breast, lung, head and neck, and kidney are frequently associated with hypercalcemia.

Note: In good health the ratio of calcium to phosphorus in the blood is 10:4. If there is a glandular imbalance, especially in regard to the parathyroid glands, then this ratio will be maintained at a different level, causing long-term health deterioration. In particular, a high ratio of phosphorus to calcium sensitizes the body and increases inflammatory tendencies.

Blood glucose concentration is regulated by insulin *(lowers glucose levels)* and glucagon *(increases glucose levels)*. Glucose normally does not appear in the urine although it is freely filtered because it is reabsorbed in the proximal convoluted tubule of the kidney. Serum glucose will be **increased** in diabetes mellitus, adrenal tumors, increased growth hormone, and liver dysfunction.

What is the **most common** type of mid-facial fracture?

- LeFort II
- Zygomaticomaxillary complex
- Zygomatic arch
- Nasoorbital ethmoid fractures

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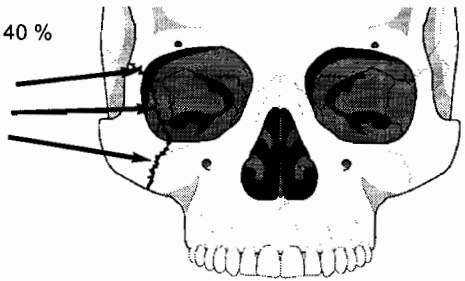
• **Zygomaticomaxillary complex**

The most common mechanism producing facial fractures is auto accidents. About 70 % of auto accidents produce some type of facial injury, although most are limited to soft tissue. The face seems to be a favorite target in fights or assaults, which are the next most common mechanism.

Fracture type prevalence:

- Zygomaticomaxillary complex *(tripod fracture)* → 40 %
- LeFort I → 15 %
  II → 10 %
  III → 10 %
- Zygomatic arch → 10 %
- Alveolar process of maxilla → 5 %
- Smash fractures → 5 %
- Other → 5 %

Frontal view of the zygomaticomaxillary complex



The most common facial fracture is the tripod or zygomaticomaxillary complex fracture, so called because it involves separation of all three major attachments of the zygoma to the rest of the face.

Notes:

1. The maxilla and mandible are in a critical relationship to the upper airway; therefore displacement of fractures can cause obstruction of the airway resulting in respiratory arrest. Control of the airway is **vital** to any treatment of a patient with facial fractures.
2. **Maxillary fractures have** a greater tendency towards the production of facial deformity than do mandibular fractures.

All of the following drugs can reduce salivary flow during dental treatment. Which one, **however,** works by reducing anxiety and sensitivity during the procedure?

- Scopolamine
- Atropine
- Local anesthesia
- Benztropine

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• **Local anesthesia**

Local anesthesia acts by reducing sensitivity **which therefore** reduces anxiety and stress related to treatment; salivation is also decreased.

Scopolamine, atropine and benztropine are **anticholinergic** drugs. Not only do they decrease the flow of saliva, but also decrease the secretion from respiratory glands during general anesthesia.

If the facial nerve was cut as it exited the stylomastoid foramen, which of the following muscles would not be effected?

- Posterior belly of the digastric
- Stylohyoid
- Stapedius
- Mylohyoid
- All of the above muscles would be effected

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• **Mylohyoid**

The **facial nerve** originates in the pons, traverses the facial canal of the temporal bone, and exits the cranium through the **stylomastoid foramen.** From the stylomastoid foramen it extends laterally around the neck of the mandible through the **parotid** gland. **Note:** If you cut the facial nerve just after its exit from the foramen, it would cause a loss of innervation to the **muscles of facial expression.**

The facial nerve has **four components** with distinct functions:

1. **Brancial motor** *(special visceral efferent)* → Supplies the muscles of facial expression; poster or belly of digastric muscle; stylohyoid, and stapedius.
2. **Visceral motor** *(general visceral efferent)* → Parasympathetic innervation of the lacrimal, submandibular, and sublingual glands, as well as mucous membranes of nasopharynx, hard and soft palate.
3. **Special sensory** *(special afferent)* → Taste sensation from the anterior 2/3 of tongue; hard and soft palates.
4. **General sensory** (general somatic afferent) → General sensation from the skin of the concha of the auricle and from a small area behind the ear.

Clinical information:

1. **Bell's palsy** → facial paralysis, a functional disorder of the facial nerve, caused by nerve irritation or viral infection and is, therefore, usually temporary.
2. **Facial trauma** → trauma to the facial nerve destroys the ability to contract the facial muscles on the affected side of the face and distorts taste perception. The affected side of the face tends to sag since muscle tonus is lost.

According to Geudel's stages of anesthesia, the proper use of nitrous oxide achieves which level of anesthesia?

- Stage 1
- Stage 2
- Stage 3
- Stage 4

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- **Stage 1**

Geudel's Stages of Anesthesia

- **Stage I** *(amnesia and analgesia)* → begins with the **administration of anesthesia** and continues to the loss of consciousness. Respiration is quiet, though sometimes irregular, and reflexes are still present.

- **Stage II** *(delirium and excitement)* → begins with the **loss of consciousness** and includes the onset of total anesthesia. During this stage the patient may move his limbs, chatter incoherently, hold his breath, or become violent. Vomiting with the attendant danger of aspiration may occur. The patient is brought to Stage III as quickly and as smoothly as possible.

- **Stage III** *(surgical anesthesia)* → begins with the establishment of a **regular pattern of breathing**, total loss of consciousness and includes the period during which signs of respiratory or cardiovascular failure first appear. **This stage has four planes.**

- **Stage IV** *(premortem)* → **signals danger.** This stage is characterized by pupils that are maximally dilated and skin that is cold and ashen. Blood pressure is extremely low, often unmeasurable. **Cardiac arrest is imminent. Remember:** The eyes appear greatly enlarged in size and nonreactive to bright light when functional circulation to the brain has stopped.

Which type of biopsy is most often used to observe the borders of a suspicious lesion?

- Incisional
- Excisional
- Needle
- Exfoliative cytology

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• **Excisional**

After removal, the tissue should be immediately placed in **10% formalin** solution *(4% formaldehyde)* that is at least 20 times the volume of the surgical specimen. The tissue **must** be totally immersed in the solution, and care should be taken to be sure that the tissue has not become lodged on the wall of the container above the level of formalin.

Types of biopsies:

- **Incisional** → take only **part** of lesion
- **Excisional** → entire lesion is removed
- **Needle** → aspirational biopsy
- **Exfoliative cytology** → pap smear

} **most often used for oral lesions**

Remember: A **negative** incisional biopsy report of a **highly suspicious** oral lesion suggests that another biopsy specimen is necessary in view of the clinical impressions. The key is a **highly suspicious** oral lesion. Tissue samplings should be obtained from multiple sites of the lesion.

Important: It can not be overemphasized that all pertinent clinical information and the findings of other diagnostic modalities must be provided to the pathologist at the time of the initial submission of the specimen.

Which of the following occurs during the compensatory stage of shock?

- Decreased peripheral resistance
- Increased heart rate
- Metabolic acidosis
- Organ damage

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• **Increased heart rate**

The term shock denotes a clinical syndrome in which there is inadequate cellular perfusion and inadequate oxygen delivery for the metabolic demands of the tissues. **Important:** Reduced cardiac output is the **main factor** in all types of shock.

In general, **shock is characterized** by:
• Increased vascular resistance → cool mottled skin, oliguria
• Tachycardia
• Adrenergic response → diaphoresis, anxiety, vomiting, diarrhea
• Myocardial ischemia
• Mental status change

The **stages of shock** include: *1)* **Compensatory** *(early)* stage → compensatory mechanisms *(increased heart rate and peripheral resistance)* maintain perfusion to vital organs, *2)* **Progressive** stage → metabolic acidosis occurs *(compensatory mechanisms are no longer adequate), 3)* **Irreversible** *(refractory stage)* → organ damage, **survival is not possible.**

Major Categories of Shock:
• **Hypovolemic** shock is produced by a reduction in blood volume. Cardiac output will be low due to inadequate left ventricular filling. Causes include severe hemorrhage, dehydration, vomiting, diarrhea, and fluid loss from burns.
• **Cardiogenic** shock is circulatory collapse resulting from pump failure of the left ventricle, most often caused by **massive myocardial infarction.**
• **Septic** shock is due to **severe infection.** Causes include the endotoxin from gram-negative bacteria.
• **Neurogenic** shock results from severe **injury or trauma** to the CNS.
• **Anaphylactic shock** occurs with severe **allergic reaction.**

When **removing maxillary teeth,** the upper jaw of the patient should be **where** in relation to the dentist's shoulder?

- Below
- Above
- At the same height
- It makes no difference where the patient's upper jaw is in relation to the dentist's shoulder

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• **At the same height**

For **mandibular extractions,** the patient should be positioned so that the occlusal plane of the mandibular arch is **parallel to the floor** when the mouth is opened. The chair should be as low as possible.

Positioning of the surgeon → When extracting **maxillary teeth,** it is usually best to stand in front of and to the side of the patient for maximum visibility and leverage. When extracting **mandibular teeth,** it is often better to stand directly to the side or behind the patient.

The **fingers of the left hand** *(for a right-handed dentist)* serve to:
- Retract the soft tissue.
- Provide the operator with sensory stimuli for the detection of expansion of the alveolar plate and root movement under the plate.
- Help guide the forceps into place on the tooth.
- Protect teeth in the opposite jaw from accidental contact with the back of the forceps.
- Support the mandible while performing mandibular extractions.

Remember → recommended sequence of extraction:
- Maxillary teeth before mandibular teeth
- Posterior teeth before anterior teeth

What **two** areas are impacted maxillary third molars occasionally displaced into?

- Canine space
- Pterygomaxillary space
- Infratemporal space
- Pharyngeal space
- Maxillary sinus

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- **Infratemporal space**
- **Maxillary sinus**

Impacted maxillary third molars are occasionally displaced into **two** areas:

- **Maxillary sinus** *(antrum)* → from which they are removed via a **Caldwell-Luc approach**
- **Infratemporal space** → during elevation of the tooth the elevator may force the tooth posteriorly through the periosteum into the **infratemporal fossa.** If access and light are good, the tooth may be retrieved with a hemostat. If the tooth is not retrieved after a short amount of time, the area should be closed. The patient should be informed that the tooth has been displaced and will be **removed by an oral surgeon** who will use a special technique to remove it.

Note: To minimize the chance of dislodging an impacted maxillary third molar into the infratemporal fossa during its surgical removal, place a broad retractor distal to the molar while elevating it.

Remember:

1. When performing a surgical removal of a **mandibular molar,** do not section through the entire tooth. The lingual plate is often thin, and complete sectioning may perforate the plate and injure the lingual nerve.
2. The **inferior alveolar nerve** most often lies **buccal** to the roots of a mandibular third molar.
3. Buccal to lingual movement is not efficient when removing **mandibular posterior teeth** because mandibular bone is too dense and does not expand in a similar fashion to that of the maxillary bone.

Innervation of the soft tissue of the posterior **two-thirds** of the hard palate is derived from the:

- Nasopalatine nerve
- Supratrochlear nerve
- Greater *(anterior)* palatine nerve
- Infraorbital nerve

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• **Greater *(anterior)* palatine nerve**

The **hard palate** is perforated by the following foramina:

- The **incisive foramen,** posterior to the maxillary incisors, which transmits the nasopalatine nerves and the terminal branches of the sphenopalatine artery.
- The **greater palatine foramen,** medial to the third molar, which transmits the greater palatine vessels and nerve.
- The **lesser palatine foramen,** just posterior to the greater palatine foramen, which transmits the lesser palatine vessels and nerve.

Nerves of the palate:

Sensory Innervation to the palate → is supplied by the **maxillary** *(CN V-2)* **nerve.** The **anterior** part of the hard palate is supplied by the **nasopalatine nerve** which passes through the incisive foramen. The **posterior** part of the hard palate is supplied by the **greater palatine nerve** which passes through the greater palatine foramen. The **soft** palate is supplied by the **lesser palatine nerve** which passes through the lesser palatine foramen.

Motor Innervation → the tensor veli palatini is innervated by the motor root of the trigeminal nerve *(CN V)*. All other muscles are innervated by the vagus nerve *(CN X)* via the pharyngeal plexus.

To anesthetize the **greater** *(anterior)* **palatine nerve,** you need to deposit local anesthetic at the greater palatine foramen. The greater palatine foramen is situated **between the second** and **third maxillary molars** about 1 cm from the palatal gingival margin toward the midline.

The preferred test to assess the level of anticoagulant therapy in a patient taking warfarin is:

- Prothrombin time *(PT)*
- Ivy bleeding time
- International normalized ratio *(INR)*
- Partial thromboplastin time *(PTT)*

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• **International normalized ratio *(INR)***

The **INR** now is the preferred laboratory test for assessing anticoagulant therapy in the patient taking warfarin. The PT test has been used but this test was of limited value because of the variability among laboratories. To promote standardization of the PT test, the World Health Organization developed an international reference thromboplastin from human brain tissue and recommended that the PT ratio be expressed as the INR.

Remember: While the INR range for minor oral surgery is subjective, local hemostatic measures generally are very effective if the INR is less than 4. No oral surgery, even of a minor nature, should be performed if the INR is greater than 5. For most patients on long-term warfarin therapy, the target INR is 2 – 3; these patients can be managed safely and effectively in the dental office. The normal INR for a non-anticoagulant patient is 1.

Important point for oral surgery: The risks of discontinuing oral anticoagulants *(even for short periods of time)* are significantly greater than the risk of postoperative bleeding when performing minor oral surgery. Discontinuing oral anticoagulant therapy is associated with increased morbidity and mortality. The dentist should consult with the patient's physician and request an INR test the day of surgery. If the results are acceptable, the dentist should proceed with the surgical procedure using local hemostatic measures. The responsibility for altering any patient's anticoagulant therapy always rests with the attending physician, **not the dentist.**

Anticoagulants include dicumarol, heparin, antithrombin III, enoxaparin, and warfarin. **Remember:** Aspirin, ibuprofen and other NSAID's can interfere with platelet function and are best **avoided in anticoagulated patients.**

The opioids are the most powerful drugs available for the relief of pain. All of the following are **strong** agonists *(e.g., those with the highest analgesic efficacy)* **except:**

- Morphine
- Oxycodone
- Meperidine
- Fentanyl
- Methadone

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• **Oxycodone**

Analgesics fall into two categories: opioid *(narcotic)* analgesics, and non-opioid analgesics *(NSAIDs)*. Opioid analgesics include: morphine, codeine, fentanyl, meperidine, methadone, propoxyphene, levorphenol, hydrocodone, oxycodone and pentazocine.

Codeine, hydrocodone, and oxycodone are mild to moderate agonists. Propoxyphene is a very weak agonist drug. Oxycodone is considered to have the **highest dependency liability** when compared to drugs such as codeine, propoxyphene and pentazocine.

Oxycodone *(contained in Percodan and Percocet)* and **Hydrocodone** *(contained in Lorcet, Lortab and Vicodin)* are opioids similar in structure to morphine and codeine.

- **Percodan** → oxycodone + aspirin : is the strongest pain medication you can prescribe and have the patient still be ambulatory. Do not take on an empty stomach.
- **Percocet** → oxycodone + acetaminophen
- **Lorcet, Lortab and Vicodin** → hydrocodone + acetaminophen : all combine a narcotic analgesic and a cough reliever *(hydrocodone)* with a nonnarcotic analgesic *(acetaminophen)* for the relief of moderate to severe pain.

Codeine is a narcotic analgesic and antitussive *(cough suppressant)*. It is weaker than morphine, less addictive, and less constipating. Codeine is usually combined with other drugs, for example, **Empirin** *(Aspirin + Codeine)*, and **Tylenol #2, 3,** and **4** *(Acetaminophen + Codeine).*

Remember: Naloxone is a μ-opioid receptor competitive antagonist used to counter the effects of opioid overdose.

Body temperature can be measured in several different ways, which one is the **most** accurate?

- Orally
- Axillary
- Rectally
- Aurally

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• **Rectally** → **axillary** is the **least** accurate

General considerations when **checking vital signs:**
- The patient should **not** have had alcohol, tobacco, caffeine, or performed vigorous exercise within 30 minutes of the exam.
- Ideally the patient should be sitting with feet on the floor and their back supported. The examination room should be quiet and the patient comfortable.
- History of hypertension, slow or rapid pulse, and current medications should always be obtained.

Routine Vital Signs:
- Blood pressure → normal 120 / 80
- Pulse rate → normal 72
- Respiration rate → normal 15
- Temperature can be measured in several different ways:
  - **Oral** with a glass, paper, or electronic thermometer *(normal 98.6°F / 37°C)*
  - **Axillary** with a glass or electronic thermometer *(normal 97.6°F / 36.3°C)*
  - **Rectal** or "core" with a glass or electronic thermometer *(normal 99.6°F / 37.7°C)*
  - **Aural** *(the ear)* with an electronic thermometer *(normal 99.6°F / 37.7°C)*

Note: Abnormalities of vital signs are often clues to diseases, the alterations in vital signs are used to evaluate a patient's progress.

Five major areas to be discussed when taking a **patient history** → **1.** Chief complaint **2.** History of present illness **3.** Specific drug allergies **4.** Review of systems *(heart, liver, kidney, brain, etc.)* **5.** Nature of systems.
In **complicated cases,** don't be hesitant to call patient's physician, previous dentists, or other health professionals.

All of the following statements concerning the **masticator space** are true **except:**

- Infections of the zygomatic or temporal bones may pass to the masticator space, and so may abscesses from the mandibular molar teeth.
- It is formed by the splitting of the superficial layer of cervical fascia to enclose the ramus of the mandible, the masseter, the medial pterygoid, and the lower portion of the temporalis muscle.
- It is traversed particularly by the ophthalmic nerve *(V-1)* and the maxillary vessels.
- Abscesses within this space may point at the anterior aspect of the masseter muscle, either into the cheek or the mouth, or they may point posteriorly below the parotid gland.
- It lies largely among the muscles of mastication.

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• **It is traversed particularly by the ophthalmic nerve *(V-1)* and the maxillary vessels**

***This **false;** it is traversed particularly by the **mandibular nerve** *(V-3)* and the maxillary vessels.

The **masseteric, pterygomandibular, and temporal spaces** as a group are known as the masticator space because they are bounded by the muscles and fascia of mastication. Infections of the masticator space are practically always of dental origin, particularly the mandibular molar region. **Note:** Needle tract infections following an inferior alveolar block injection would initially involve the pterygomandibular space.

Clinically, the picture of masticator space infection is dominated by **trismus,** pain, and swelling occurring within a few hours following a molar extraction or trauma to the mandible. These signs increase rapidly to reach a peak in 3 to 7 days. Spontaneous intraoral drainage usually takes place between the 4th and 8th day. If this does not occur, surgical drainage is indicated.

Notes:

1. The **most definite clinical sign** indicating extension of an odontogenic infection into the masticator space is **trismus.** Trismus is difficulty in opening the mouth due to a tonic spasm of the muscles of mastication.
2. Trismus may also result from passing the needle through the **medial pterygoid** muscle during an inferior alveolar nerve block.
3. The submaxillary, submental and sublingual spaces are collectively referred to as the **submandibular space.**

The mandibular left second molar of a 14-year-old boy is unerupted. Radiographs show a small dentigerous cyst surrounding the crown. **What is the treatment of choice?**

- Surgically extract the unerupted second molar
- Uncover the crown and keep it exposed
- Prescribe an anti-inflammatory medication and schedule a follow-up appointment in six months
- No treatment is necessary at this time

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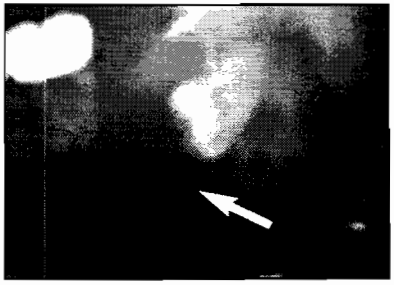
• **Uncover the crown and keep it exposed**

Dentigerous cysts are those associated with the crowns of unerupted teeth. Some literature refers to these cysts as **"follicular"** or **"primordial"** cysts. **Note:** They are probably the result of degenerative changes in the **reduced enamel epithelium.**

Remember: If cysts form when a tooth **is erupting,** they are called **eruption cysts.** These cysts interfere with normal eruption of the teeth. Eruption cysts are more commonly found in the child and young adult, and may be associated with any tooth. If treatment is indicated, simple incision or **"deroofing"** is all that is needed.



Dentigerous cyst

Eruption cyst

Which **muscle** is responsible for the forward displacement of the condylar head when the neck of the condyle is fractured?

- Masseter muscle
- Mylohyoid muscle
- Lateral pterygoid muscle
- Medial pterygoid muscle

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• **Lateral pterygoid muscle**

Three groups of muscles are responsible for the **displacement** of mandibular condyles:

Group I → masseter, medial pterygoid & temporalis. They elevate the mandible during mastication and will cause an upward displacement of the proximal segment.

Group II → digastric, mylohyoid, geniohyoid & lateral pterygoid. They depress the mandible and will displace the distal fractured segment inferiorly and posteriorly.

Group III → lateral pterygoid. When the neck of the condyle is fractured, this muscle will cause forward displacement of the condylar head.

All of the following are true statements concerning the principles of suturing technique **except:**

- The needle should be perpendicular when it enters the tissue
- Sutures should be placed at an equal distance from the wound margin *(2–3 mm)* and at equal depths
- Sutures should be placed from mobile tissue to thick tissue
- Sutures should be placed from thin tissue to thick tissue
- Sutures should not be overtightened
- Tissues should be closed under tension
- Sutures should be 2–3 mm apart
- The suture knot should be on the side of the wound

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• **Tissues should be closed under tension**

***This is **false;** sutures **should not** be overtightened or closed under tension.

The **interrupted suture** is the most common suture method. Because each suture is independent, this procedure offers strength and flexibility in placement. Due to this advantage, if one suture is lost or becomes loose, the integrity of the remaining sutures is not compromised. The **major disadvantage** is the time required for placement of this pattern of sutures. *(See figure #1 below).*

Advantages of a **continuous** pattern or method *(See figure #2 below):*
• Ease and speed of placement
• Distribution of tension over the whole suture line
• A more watertight closure than the interrupted pattern or method

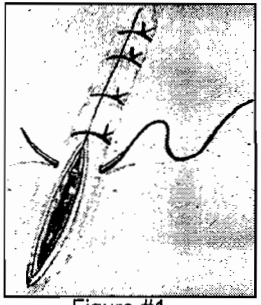
Figure #1

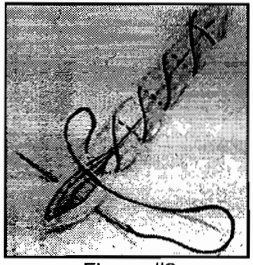
Figure #2

A position in which the patient is on an elevated and inclined plane, usually about 45°, with the head down and legs and feet over the edge of the table is called the:

- Lithotomy position
- Flank position
- Trendelenburg position
- Valentine position

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• **Trendelenburg position** → see picture below

It is used in abdominal operations to push abdominal organs towards the chest. This position is also usually used in **treating shock**, but if there is an associated head injury, the head should not be kept lower than the trunk.

Important: In anaphylaxis the patient should be placed in the Trendelenburg position.

In reference to the bone-implant interface, which of the following yields the most predictable long-term stability?

- Fibro-osseous integration
- Osseointegration
- Biointegration

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• **Osseointegration**

The bone-implant interface:

Fibro-osseous integration

- Connective tissue-encapsulated implant within bone
- Success rate of 50% over 10-year period
- Not seen often with newer materials

Osseointegration

- A direct structural and functional connection between living bone and the surfaces of a load-carrying implant without soft-tissue
- Yields most predictable long-term stability
- Several important factors involved → materials, surface characteristics, bone, timing

Biointegration

- Implant interface that is achieved with bioactive materials such as hydroxyapatite *(HA)* or bioglass that bond directly to bone.
- HA coated implants appear to develop bone faster than do non-coated implants but, after a year, there is little difference between coated and non-coated.

Notes:

1. To ensure the development of keratinized tissue around a dental implant the best time to augment the soft tissue is **Stage II surgery.**
2. **Guided tissue regeneration** is a surgical procedure used to eliminate a bony defect around a dental implant. This process decreases the connective tissue growth while increasing the growth of bone in the defect.
3. A gentle surgical technique requires that you **do not heat bone** above 116°F. Above this temperature, bone tissue damage occurs.

To prevent infective endocarditis in patients at risk for such infections, the American Heart Association *(AHA)* frequently issues guidelines for prophylactic antibiotic coverage during dental procedures. In accordance with the most recently revised AHA guidelines, which of the following **is not** an acceptable antibiotic for the prevention of infective endocarditis?

- Cephalexin
- Amoxicillin
- Clarithromycin
- Erythromycin
- Azithromycin
- Clindamycin

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• **Erythromycin**

In **adults,** the new antibiotic regime recommended for the prevention of infective endocarditis is:

- Amoxicillin: 2.0 grams, 1 hour prior to the procedure *(four 500 mg tablets)*

For those patients **allergic** to penicillin,

- Clindamycin: 600 mg, 1 hour to the procedure *(four 150 mg tablets)*

The guidelines for **children** are:

- Amoxicillin: 50 mg/kg, 1 hour prior to the procedure

For those patients **allergic** to penicillin,

- Clindamycin: 20 mg/kg, 1 hour prior to the procedure

These **new** guidelines involve a **number of changes** from the previous set of guidelines:

- Only one antibiotic dosage is required.
- The recommended antibiotic for penicillin-allergic patients is clindamycin **not** erythromycin
- Prophylaxis is no longer required for many dental procedures

Alternatives for patients who are **allergic to penicillin** and who **cannot take clindamycin** include cephalexin, clarithromycin, and azithromycin.

OPERATIVE

Legend

Major Topic	Abbreviation
Amalgam	Amal
Bases / Liners / Cements	B / L / C
Caries	Caries
Composites	Comp
Gold– Direct and Indirect	Gold
Instruments / Burs	Instr / Burs
Miscellaneous	Misc.
Pins	Pins
Rubber Dam	R Dam
Sealants / Fluoride	S / Fl
Terms	Terms

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OPERATIVE

B / L / C

After the initial setting period, _____ tend to have the least solubility, and _____ the most.

- Zinc phosphate cements, Zinc polycarboxylate cements
- Zinc polycarboxylate cements, Glass ionomer cements
- Glass ionomer cements, Zinc polycarboxylate cements
- Glass ionomer cements, Zinc phosphate cements

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- **Glass ionomer cements, Zinc polycarboxylate cements**

***Zinc polycarboxylate** cements are the most soluble *(they are slightly more soluble than zinc phosphate cements).*

Glass ionomer cements are hybrids of silicate and polycarboxylate cements designed to combine the **fluoride releasing properties** of silicate particles with the chemically adhesive and more biocompatible characteristics of the **polyacrylic acid matrix** compared to the extremely acidic matrix of silicate cement.

Advantageous physical properties of glass ionomer cements:

- Release of **fluoride** → anticariogenic
- **Chemical adhesion** to the prepared tooth and certain metals. **Micromechanical bond** to composite resins. **Important:** Chelation of calcium ions on tooth structure by ionized polyacrylic acid side-groups is the principal mechanism of chemical adhesion to tooth structure.
- **Biocompatibility is high,** thus with enough dentin remaining *(0.5-1 mm)* no pulpal protective agent *(calcium hydroxide)* is required.
- **Good thermal insulators** → equal to that of natural dentin
- **Thermal expansion** is similar to that of tooth structure
- **After initial setting,** they have **low solubility in the mouth**

Note: Its **disadvantage** as a cement is that it has a **higher cement film thickness** than zinc phosphate cements.

Remember: No lab test of cement has correlated solubility with clinical retention.

Generally, glass ionomer cements contain:

- Zinc oxide and distilled water
- Fluoro alumino-silica powder and orthophosphoric acid
- Zinc oxide and polyacrylic acid
- Fluoro alumino-silica powder and polyacrylic acid

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• **Fluoro alumino-silica powder and polyacrylic acid**

Glass ionomer cements are mixed powder-liquid component systems. The powder is a **fluoro alumino-silicate glass** that reacts with a liquid which is **polyacrylic acid** to form a cement of glass particles surrounded by a matrix of fluoride elements.

When set, glass ionomer cements have the potential to inhibit the development of recurrent caries at its margin as a result of fluoride release from its surface. In **addition,** glass ionomer cements have the potential to absorb fluoride when local ionic concentrations are high and then release the fluoride slowly when the environmental concentration decreases, **thus acting** as a fluoride sponge.

Classification of glass ionomer cements:

1. **Conventional** → used as a luting agent. **Examples:** Ketac-Cem and G.C. Lining Cement.
2. **Light-cured** → used as a base or liner. **Advantages include:** an extended working time, short on-demand setting times, a set mass that is stronger, more adhesive, and more resistant to desiccation than self-cured glass ionomers. **Note:** Liquid has HEMA added to it. **Examples** include Vitremer and XR Ionomer
3. **Resin-modified** *(Hybrid)* **light-cured** → can be used for any application where glass ionomers are good choices. Combine an acid-base reaction of the traditional glass ionomer with a self-cure amine-peroxide polymerization reaction. **Note:** Liquid has HEMA added to it. **Examples:** RelyX, formerly known as Vitremer Luting Cement and Fuji Plus.

OPERATIVE

B / L / C

The ph of ZOE cements is:

- Near 1
- Near 10
- Near 3
- Near 7

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• **Near 7** → which makes it one of the least irritating of the dental cements

Zinc oxide-eugenol cement is a soft, sedative - type cement. It is used as a **sedative** or **temporary** filling material, as an insulative base, and in interim caries treatment. The powder is zinc oxide and the liquid is eugenol. Eugenol has a **palliative effect** upon the dental pulp, and this is one of the **main advantages** of using this type of cement.

A conventional mixture of zinc oxide and eugenol is relatively weak. In recent years "reinforced" or "improved" zinc oxide-eugenol cements have been introduced *(called reinforced* **ZOE** *or* **ZOE-EBA***)*. In **reinforced ZOE** *(Type III ZOE)* the powder is composed of zinc oxide and finely divided polymer particles *(polymethyl-methacrylate)* in the amount of 20 to 40% by weight. **In addition,** the zinc oxide powder is **surface treated** by an aliphatic monocarboxylic acid, such as propionic acid. **Note:** This combination of surface treatment and polymer reinforcement results in a material that has good strength and toughness which markedly improves abrasion resistance. Reinforced ZOE is fine for basing large and complex cavities. This material is able to withstand the pressure of amalgam condensation and it has minimal effect on the pulp.

Contraindications to the use of ZOE include:

1. On dentin or enamel prior to bonding → compromises bonding.
2. As a **base or liner for composite resins** → eugenol interferes with polymerization.
3. Patients who are **allergic to eugenol** *(or oil of cloves)* → this is somewhat common.
4. Direct pulp capping → eugenol is a pulpal irritant when in **direct pulpal contact.**

Remember: ZOE is soluble in oral fluids and is **difficult** to remove from cavity preparations.

OPERATIVE

B / L / C

IRM *(Intermediate Restorative Material)* powder and liquid should be mixed in:

- Less than three minutes
- Less than one minute
- Less than two minutes
- Any of the above

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- **Less than one minute**

***The resulting putty consistency is then inserted into the cavity Zinc Oxide and Eugenol *(ZOE)* is not very durable, and it wears away after just a few weeks, but it works to relieve pain, calm the nerve and protect the tooth. During the Vietnam war, the US Army invented a more durable form of ZOE called **Intermediate Restorative Material** *(IRM)* which is fortified with plastic powder.

Uses:

- As an intermediate restorative material for both Class I and II restorations.
- As a base under non-resin restorations
- Restoration of deciduous teeth *(when permanent teeth are two years or less eruption*
- Restorative emergencies

Advantages:

- High strength comparable to zinc phosphate
- Excellent abrasion resistance
- Good sealing properties
- Low solubility

Important: Because of its zinc-oxide eugenol composition, **IRM will interfere** with subsequent placement of a resin filling.

OPERATIVE

B / L / C

Zinc phosphate cement will:

- Shrink slightly upon setting
- Expand slightly upon setting
- Shrink immensely upon setting
- Expand immensely upon setting

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- **Shrink slightly upon setting**

***The cement shrinks much more when it is in contact with air than when it is under water. Thus, the cement should not be allowed to dry out.

Zinc phosphate cement is the oldest of the luting cements and thus is the one that has the longest "track record" and serves as the standard to which newer systems can be compared. It is a powder-liquid system; the powder is mostly **zinc oxide** *(also consists of magnesium oxide in the approximate ratio of 9 to 1)* and the liquid is **orthophosphoric acid.** The primary use of zinc phosphate cement is as a **luting agent** for the cementation of cast restorations. It can also be used as a base material when a high compressive strength is needed.

Important: The initial mixture of this cement is **very acidic *(3.5)* and can cause irreversible pulpal damage** if a cavity varnish *(2 coats)* is not placed on the tooth **prior to cementation** of the crown.

It has **superior strength** compared to other cements, and its retention is dependent upon **mechanical interlocking** *(as opposed to glass ionomer and polycarboxylate cements which adhere to tooth structure by virtue of the polyacrylic acid in the liquid).*

Notes:

1. Zinc phosphate cement liquid that has lost some of its water content will cause the setting time of the mix to be **lengthened.**
2. Mixing zinc phosphate cement very rapidly will **decrease** the final compressive strength of the cement.

OPERATIVE

B / L / C

When mixing **zinc phosphate cement**, a cool glass slab is used to:

- Decrease the working time by retarding the exothermic chemical reaction
- Decrease the working time by accelerating the exothermic chemical reaction
- Increase the working time by retarding the exothermic chemical reaction
- Increase the working time by accelerating the exothermic chemical reaction

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• **Increase the working time by retarding the exothermic chemical reaction**

Mixing procedure for zinc phosphate cements:

- A **cool mixing slab** should be used. **Caution:** The temperature of the slab should **not be below** the dew point of the room.
- Mixing should be started with the addition of a small amount of powder to the liquid. This procedure, along with the cool slab, **increases the working time.**
- Small increments of powder are added approximately every 20 seconds with vigorous mixing until a creamy consistency is achieved. This will promote a **high powder-liquid ratio** and a superior cementation medium by providing the following:
  → a **lower viscosity** of the mix
  → a **stronger** final set
  → a **lower solubility** of the set cement

***Important point:** The **advantages of using the cool slab method** are a substantial increase in the working time of the mix on the slab and a shorter setting time of the mix after placement in the mouth.

Some Trade names of Various Dental Cements Commonly Used in Practice

Glass Ionomer	Resin Modified Ionomer	Resin Cement Chemically Cured	Resin Cement Dual Cured	Zinc Phosphate	Zinc Poly-carboxylate	Zinc Oxide-Eugenol
Fuji *(GC)*	Advance *(Caulk)*	Panavia 21 *(J. Morita)*	Resinomer *(Bisco)*	Tenacin *(Caulk)*	Durelon *(ESPE)*	Tem-Bond *(Kerr)*
Ketac-Cem *(ESPE)*	Vitremer Luting *(3M)*	Clearfil CR Inlay *(J. Morita)*	Enforce *(Dentsply)*	Fleck's *(Mizzy)*	Tylok Plus *(Caulk)*	Fynal *(Caulk)*

OPERATIVE

B / L / C

All of the following statements are true regarding glass ionomer restorations **except:**

- Glass ionomer is often the ideal material of choice for restoring root surface caries in patients with high caries activity
- The best surface finish for a glass ionomer restoration is that obtained against a surface matrix
- Glass ionomer adheres to mineralized tooth tissue
- Glass ionomers are somewhat esthetic and polish much better than composites

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- **Glass ionomers are somewhat esthetic and polish much better than composites**

***It is true that glass ionomers are somewhat esthetic, however, they **do not polish** as well as composites.

Both self-cured and light-cured versions of glass ionomers are available. Light-cured glass ionomers are preferred because of both the extended working time and their improved physical properties. Because of their **limited strength** and **wear resistance,** glass ionomers are indicated generally for the restoration of **low stress areas** where caries activity potential is of **significant concern.**

Compared to composites, **glass ionomers:**
- Have a **lower** compressive strength, tensile strength, and hardness.
- Are generally **very technique sensitive** because of their high solubility when first mixed.

Note: With the newer hybrid or light-cured resin-modified glass ionomers, the above properties **have been improved.**

Glass ionomers are generally considered the nearly ideal base / liner material because of the following properties:
- **Adhesive bond** to tooth structure
- **Snap set** in the light-cured form *(for example, Vitrebond)*
- **Anticariogenic** → due to fluoride release
- **Bond to composite** → makes for excellent liners for Class V root caries restorations. Sometimes called the **"sandwich technique".** This technique achieves all the benefits of the glass ionomer cements plus the high polishability, surface hardness, and strong bond to enamel of the composite resin.

OPERATIVE

B / L / C

Which of the following is used as both a restorative agent and a cement?

- Zinc polycarboxylate
- Zinc phosphate
- Glass Ionomer
- Zinc oxide-eugenol

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• **Glass Ionomer**

***Only glass ionomer** is used as a cement *(luting agent)* and a permanent restorative material. Glass ionomer cements are often used for root surface carious lesions because of the potential advantage of fluoride release in helping to control the spread of caries.

Commonly Used Dental Cements	
Uses	**Cements**
Luting	Zinc Phosphate, Zinc Polycarboxylate, Glass Ionomer
Temporary Luting or Temporary Restoration	Zinc oxide-eugenol, Zinc oxide EBA *(ethoxy benzoic acid)*
Pulp Capping & Thermal-Insulating Base	Calcium hydroxide, Zinc oxide-eugenol
Cavity Liners and Bases	Glass Ionomer, Polycarboxylate, Zinc oxide-eugenol
Cementing Veneers & Composite Inlays	Resin cements
Core Buildup Under a Crown or Bridge Preparation	Metal-modified glass-ionomer

Note: ZOE, reinforced ZOE, ZOE-EBA, silicate, and zinc silicophosphate cements are no longer routinely used to permanently cement restorations. **Zinc phosphate cement** has been extensively replaced by polycarboxylate or glass ionomer cements. These cements are based on ion-crosslinked **polyacrylic acid** matrices that have the potential to react chemically with residual powder particles and the surface of tooth structure.

Zinc polycarboxylate cement was a truly innovative development in dentistry because it was the first material to:

- Mechanically bond to tooth structure
- Chemically bond to tooth structure
- Have a significantly greater compressive strength as compared to zinc phosphate cement
- Have a much lower film thickness as compared to zinc phosphate cement

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- **Chemically bond to tooth structure**

Zinc polycarboxylate cement was the first system developed with a potential for adhesion to tooth structure via chelation. The polycarboxylate cements are powder / liquid systems. The liquid is an aqueous solution of **polyacrylic acid** and **copolymers**. The powder is **zinc oxide** and **magnesium oxide.**

Zinc polycarboxylate cements have a compressive strength slightly lower than that of zinc phosphate while the **tensile strength is higher.** Its final strength is dependent on the powder / liquid ratio, with more powder giving greater strength. The strength of the set material is **sufficient** for amalgam condensation and its effect on the pulp is mild enough to eliminate the need for sublining. Thermal conductivity is **low** and thus the material gives good protection against thermal stimuli applied to metallic restorations.

An **advantage** of zinc polycarboxylate cement is that it can bond to tooth structure → this is attributed to the ability of the carboxylate groups in the polymer molecule to **chelate to calcium** in the tooth. The most commonly noted **disadvantages** of polycarboxylate cement is its **marked thickness** and **short working time.** Currently marketed brands include Durelon *(3M ESPE)*, Liv Carbo *(GC America)*, Shofu Polycarboxylate *(Shofu)*, and Tylok Plus *(Dentsply/Caulk)*. The most recent innovation in these cements has been the development of Durelon Maxicap, an encapsulated version of Durelon. Because it is mixed and expressed from a capsule, the traditional difficulties of short working time and excessive thickness are overcome.

Remember: When cementing a cast restoration, always apply cement to both restoration and the tooth.

OPERATIVE

B / L / C

If a **zinc phosphate cement base** is used when restoring a tooth, when should the varnish be applied?

- **Prior** to placement of the base
- **After** placement of the base
- **Makes no difference** when the varnish is applied
- Varnish should **not be used** in conjunction with zinc phosphate cement

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• **Prior to placement of the base**

It should be emphasized that the use of a base in conjunction with amalgam or gold foil **does not alleviate** the need for a varnish as an aid in sealing the cavity margins against leakage. However, the type of base governs the respective order of application of the varnish and the base. If a zinc phosphate cement base is to be used, then the cavity varnish should be applied to the cavity walls **prior to** placement of the base. On the other hand, if a biocompatible agent *(e.g., a calcium hydroxide, zinc oxide-eugenol, or polycarboxylate cement base)* is employed, then these should be placed against the dentin, and the varnish should not be applied **until the base material** has hardened. **Important:** If varnish is added **before** a biocompatible base it may prohibit positive qualities such as eugenol's soothing effect or polycarboxylate's chelation and adhesion.

Zinc phosphate cements provide good pulpal protection from thermal, electrical, and pressure stimuli, but may damage the pulp as a result of an initial low pH. This, however, can be of benefit as it provides an antibacterial effect which reduces the number of viable microorganisms in the cavity floor and thus decreases pulpal irritation.

Important: Cements used for **bases** should be mechanically stronger than when used as luting agents and are mixed with the **maximum powder content** that is possible. A **low powder-to-liquid ratio** produces a low viscosity cement that is needed for **luting agents.**

Note: The varnish will **reduce the initial microleakage** of an amalgam restoration.

OPERATIVE

B / L / C

The thermal insulating capacity of a cement base is principally related to:

(A) Thickness of the base placed

(B) Coefficient of thermal expansion of the set base

(C) Thermal conductivity of the set base

(D) Powder / liquid ratio of the cement mix

(E) Density of the base placed

- A, B, C
- B, D, E
- A, C, E
- A, C, D

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• **A, C, E**

The most important consideration for **pulp protection** in restorative techniques is the **thickness of** the remaining dentin.

In general, cements that are thicker than 2 mm are termed bases and as such function to **replace lost dentin structure** beneath restorations. A base may be used to provide thermal protection under metallic restorations, to increase the resistance to the forces of condensation of amalgam, or to block out undercuts when taking impressions for cast restorations.

Important: A base **should not** be used unnecessarily.

Remember: Calcium hydroxide is very effective in promoting the formation of secondary dentin, which is an important aid in the repair of the pulp.

***The only distinction between a base, a cement, and a cavity liner is their final **application thickness.**

1. **Cements** for luting have a desired final film thickness of **15 to 25 microns.**
2. **Cavity liners** *(either solution or suspension liners)* have a desired final film thickness of **5 microns.**
3. **Bases** have a final application thickness of **1-2 mm** *(they may be thicker depending on the amount of dentin that has been destroyed).*

Note: The selection of appropriate bases and liners to restore the axial wall of a Class II restorations is dependent upon the biological effect required and the **thickness of the remaining dentin.**

OPERATIVE

B / L / C

Which of the following properties is **most characteristic** of the current cement bases?

- High coefficient of thermal expansion
- High ductility
- Adhesion to tooth structure
- Low thermal conductivity

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- **Low thermal conductivity**

It is common practice to insert a **low thermal conducting cement material** as a base in deep portions of cavities underlying metallic restorations to insulate the pulp against thermal and electrical shock and pressures induced during placement of the restorations. ***Bases** in essence serve as a replacement or substitute for the protective dentin that has been destroyed by caries and / or cavity preparation.

Materials that have been employed as bases *(bases are typically 1-2 mm thick):*

- Zinc phosphate cement → remember to seal dentinal tubules with varnish **prior** to application
- Zinc polycarboxylate cement → provides adhesion
- ZOE
- Glass ionomer cement → provides fluoride release and adhesion
- "Hard setting" calcium hydroxide materials → thicker than when used as a liner

Remember: All of the above are suitable as a base under amalgam restorations, however, for **composites**, ZOE **cannot be used** because the eugenol will inhibit the composite setting reaction.

Bases are classified as either primary or secondary:

- **Primary bases** are placed on the dentin in close proximity to the pulp primarily to provide protection from toxic and thermal irritants. Under **amalgam and tooth-colored restorations**, the primary base is usually **calcium hydroxide**, whereas, under gold restorations, the primary base is usually zinc phosphate cement or zinc polycarboxylate cement. Glass ionomers are commonly used today as well.
- The most common use of a **secondary base** is the placement of zinc phosphate cement over a **calcium hydroxide base** which has been placed over a pulpal exposure *(direct pulp cap)*.

Note: A primary base is **not needed** under zinc polycarboxylate cements that are used to base to ideal form since these cements are **non-irritating to the pulp.**

OPERATIVE

B / L / C

The main function of cavity liners is to :

- Act as thermal insulators
- Provide a barrier against chemical irritation
- Produce a structural form for the cavity preparation
- Resist forces applied during condensation of the restorative material

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• **Provide a barrier against chemical irritation**

Cavity liners are materials that are placed as thin coatings over exposed dentin. Their main purpose is to **protect** the pulp by creating a barrier between the dentin and pulpally irritating agents *(i.e., acids from etchants or cements, restorative materials, etc.)* by **sealing** the dentinal tubules.

Cavity liners are usually classified into two main groups:

1. The typical **cavity varnish** *(also called solution liner)* is principally a natural gum, such as copal, rosin, or a synthetic resin dissolved in an organic solvent such as acetone, chloroform, or an ether. **Copalite** is the most commonly used.
2. The typical **suspension liner** is a liquid in which **calcium hydroxide,** and occasionally **zinc oxide,** is suspended in a solution of natural or synthetic resins. **Dycal** *(calcium hydroxide)* is most commonly used.

Notes:

1. **Suspension liners** are thicker *(20–25 microns)* than solution liners *(1–5 microns).*
2. **Solution liners** *(Copalite)* have been shown to reduce the **initial microleakage** of a restoration. **Important:** This type of liner should not be used under a composite, they will **inhibit** the polymerization of the resin.

***These liners** *(both solution and suspension)* are being replaced by the new dentin bonding agents. **Remember:** Zinc phosphate cement may also be used as a liner when a particularly strong one is needed. It should always be preceded by **at least two coats** of cavity varnish to prevent pulpal irritation. When using polycarboxylate cement as a liner, this is not necessary since this cement is non-irritating to the pulp → apply varnish after placing the polycarboxylate cement liner.

Cavity varnishes used in conjunction with amalgam restorations:

- Provide thermal insulation
- Act as antibacterial agents
- Promote healing of the pulp
- Reduce microleakage

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- **Reduce microleakage**

The three most commonly used cavity lining agents are:

1. **Cavity varnish** *(solution liner)* → seals dentinal tubules without adding bulk
   - **Reduces marginal leakage** → improves the marginal seal for the short term
   - Helps prevent penetration of acid
   - Protects pulpal tissue from the phosphoric acid in zinc phosphate cements
   - Prevents mercury penetration into the dentinal tubules from amalgam restorations → prevents discoloration of dentin

Note: Cavity varnish **does not** act as a thermal barrier. It should **not be** used with composite restorations since in will inhibit polymerization.

2. **Zinc oxide eugenol** *(suspension liner)*
   - Prevents penetration of acids
   - **Prevents thermal shock**
   - Has adequate strength so that it can be used under permanent restorations

Note: ZOE is a particularly good liner due to the fact that it is **palliative** *(soothing)* to the pulp. Commonly used for **temporary fillings.**

3. **Calcium hydroxide** *(suspension liner)*
   - **Prevents thermal shock**
   - Prevents passage of acid from restorative materials
   - Has enough strength to resist forces used in placing restorations

Important: Calcium hydroxide has the **ability to stimulate the formation of secondary dentin** when placed near or in direct contact with the pulp. Also, when viewed radiographically, calcium hydroxide can be easily confused with caries → **both are radiolucent.**

What is the outermost zone of carious dentin which still is capable of remineralization?

- Normal dentin
- Sub-transparent dentin
- Transparent dentin
- Turbid dentin
- Infected dentin

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- **Transparent dentin**

Zones of carious dentin → from innermost to outermost:

- **Zone 1** *(normal dentin)* → totally normal dentin with no bacteria in the tubules.
- **Zone 2** *(sub-transparent dentin)* → zone of demineralization created by the acid from caries. Damage to the odontoblastic process is evident, however, no bacteria are found in this zone. **Capable of remineralization.**
- **Zone 3** *(transparent dentin)* → softer than normal dentin, shows further demineralization. No bacteria are present. **Capable of remineralization.**
- **Zone 4** *(turbid dentin)* → is the zone of **bacterial invasion,** tubules are filled with bacteria. Zone is **not capable of remineralization** and must be removed prior to restoration.
- **Zone 5** *(infected dentin)* → the outermost zone, consists of decomposed dentin that is **filled with bacteria.** Must be totally removed prior to restoration.

<u>Four zones of an incipient lesion in enamel:</u>

1. **Translucent zone** → the deepest zone, represents the **advancing front** of the enamel lesion.
2. **The dark zone** → does not transmit polarized light. Areas of demineralization and remineralization.
3. **The body of the lesion** → the largest portion of the incipient lesion. Area of demineralization.
4. **The surface zone** → relatively unaffected by the caries attack.

Caries that is often found in older patients and **attacks the cementum** and radicular *(root)* dentin is called?

- Residual caries
- Secondary *(recurrent)* caries
- Root surface *(senile)* caries
- None of the above

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- **Root surface *(senile)* caries**

***Root surface caries are sometimes referred to as **senile caries.**

The rising incidence of root surface caries can be attributed to the aging of populations and the fact that most adults are retaining more teeth. In this population, there is **increased gingival recession** with exposure of root surfaces, leading to the development of root surface caries. Root surface caries generally spreads more on the surface, rather than in depth. In older patients, rampant caries can be caused by poor oral hygiene, decreased salivary flow, and side effects of medications. **On a dental radiograph,** root surface caries appears as a cupped-out or crater-shaped radiolucency just below the cemento-enamel junction *(CEJ)*. Early lesions may be difficult to detect on a dental radiograph

Remember: Glass ionomer is a **desirable restorative material** for root surface caries where esthetics is not a major factor.

Notes:

1. **Residual caries** is caries that remain in a completed cavity preparation, whether by dentist intention or by accident.
2. **Secondary** *(recurrent)* **caries** is decay appearing at the margins of a restoration and under it.
3. The **etiology of root surface caries** is now-a-days believed to be the same as for coronal caries → S. mutans, S. sanguis, A. viscosus, A. naeslundii, Lactobacillus, and Veillonella.

Chronic caries is characterized by all of the following **except:**

- Common in adults
- Extrinsic pigmentation
- Slowly progressing or arrested
- Pain is common

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- **Pain is common**

***With chronic caries, pain is **uncommon.**

Chronic caries is sometimes referred to as slow or arrested caries:

Other characteristics include:

- The lesion is **shallow** *(small lesion)*
- The entrance to the lesion is **wide**
- Dark pigmentation with leathery dentin

***Chronic caries should be **completely removed** when found in enamel and close to the DEJ.

Acute caries which is sometimes referred to as rampant caries, is characterized by:

- The entrance to the lesion is **small**
- The lesion is **deep and narrow** *(large lesion)*
- **Pain** may be a feature
- **Little or no** staining
- Most frequently found in **children**
- Often multiple, soft-to-the-touch lesions
- **Rapidly** progressing

Note: Changes of the pulp and dentin depend on the rate of the carious progression. The **response** of the pulp to carious attack or the trauma of operative procedures depends on the **blood supply** of the pulp and its **cellular activity.**

Defense mechanisms of the pulp *(to protect it from irritation):*

- Sclerotic dentin *(peritubular dentin formation)* → initial defense
- Reparative dentin *(irritation dentin formation)* → second line of defense
- Its vascularity *(inflammation)*

Which of the following is formed in large quantities following the degradation of sucrose by Streptococcus mutans?

- Acetic acid
- Lactic acid
- Butyric acid
- Propionic acid

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• **Lactic acid**

Pit and fissure caries has the highest prevalence of all dental caries. Smooth surface areas, especially the **proximal enamel surfaces** immediately gingival to the contact area are the second most susceptible areas to caries. Streptococci and lactobacilli species are common in this area. The **facial and lingual root surfaces** may have plaque containing filamentous actinomyces species which can cause root surface caries. **Remember:** Lactobacillus species **do not produce** dextran *(as do Streptococci species)* but produces a different extra-polysaccharide called **lexan.** The way that the lactobacillus species cause dental caries in the pit and fissure areas is that it gets packed into those pit and fissure areas thereby exerting its effect.

Flouride treatments will dramatically reduce **smooth surface caries** though they are not as effective in preventing pit and fissure caries. Sealing the pits and fissures just after tooth eruption may be the single most important procedure to help protect these areas from caries destruction.

Remember: Dental caries is **initiated** at the tooth surface as a result of the growth of streptococci, specifically S.mutans, S.mitis, S.sanguis *(which is the most frequently isolated Streptococcus in the oral cavity)*, and S.salivarius. These bacteria produce **dextran sucrase** *(also called glucosyltransferase)*, which catalyzes the formation of extracellular **glucans** *(dextrans and mutans)* from **dietary sucrose**. Glucan production contributes to the formation of dental plaque. This dental plaque holds the **lactic acid** which is produced by these Streptococci against the tooth. This acid eats through the enamel of the tooth, creating caries.

<u>Important: Predominant bacteria found in dental plaque:</u>
- Streptococcus sanguis *(found the earliest)*
- Actinomyces viscosus and naeslundii
- Streptococcus mutans, mitis, and salivarius
- Veillonella, Lactobacilli, and Fusobacterium

Which of the following **is not** an essential factor needed for the initiation of a carious lesion?

- Susceptible host *(tooth)*
- Microflora with cariogenic potential *(plaque)*
- Saliva
- Suitable substrate *(dietary carbohydrates)*

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• **Saliva**

Dental caries is an infectious microbiological disease that results in the localized dissolution of tooth structure. For caries to occur, a susceptible host *(a tooth)*, microflora with cariogenic potential *(plaque)*, and a suitable substrate *(dietary carbohydrates)*, all interact to promote the severity of the disease.
Note: A certain period of time is also required for caries to develop.

The greatest percentage of tooth loss in the first two decades of life *(except from the natural loss of deciduous teeth)* is due to **untreated dental caries.** The rate at which the carious destruction of dentin progresses tends to be slower in older adults than in young persons due to **generalized dentinal sclerosis** which occurs with aging.

Notes:

1. Flouride and occlusal sealants modify the susceptible host *(tooth)*.
2. **Saliva helps prevent caries** by diluting acid and acting as a reservoir for Ca, PO_4, fluoride and other ions for remineralization and hypermineralization of the enamel. It also affects caries through its antimicrobial properties.
3. Enamel **demineralization** occurs at pH 5.5. **Remineralization** of the damaged tooth structure occurs as the pH rises above 5.5.
4. The **prevalence of caries** has been declining in children. A decline in adult caries is not as evident. **Fluoridation** has received the most credit for the decline in the development of caries.
5. Pregnant patients, compared with similar non-pregnant patients, **are likely** to have the same degree of dental caries, but more inflamed gingival tissues.
6. In order to create **smooth surface caries,** a microorganism must be able to produce **dextran,** which is an extrapolysaccharide which enables it to attach to the tooth surface. Dextran is a similar product to that which is produced by barnacles to allow them to attach to the bottom of a ship. It is a very tenacious sticky material. Some members of the streptococci family are able to produce enough dextran to attach to the tooth's surface.

There is abundant evidence that **the initiation of dental caries** requires a high proportion of:

- Staphylococcus aureus within saliva
- Streptococcus mutans within dental plaque
- Streptococcus mutans within food
- Staphylococcus aureus within dental plaque

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• **Streptococcus mutans within dental plaque**

The **first event** in the development of caries is the deposit of **plaque** on the teeth. **Dental plaque** is a highly organized gelatinous mass of bacteria that adheres to the tooth surface. **Streptococcus mutans** produce great amounts of lactic acid *(acidogenic)*, are tolerant of acidic environments *(aciduric)*, are vigorously stimulated by sucrose, and appear to be the **primary organisms** associated with dental caries, however, they are not the only organisms required for caries initiation. Other mutans streptococci species in humans can do this as well *(for example, S. sobrinus)*.

Factors to which the tooth surface is **directly exposed**, and which contribute to the development of dental caries:

Factor	High Risk
Amount of plaque	Large amount of plaque on teeth, meaning many bacteria that can produce acids (low ph, demineralization)
Type of bacteria	Large proportion of "cariogenic" types of bacteria, resulting in lower ph and sticky plaque and also prolonged acid production
Type of diet	High in carbohydrates, in particular sucrose; "sticky" diet leading to low ph for a longer time
Frequency of carbohydrates	High sugar frequency resulting in longer time per day with low ph
Saliva secretion	Reduced saliva flow leading to prolonged sugar clearance time and to a reduced amount of other saliva protective systems
Saliva buffer capacity	Low buffer capacity resulting in prolonged time with low ph
Fluorides	Absent: reduced remineralization

All of the following are true concerning dental caries **except:**

- Dental caries is an infectious microbiological disease of the teeth that results in localize dissolution and destruction of the calcified tissues
- The evidence for the role of bacteria in the genesis of dental caries is overwhelming
- Mutans streptococci is the principal etiological agent of dental caries
- Organisms which cause caries are called "cariostatic"
- Lactic acid produced by acidogenic bacteria is the main cause of enamel decalcification

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- **Organisms which cause caries are called "cariostatic"**

***Cariostatic** means to inhibit or arrest dental caries formation. **Cariogenic** means conducive to the production of dental caries.

Cariogenic bacteria:

- **Mutans streptococci,** of which Streptococcus mutans and Streptococcus sobrinus are the two most commonly found in man.
- Lactobacilli casei

Note: Most current research suggests that the microbial etiology of root caries is very similar to coronal caries → in the past it was thought that Actinomyces species *(viscosus and naeslundii)* were most commonly associated with root surface caries.

Essential properties of cariogenic bacteria:

- **Acidogenic** *(produce acid)* and **aciduric** *(being able to tolerate an acid environment)*
  ***Note: Lactic acid** is formed in large quantities following the degradation of sucrose by **mutans streptococci.**
- The **ability to attach** to the tooth surface. **Note:** Streptococci species have special receptors for adhesion to the surface and also produce a sticky matrix that allows them to cohere to each other.
- The **ability to form** a protective matrix. **Note:** Streptococci species produce an extracellular insoluble **dextran,** which protects them from being removed from the teeth by saliva, liquids, foods, and masticatory forces. **Remember:** Glucans occur as **dextrans or mutans.** They are synthesized from sucrose by plaque bacteria as extracellular polymers of glucose. **Levans** are polymers of fructose.

Dental plaque describes the soft white film of organized bacterial colonies *(main component),* salivary glycoproteins, and inorganic material that readily forms on the surface of teeth. **Note: The strong correlation between the presence of dental plaque and the appearance of dental caries and periodontal disease has been recognized for many years.**

All of the following are **advantages of the visible light curing systems** compared to the old ultraviolet light curing systems **except:**

- A greater depth of resin can be cured by visible light
- The resin can be polymerized through enamel, which is particularly advantageous in Class III restorations
- The intensity of visible lights remains relatively constant until the bulb fails completely
- The light tip is placed against the surface of the restoration and activated in order to get the best cure of the material

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- **The light tip is placed against the surface of the restoration and activated in order to get the best cure of the material**

***This is **false;** the light tip is placed very close to, **but not against,** the surface of the restoration and activated.

Visible light curing systems have **totally displaced** the UV light systems. Also, visible light curing systems are much more widely used than the chemically activated ones *(self cured)*. An **advantage** of light curing systems as a whole is that the dentist has complete control over the working time and is not confined to the built-in curing cycle of the self-cure. This is particularly beneficial when large restorations are placed.

Note: To deal with problems of incomplete curing with VLC due to the thickness of restorations and filler particles scattering light, manufacturers have developed composite resins that are **dual-cured** which combines self-curing and visible light-curing. Another polymerization method is **staged curing** which is a two-staged cure. **However,** VLC composites are still the most popular today.

Remember: Visible light cured composites are single component pastes, and the polymerization process is activated by an external energy source. The **alpha-diketone initiator** *(generally camphor quinone)* absorbs energy from a visible *(474 nm–blue light)* light source. The ketone absorbs energy and reacts with an **amine** *(added to the system to enhance the effect of the light-sensitive catalyst)* to produce free radicals.

All of the following statements are true concerning **posterior** composite restorations **except:**

- Posterior composite restorations are frequently **indicated** in the treatment of occlusal lesions which allow conservative preparations
- Posterior composite restorations are **contraindicated** in a patient with heavy occlusion *(bruxism)*
- Posterior composites are **indicated** for cusp replacement as long as a dry operating field is maintained
- Posterior composite restorations may be **indicated** for the restoration of Class II cavities in premolar teeth where the appearance is very important, the cavity margins are in the enamel, and the occlusal contacts are on the enamel

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- **Posterior composites are indicated for cusp replacement as long as a dry operating field is maintain**

Although the ADA does not endorse composite resins as a substitute for amalgam in posterior teeth, composite restorations can be excellent if strict guidelines are followed for tooth selection and if the restorations are done properly. But remember, **composite resin restorations** are inferior to amalgam in terms of compressive strength and abrasion resistance *(occlusal wear)*. Also, current composite resins have no capability to provide an anticariogenic effect as do freshly placed glass ionomer or resin modified glass ionomers, for example.

The most serious **limitation** of the visible light-cured posterior composite restoration is the **polymerization shrinkage,** which can cause internal stresses and gap formations at butt-joint interfaces, which are seen at the gingival floor of Class II and V restorations. **Important:** Polymerization shrinkage in a composite creates stress that can damage surrounding enamel walls of the cavity preparation. The amount of stress created depends upon the **C-factor** *(the ratio of bonded to unbonded surface areas)* of the composite restoration. When the restoration has a high C-factor, the cavity is more likely to be damaged. Incremental curing reduces the C-factor and, therefore, reduces the residual stress of the resulting composite restoration.

Note: The **major indication** for the use of posterior composites is the demand for esthetics by the dentist and patient. Other criteria are non-involvement of cusps, minimal occlusal contact, no excessive wear, and the isthmus **must be** no wider than one-third of the intercuspal distance. **Remember:** Composite is the **material of choice** if the patient has a **documented** allergy to mercury.

Important: In the past, posterior composite restorations were contraindicated in a patient with a caries-active mouth. New concepts stress that you should manage the disease *(i.e., dental caries)* before or at the same time as you are treating the consequence of the disease *(i.e., by placing restorations)*. Therefore, the current literature does not see a special problem for these restorations in caries-active patients. They have as bad a prognosis as any other restorative treatment if the disease is not managed simultaneously.

Which property of **filled resins** is primarily to blame for the failure of **Class II** composite restorations?

- Low flexural strength
- Low compressive strength
- Low tensile strength
- Low wear resistance

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• **Low wear resistance**

Ideally, composite resins should be used only to restore **minimal cavities in posterior teeth.** Its use should be restricted to those instances where it will **not be** subjected to excessive occlusal forces and where, when teeth are in occlusion, there is cusp-to-cusp contact and not cusp to restoration.

For **Class III** preparations using resins, the rule of extension for prevention into embrasures is **disregarded** for Class III esthetic restorations. This compromise is for esthetic reasons, as well as the unnecessary removal of tooth structure which will often involve the incisal edge. If possible, the **outline form** should place the gingival margin incisally from the crest of the gingiva.

When placing the composite resin in a **Class III** preparation, the **wooden wedge** is placed in order to provide some separation of the teeth *(for contact)*, to stabilize the mylar strip, and to avoid creation of excess gingival flash. **Important:** Restoring the contact area must be done properly and diligently.

Remember: For **Class III** composite preparations the **retentive grooves** are placed along the **gingivo-axial** and **inciso-axial** line angles *(entirely in dentin).* These grooves will provide for mechanical lock in the preparation. Small, rounded retentive areas are preferred, as contrasted to sharp angles, since it is difficult to insert viscous composite material into the sharp angles.

Note: Once proper finishing has been completed, a thin layer of unfilled resin can be applied as a glaze *(this seals the margins and smooths the surface).* The difficulty in finishing composite resin restorations is due **primarily** to the softness of the resin matrix and hardness of the filler particles. The most desirable finished surface for composites is **obtained with aluminum oxide disks.**

What mixture of different size particles allows advantages of higher filler levels and good finishing?

- Micro-filled resin based composites
- Flowable resin based composites
- Packable resin based composites
- Hybrid resin based composites

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• **Hybrid resin based composites**

***This resin is highly filled with glass and SiO2 to obtain an optimum of mechanical properties *(greater than micro filled resins)* and an adequate aesthetic appearance. Hence, they are the most commonly applied composite in modern dental practice.

The first composite resins that were developed contained **large filler particles** *(10-100 microns in diameter)* and became known as **macrofill** materials. However, **large size filler particles** reduce the degree of surface smoothness that can be achieved and also reduce the resistance to wear. In **response to this problem,** manufacturers have produced and continue to produce smaller and smaller particles that **result in better finishing characteristics and a greater resistance to wear. Note:** The newer microfilled and hybrid resins are about 40-60% filler by weight as compared to 70-80% filler that is characteristic of the macrofill materials.

Classification of composites:

1. Based on **filler** particle size *(diameter in microns)*
   - **Macrofill** *(traditional composites)* → 10-100
   - **Midifill** *(small particle composites)* → 1-10
   - **Minifill** → 0.1-1
   - **Microfill** *(fine particle composites)* → 0.01-0.1 ***develop **smoothest** finish
   - **Hybrid** → mixture of particles, usually midifill or minifill **with** microfill
2. Based on **polymerization** method: Composite resins are dimethacrylate monomers and polymerize by the addition mechanism that is initiated by **free radicals.** These free radicals can be generated by **chemical activation or external energy** *(heat, light)*.
   - **Chemically activated** *(self-cured)* → two paste system, one contains the **benzoyl peroxide** initiator, the other a tertiary amine activator.
   - **Light-activated** → visible light has replaced UV light. One paste system which contains a diketone **photoinitiator** molecule *(generally camphoroquinone)* and an amine activator.

Composite filler particles function to do all of the following **except:**

- **Increase** the coefficient of thermal expansion
- **Increase** the tensile strength and compressive strength
- **Reduce** the polymerization shrinkage
- **Increase** the hardness
- **Improve** the wear resistance

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• **Increase the coefficient of thermal expansion**

***This is **false;** fillers **reduce** the coefficient of thermal expansion.

Remember: As the overall filler content increases, the physical, chemical, and mechanical properties generally improve. Obviously, there is a limit to the amount of filler that can be added to a resin because as the filler level is increased, the fluidity decreases. **Note: Restorative composites** have a high filler content while **flowable composites** have a low filler content.

Composition of composites *(filled resins):*

- **Filler particles** → the filler particles used are either barium silicate glass, quartz or zirconium silicate,usually combined with 5-10% weight of very small-sized *(0.04 μm)* particles of colloidal silica.
- **Matrix** → difunctional monomers either **BIS-GMA** or urethane dimethacrylate **(UEDMA)**. In some cases, a proportion of a lower molecular weight monomer such as **TEGDMA** is introduced to lower the viscosity.
- **Coupling agent** → **silane** which acts as an adhesive between the inert filler and the organic matrix.

***Recently**, ions have been added to the filler to produce desirable physical changes. Lithium and aluminum ions make the glass easier to crush to generate small particles. Barium, zinc, boron, zirconium, and yttrium ions produce **radiopacity** in the filler particle.

Notes:

1. The normal **wear mechanism** of the composite resins is best explained by the following events: abrasion of the matrix, followed by exposure of filler particles and subsequent dislodgement of these filler particles.
2. With any of the restorative resins, cavity varnish or zinc oxide eugenol **should not be used** as they **might inhibit** polymerization. The use of a cavity varnish might prevent direct contact between the composite and the tooth structure, preventing bonding.

When comparing the physical properties of filled resins to unfilled resins, all of the following are true **except:**

- Filled resins are harder
- Unfilled resins have a higher coefficient of thermal expansion
- Filled resins have a higher compressive strength
- Unfilled resins have a lower modulus of elasticity
- Filled resins have a lower tensile strength

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• **Filled resins have a lower tensile strength**

***This is **false;** filled resins have a **higher** tensile strength.

The most common classification method for composite resins is based on filler content, filler particle size, and the method of filler addition. Almost all **important properties** of composite resins **are improved** by using higher filler levels. However, as the filler level is increased, the fluidity decreases.

Highly filled resins typically contain large filler particles but this composition results in a rough finished surface. **Smaller filler particles** are used to produce a resin that has a **relatively smooth finished surface.**

Resin filler particles are called:
- **Macrofillers** → 10-100 microns in diameter
- **Midifillers** → 1-10 microns in diameter
- **Minifillers** → 0.1-1 micron in diameter
- **Microfillers** → 0.01-0.1 micron in diameter. Examples include Denta-colour, Durafill, Heliomolar RO, and Silux Plus.

***Hybrid resins** contain a mixture of particles with different diameters which allows higher filler levels and still permits good finishing. The principal particle size is in the 0.1 to 1 micron range. Charisma, Herculite XRV, Prodigy, Tetric, TPH (*Total Performance Hybrid)*, and Z-100 are examples of this type.

Note: Hybrid and microfill resins utilize colloidal **silica fillers** which are useful for increasing the hardness and wear resistance of the base resin material while maintaining high polishability and overall esthetic qualities.

***New resins with **nanofillers** that range in size from .005 to 0.01 micron have recently been developed. These particles are so small that very high filler levels can be achieved while still maintaining workable consistencies. Supreme H-NF and Simile H-NF are examples.

Today the most popular way to polymerize **matrix monomers** is:

- Self-cured
- Chemically cured
- Ultraviolet light-cured
- Visible light-cured

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- **Visible light-cured**

Important points to remember when using a visible light-curing unit:

- **Hold the light** as close to the resin as possible → within 2 mm to be effective.
- **Place a shield** between the light tip and the operator's eyes. Patients who have had recent cataract removal should have protection also. **Note:** Studies have shown that the visible light used in polymerization of photo-activated materials can cause retinal damage → always use a shield and eyeglasses for protection.
- **For deep restorations,** you have to cure the composite in increments → if you don't, the deeper areas will not be cured. **Important:** No more than 1.5–2 mm increments should be light cured at one time.
- **Make sure the bulb in the light** is still powerful enough → they have commercially available products to test the bulb.
- **With darker resin shades,** cure a little longer.

Remember:

- **For large restorations** *(those that are wider than the diameter of the light tip)*,cure each area for the full required time. Do not back off light tip until it lights up entire surface of restoration.
- **Visible light-curing** involves light energy in the range of 410–500 nm with a peak intensity of about 470 nm.
- The **minimum acceptable level** for visible curing light outputs is 300 mW/cm^2.

All of the following are current monomers for composite resins **except:**

- BIS-GMA
- PMMA
- UEDMA
- TEGDMA

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• **PMMA**

Dental resin composites typically contain a mixture of soft, organic resin matrix *(polymer)* and hard, inorganic filler particles *(ceramic)*. Other components are included to improve the efficacy of the combination and initiate polymerization. The resin matrix consists of monomers, an initiator system, stabilizers and pigments. The inorganic filler consists of particles such as glass, quartz and colloidal silica. The matrix and filler are bonded together with a coupling agent. The performance of resin composites is dependent upon these basic components. The recent improvement in these materials has primarily focused on filler technology, but the resin monomers have remained largely unmodified.

The **most common monomers** used are BIS-GMA, urethane dimethacrylate *(UEDMA)*, and triethylene glycol dimethacrylate *(TEGDMA)*. BIS-GMA is extremely viscous at room temperature due to hydrogen bonding by hydroxyl groups. Lower viscosity is obtained by mixing BIS-GMA with dimethacrylate monomers *(TEGDMA)* of lower molecular weight to facilitate the addition of fillers.

Important: The high filler content and the BIS-GMA resin matrix drastically **reduce** the coefficient of thermal expansion *(as compared to the unfilled acrylic resins)*. The filler also **reduces** polymerization shrinkage and increases hardness.

The first materials that were used as esthetic materials were based on silicate cements. Due to solubility problems the silicate cements were replaced by **unfilled acrylic resins.** Unfilled acrylic resins contracted excessively during polymerization permitting subsequent marginal leakage and were not strong enough to support occlusal loads. These unfilled acrylic resins have been replaced by **filled resins** *(also called composite resins)*. A filled resin is one in which an **inorganic inert filler** *(usually silica or quartz)* has been added to the resin matrix.

The **main disadvantages** to using methyl methacrylate as a permanent restorative material are its:

- High insolubility in saliva and high modulus of elasticity
- Low resistance to abrasion and high thermal coefficient of expansion
- Poor initial esthetics and high modulus of elasticity
- Low solubility in saliva and low modulus of elasticity

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• **Low resistance to abrasion and high thermal coefficient of expansion**

***Ideal dental restorative materials should have a coefficient of thermal expansion similar to tooth *(very low)* and a high resistance to abrasion. Those with high coefficients often leak at the margins.

For both inlays and onlays, plastic *(acrylic)* provisional restorations are fabricated prior to the final restoration being cemented. Their physical properties enable them to withstand occlusal forces and the adverse oral environment for short periods of time. These **temporaries must:** restore and maintain proximal contacts, restore and maintain the occlusion, restore and maintain tooth contours and the margins should be closed and flush with the tooth.

Methyl methacrylate, ethyl methacrylate, and ethylene imine resins have been employed to produce provisional restorations. However, **methyl methacrylate** *(MMA)* is by far the most common. It is the liquid monomer that is mixed with the polymer **polymethyl methacrylate** *(the powder).* The monomer partially dissolves the polymer to form a plastic dough.
Note: The monomer is polymerized by the action of an **initiator** *(benzoyl peroxide).*
Important: Methyl methacrylate maintains the occlusal and interproximal contact relationships.

Remember: Polymerization **should not go** to completion in the mouth for fear of overheating the pulp and that the provisional will not be able to be removed from the tooth. These provisionals are usually cemented in with a ZOE cement.

Which restorative material has the lowest **thermal conductivity and diffusivity?**

- Amalgam
- Gold
- Unfilled resin
- Filled resin

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• **Unfilled resin**

This characteristic probably offsets to some degree the undesirable effects of the relatively high coefficient of thermal expansion, which is **7 to 8 times that of the tooth.** Due to this low thermal conductivity and diffusivity, the unfilled resin restoration changes temperature quite slowly. Therefore, it takes considerably longer for the unfilled resin restoration to become hot or cold, as compared to metallic restorations, which have a high thermal conductivity and diffusivity.

Notes:

1. The **compressive strength** of the unfilled resin is low; the **yield strength** and **tensile strength** are even lower.
2. Unfilled resins are the **softest** of all restorative materials.
3. Compared with amalgam, filled resin, direct gold and silicates; **unfilled resins** show the **greatest extent** of marginal leakage related to temperature change.

Remember: A **low coefficient** of thermal conductivity is most characteristic of currently available cement bases.

A properly **acid-etched** enamel surface appears:

- Somewhat yellow in color
- Identical to unetched enamel
- Dull white and chalky
- Slightly gray with a shine

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- **Dull white and chalky**

***Any other appearance is an indication that the acid etching has been contaminated and must be reapplied.

One of the most effective ways of **improving** the marginal seal and mechanical bonding of composite resins to tooth structure is to condition or pretreat the enamel with acid prior to insertion of the resin. This procedure is referred to as **"acid-etch"** technique.

The acid **cleanses the surface** of debris left after cavity preparation, thus providing an opportunity for **better wetting** of the enamel by the resin. Even more important though, is that a selective dissolution of the enamel occurs during etching. While enamel is normally porous, the acid removes calcium salts, increasing both the size and number of microspaces present. This **"acid-etch technique"** conserves tooth structure, reduces microleakage, improves esthetics, and provides micro-mechanical retention.

Important: Acid etching paves the way for resin "microtags" which produces a much improved bond of the resin to the tooth. The effective tag length as a result of etching on adult anterior teeth has been demonstrated to be approximately 7 to 25 μm .

Note: Studies indicate that acid-etched composite resin restorations have the **best initial seal** *(microleakage)*, however, over time this weakens *(amalgam has the best seal over time).*

Enamel is **etched** with:

- Maleic acid
- Phosphoric acid
- Polyacrylic acid
- Tartaric acid

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• **Phosphoric acid**

Teeth are normally treated with a **37% solution** of phosphoric acid to roughen the surface of the enamel. This forms little tags approximately 7-25 micrometers in length, **providing mechanical retention.** Enamel rods are most effectively etched at the **enamel rod ends.** When using the acid etch technique all enamel cavosurface margins should be chamfered *(this process forms obtuse angles)*. This chamfer or bevel **affords more** surface area for etching to enhance the seal and retention *(reduces microleakage)*. Also, this bevel improves esthetics and **exposes enamel rod ends** for acid attack.

Remember: Once you etch the tooth, it cannot be contaminated with saliva. If it does, **you must complete the entire etching procedure again.**

Traditionally, etching enamel surfaces with orthophosphoric acid has been a usual clinical procedure to increase the bond strength between the composite resin and etched enamel. Studies have shown that normally, phosphoric acid at a concentration between **30-40%** provided very retentive enamel surfaces. Concentrations greater than 50% resulted in the formation of a monocalcium phosphate monohydrate that inhibits further dissolution, but in concentrations **not lower than 10%** the phosphoric acid solution did not produce adverse effects on the bond strength.

The **outline form** of a Class V composite preparation resembles that of a Class V amalgam preparation **except** for what important feature?

- No retentive grooves are necessary
- The internal line angles are much more rounded
- Pulp protection is not required
- None of the above

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- **The internal line angles are much more rounded**

***When restoring teeth with composite resin, it is much easier to compress the material into rounded line angles.

The **outline form** of a Class V restoration is not always uniform, as it will vary depending on the location and amount of caries or decalcification → the size and location of the carious lesion **determines** the outline form of the cavity preparation. When the carious tissue has been removed and the margins are on reliable enamel or dentin, the **outline** will usually be rectangular with the corners round, ovoid, or kidney-shaped, very much resembling the amalgam Class V preparation **except** that the internal line angles are much more rounded.

Recent research indicates that preparations with **bevels** are more resistant to microleakage as compared with those without bevels when an **acid etch technique** is used. The **bevel** permits the acid to attack the enamel rods at the appropriate angle for maximum effect. The cavosurface margin is **beveled** wherever it is placed on enamel → this is a major difference between composite and amalgam preps. When possible an enamel bevel 0.2 to 0.5 mm wide is advocated as the final stage of preparation. This bevel is etched and provides retention for the restorative material as well as **improving the marginal seal** and maintaining the strength of the resin with sufficient bulk. Retentive grooves supplement the etched enamel retention *(these grooves are placed in both incisal and gingival axial line angles).*

Whenever possible, use a composite syringe to place the composite resin in the restoration, this will minimize the possibility of trapping air in the final restoration.

Which component of a dentin bonding system functions primarily to penetrate through the remnant smear layer and into the intertubular dentin to form an interpenetrating network around dentin collagen?

- Etchant
- Primer
- Adhesive
- None of the above

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• **Primer**

Dentin bonding systems involve an unfilled, liquid acrylic monomer mixture which is placed onto an acid conditioned and primed dentin surface. Typical acid conditioners include 32–40% phosphoric acid, 20% polyacrylic acid, EDTA or maleic acid. The bonding primer is based on hydrophilic monomers such as hydroxyethyl methacrylate *(HEMA)*.

Following conditioning, the **primer** is applied. It is designed to penetrate through the remnant smear layer and into the intertubular dentin to fill the spaces left by dissolved hydroxyapatite crystals. This allows the primer to form an interpenetrating network around dentin collagen.

Finally, the unfilled resin adhesive *(which is also known as a* **bonding agent***)* is applied. The resin is then cured *(light-, self-, or dual-cured)*. This layer can now bond to composite or amalgam. Some examples of bonding systems and there bonding agent *(adhesive)* include: Wet Bond *(BisGMA, HEMA)*, One Step *(BPDMA, acetone)*, and Scotchbond Multi- Purpose *(BisGMA, HEMA, photoinitiator)*.

Adhesive categories:

Etch & rinse → three-step: conditioner, primer, adhesive
two-step: conditioner, *(primer & adhesive)*

Self-etch → two-step: *(conditioner & primer)*, adhesive
one-step: *(conditioner & primer & adhesive)*

Note: Currently there are five categories of bonding agents — The three-step etch & rinse adhesives are considered **fourth-generation** adhesives. The **fifth-generation** consists of the two-step etch & rinse adhesives. The new self-etch products are considered the **sixth generation** and **seventh generation.** The sixth generation may be subdivided into **Type 1** *(i.e., two-step; acidified primer and adhesive applied separately)* and **Type 2** *(i.e., one-step; self-etching adhesives are mixed and applied)*. The **seventh generation** adhesives require no mixing and are simply placed in one step.

With respect to onlay preparations, **"shoeing"** of a functional cusp is:

- Sometimes indicated
- Always indicated
- Never indicated

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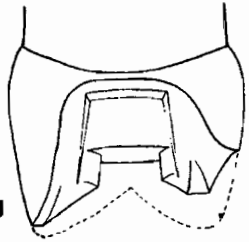
• **Never indicated**

***Important:** "Shoeing" is **never indicated** on functional cusps.

Onlay preparations:

• **Resistance form** → two types of cuspal protection:

1. **"Capping"** → refers to the **complete** coverage of functional cusp with 1.5 mm of gold.
   "Shoeing" → refers to veneering of non-functional cusp by means of a slight finishing bevel.
2. **Except** in situations demanding a minimal display of gold *(primarily the facial cusps of maxillary molars and premolars)*, capping is **always preferred** over shoeing.



Shoeing **Capping**

Onlay preps have:

• Improved resistance to fracture due to cuspal coverage
• Thickness of gold on occlusal *(1.0mm on non-functional cusp and 1.5mm on functional cusp)* resists deformation
• Reliance on tapering lingual and buccal walls for retention
• Reciprocation of mesial and distal axial walls *(near parallel)*
• External extensions over the cusps add retention if nearly parallel to line of draw

Rapid cooling by immersion in water, of a dental casting from the high temperature at which it has been shaped is referred to as:

- Annealing
- Tempering
- Quenching
- None of the above

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• **Quenching**

This usually is undertaken to maintain mechanical properties associated with a crystalline structure or phase distribution that would be lost upon slow cooling.

Two advantages gained in quenching:

1. The noble metal alloy is left in an annealed condition for burnishing, polishing, and similar procedures → it maintains its malleability and ductility.
2. When the water contacts the hot investment, a violent reaction ensues. The investment becomes soft and granular, and the casting is more easily cleaned.

Remember: The set of processes, annealing, hardening and tempering are collectively known as **"heat treating".**

- **Annealing** is the softening of a metal by controlled heating and cooling to make its manipulation easier. It makes the metal **tougher** and **less brittle.**
- **Tempering** is hardening something by heat treatment.

All of the following are **contraindications** for a Class II gold inlay **except:**

- Young patients
- In patients with a low caries index
- Cost
- Esthetics

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- **In patients with a low caries index**

A gold inlay is defined as a cast gold restoration which derives its retention from the **internal walls** of the cavity preparation.

Disadvantages and contraindications for a Class II gold inlay:

- **Expense** → gold is 6 to 7 times more expensive than amalgam
- **Time** → at least two visits is necessary
- **Minimal lesions** → best restored with gold foil
- **Large lesions** → if cavity width exceeds one-third the intercuspal width, the tooth should receive cuspal coverage
- In patients with a **high caries rate**
- **Young** patients
- **Color** → not esthetic

Advantages and indications for a Class II gold inlay:

- **Tooth contours** → where optimum contour and surface finish is desired to maintain periodontal health
- **Strength**
- In patients with a **low caries rate**
- Use against another gold restorations
- Bio-compatible

Surrounding the wax pattern with a material which can accurately duplicate its shape and anatomical features is referred to as:

- Investing
- Burnout
- Casting
- Pickling

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• **Investing**

Gypsum bonded investments are used with Type I,II, and III gold alloys. Gold alloys used for cast gold restorations shrink upon solidification. Therefore, it is necessary to compensate for the solidification shrinkage of the specific alloy used by **expanding the mold** enough to equal the shrinkage.

The **dimensional compensation** necessary is accomplished by **two** methods of expansion:

1. **Setting expansion** → occurs as a result of normal crystal growth but can be enhanced by allowing the investment to set in the presence of water, producing **hygroscopic expansion.**
2. **Thermal expansion** → is achieved through the normal expansion that occurs upon heating the silica *(quartz or cristobalite)*. **Note:** The amount of expansion depends on the particular refractory material used *(cristobalite produces greater expansion than does quartz).* ***Thermal expansion is the **principal cause** for mold expansion.

Variables that influence expansion:

- The **older** the investment is → the **less** it will expand
- If the water / powder ratio is **increased** → the expansion is **reduced**
- The **longer** the spatulation time → the **greater** the expansion
- The **longer** the time between mixing and immersion in a water bath → the **less** it will expand

Note: During **solidification** of an alloy, the number of grains forming depends on the rate of cooling and the presence of nucleating agents.

Which of the following finishing margins is essentially a **hollow ground bevel,** creating more bulk of restorative material near the margin and providing a greater cavosurface angle?

- Knife edge
- beveled shoulder
- Chamfer
- Shoulder

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• **Chamfer**

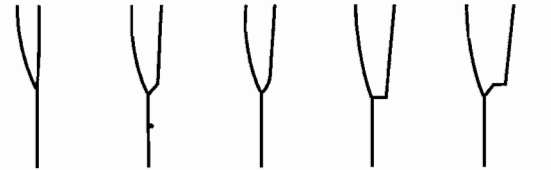
A **chamfer** is essentially a **hollow ground bevel.** Instead of a flat diagonal cut across the cavosurface margin, the chamfer is **"scooped out"** creating more bulk of restorative material near the margin and providing a greater cavosurface angle.

Cavosurface angle configurations that are used when preparing a tooth for a **cast gold restoration:**

A **bevel** is a diagonal cut across the cavosurface margin which is **flat in one dimension only** and curved in its other dimensions. It involves the external ends of enamel prisms and follows a continuous curved outline. It can be either a **short bevel** which cuts only the external one-third of the enamel prisms, a **full bevel** involving the entire thickness of enamel, or a **wide bevel** involving not only enamel but some dentin as well.

A **plane** is a diagonal cut across the cavosurface margin which is **flat in all dimensions.** A plane may involve the entire thickness of enamel *(which it usually does)* or most of it but **cannot be curved in any direction.**

Types of Finish Margins



The one **characteristic** that is common to all Class II gold **inlay** preparations is:

- The uniform depth of the pulpal floor
- The lack of undercuts
- The placement of a base
- All of the above

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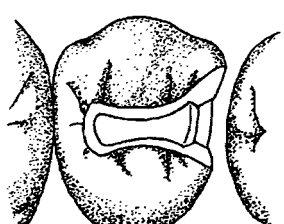
• **The lack of undercuts**

***The restoration **will not seat** if there are undercuts. Actually this holds true for **all** cast metal restorations. **See picture below of Class II inlay preparation.**

When designing a Class II preparation for an inlay, an **occlusal lock or dovetail** should be established to prevent proximal dislodgement. Also, the **marginal ridges** of posterior teeth that are restored with cast gold should be **rounded** to help form the occlusal embrasures and be in **contact with the cusps** of the opposing teeth. Marginal ridges should be the **same height** as the adjacent tooth's marginal ridge *(or else you can create an interference in retrusive movement).*

Note: When **removing** a Class II inlay, the **method of choice** is to cut through the **isthmus** to remove the occlusal and proximal pieces one at a time.



In a cast gold restoration, the majority of retention results from:

- The cement
- The properties of cast gold
- Friction between the cavity wall and the casting
- The opposing occlusion

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- **Friction between the cavity wall and the casting**

Remember: The cement's main function in a cast gold restoration is to seal the cavity, **not for retention. Retention** is designed within the preparation and results from friction between the cavity wall and the casting.

Important: For maximum retention of cast gold restorations, the axial walls should be as **parallel as possible and as long as possible.** Retention is **directly proportional** to the area of the axial walls and their parallelism. The axial walls should converge slightly from the gingival walls to the pulpal wall.

Retention form depends on:
- Length of walls *(minimum 3 mm)*
- Taper of walls *(ideal taper is 6 degrees between opposing walls)*
- Circumference

Advantages of cast gold restorations:
- They are very strong and able to withstand the forces of mastication
- They are ideal for occlusal rehabilitation
- They are kind to the gingival tissue

Disadvantages of cast gold restorations:
- Esthetics
- Cost
- Time-consuming
- Difficulty of technique
- The need to use cement, which is the weakest point in the cast gold restoration
- Gold has a high thermal conductivity

All of the following statements concerning the use of **base metal** casting alloys compared to using **noble metal** casting alloys for cast restorations are true **except:**

- Base metal alloys are harder to cast and finish
- Base metal alloys are less dense
- Base metal alloys are stronger
- Base metal alloys are more resistant to corrosion

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- **Base metal alloys are more resistant to corrosion**

***This is **false;** base metals *(also called non-precious metals)* are **less resistant** to corrosion. Base metal alloy **advantages** are principally found only in their **strength and low density.**

An **alloy** is a mixture of two or more materials that are mutually soluble in the liquid state. A **pure** metal solidifies at a constant temperature, whereas alloys solidify through a range of temperature.

Base metal alloys are based on active metallic elements that corrode, but which develop corrosion resistance via surface oxidation that produces a thin, tightly adherent film, which inhibits further corrosion. **Example:** Cobalt-chromium alloys form a Cr_2O_3 oxide film, which passivates the surface.

Types of alloy systems:

(classified on the basis of the type of structure that forms as they solidify)

- **Solid solution alloys** → the metals freeze without segregation of the individual constituents. **Note:** Are generally used in dentistry because they have a very homogenous structure an provide maximum strength.
- **Eutectic alloys** → separate into individual grains of the respective constituents. Exhibit complete liquid solubility **but** limited solid solubility. Example is the **silver-copper system.**

Remember: Noble metals *(also called precious metals)* are **very resistant to corrosion** and **do not oxidize** on casting. Noble systems for dental use are based on the noble or precious metal elements **gold, silver, palladium, and platinum.**

Copper's major effect on a gold casting alloy is:

- Corrosion resistance
- To increase the hardness
- To offset the color contributions of copper
- To elevate the melting range

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- **To increase hardness**

Effects of the Various Constituents	
Compound	**Major Effect on Gold Casting Alloy**
Gold *(Au)*	Increases resistance to tarnish and corrosion; increases ductility and malleability
Copper *(Cu)*	Principal hardener; is necessary for heat treatment *(when added in a concentration > 12 wt%)*
Silver *(Ag)*	Main purpose is to modify the red color produced by gold and copper; reduces melting temperature; increases ductility and malleability
Platinum *(Pt)*	Raises melting temperature; increases tensile strength; decreases the coefficient of thermal expansion; reduces tarnish and corrosion
Palladium *(Pd)*	Raises melting temperature; increases hardness; acts to absorb hydrogen gas which can cause porosity in the casting; prevents tarnish and corrosion; has very strong whitening effect on gold alloys even when used at a low concentration *(i.e., 5 wt%)*
Zinc *(Zn)*	Acts as a scavenger and prevents oxidation of the other metals during the manufacturing process; increases fluidity and decreases surface tension, which improves castability
Iridium *(Ir)*	Acts as a grain refiner; this increases tensile strength and percentage *elongation (i.e., ductility);* it also increases, to a much lesser degree *(if at all),* hardness and yield strength

High-gold alloys used for cast restorations are:

- Greater than 20% gold or other noble metals
- Greater than 30% gold or other noble metals
- Greater than 50% gold or other noble metals
- Greater than 75% gold or other noble metals

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• **Greater than 75% gold or other noble metals**

Four Types of High-Gold Alloys:

1. **ADA type I** → highest gold content, 83% noble metals. Intended for small inlays. Easily burnished due to high ductility.
2. **ADA type II** → greater than 78% noble metals. Intended for larger inlays and onlays. Can also be burnished.
3. **ADA type III** → greater than 75% noble metals. Intended for onlays and crowns. Capable of being heat-treated.
4. **ADA type IV** → greater than 75% noble metals. Intended for bridges and removable partial dentures. Also capable of being heat-treated. **Hardest** of high-gold alloys.

Type	Hardness	Yield Strength *(MPa)*	Percentage Elongation *(%)*
I	Soft	<140	18
II	Medium	140-200	18
III	Hard	201-340	12
IV	Extra-Hard	>340	10

– **Medium-gold alloys** are 25-75% gold or other noble metals.
– **Low-gold alloys** are less than 25% gold or other noble metals.
– **Gold substitute alloys** are alloys not containing gold. These alloys are called **passive** because the form some type of protective layer *(surface oxide film)* that offers maximum resistance to corrosion.

Examples include: Palladium-silver alloys and Cobalt-chromium alloys.

Remember: The **karat** of a gold alloy is the number of parts that are pure gold, on the basis of 24 parts as a unit *(thus, 24 karat is 100 % gold, while 18 karat is 75% gold)*. **Fineness** is measured on the basis of parts of pure gold per 1,000 *(1,000 fineness is 100% gold, while 500 fineness is 50% gold)*. **Pure gold is only used in the foil restoration.**

A **cavosurface bevel** is used when preparing a tooth for a **cast gold inlay or onlay.** What is the **principal reason** for its use?

- To allow room for the cement
- To improve the marginal adaptation
- To compensate for shrinkage of the casting gold alloy
- To provide resistance form to the preparation

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• **To improve the marginal adaptation**

The bevel on the cavosurface margin permits **closer adaptation** of the gold margin because the thinner margin of gold overlying the bevel is more ductile and is able to be burnished. **Note:** During cementation, the finishing *(burnishing)* of the margins of a cast gold restoration should be started as soon as the restoration is well-seated into the preparation.

While preparing a tooth to receive an inlay or onlay, a **gingival bevel** is used to **remove unsupported enamel** and to **compensate for casting inaccuracies.** Gingival margin trimmers, carbide finishing burs, or fine, tapered diamonds are used to place this bevel.

Note: This margin is always **placed gingival** to the contact area.

Remember:

- When preparing teeth with short clinical crowns, the facial and lingual walls should have a **minimal** gingival to occlusal divergence angle for **maximum retention.**
- From facial to lingual, the **axiopulpal line angle** of an onlay preparation is **longer** than the **axiogingival line angle** *(if it were not, the preparation would be undercut and the onlay would not seat).* For an MOD onlay prep, the axial walls must converge from the gingival walls to the pulpal wall *(for the same reason, the onlay would not seat if they diverged).*

Frequently the surface of a gold casting is dark due to the formation of a surface oxide film. This **surface film** is removed by:

- Quenching
- Age Hardening
- Pickling
- Fusion

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• **Pickling**

After the casting is removed from the ring, a stiff toothbrush is used under tap water to remove adhering investment. Pickling consists of careful heating of the casting which is placed in an acidic solution to reduce the surface oxides. The most effective pickling solution is 50% hydrochloric acid solution. **Note:** The fumes are very corrosive and will damage metal objects in the office. It is recommended to use commercially available pickling agents, which are solutions of acid salts. They act more slowly **but** will produce acceptable results.

After pickling, again brush the casting and dry thoroughly. It should be emphasized that the casting must be dry to permit thorough examination for residual investment and preence of nodules or roughness.

Two methods of pickling:

1. Place casting in pickling solution and gently heat the solution. This will produce a clean, gold-colored casting.
2. Heat the casting until it barely emits a dull glow and then drop it in the pickling solution.

Note: With this method, there is a risk of distorting delicate margins. *(Whichever method is used, **always wear** safety glasses.)*

The process of annealing gold foil is more commonly referred to as:

- Pickling
- Tempering
- Compacting
- Degassing

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• **Degassing**

Important: Gold foil is annealed *(also called degassing)* to remove volatile surface impurities prior to its placement in the cavity preparation.

Annealing, in general, comprises three stages: recovery, recrystallization, and grain growth. The higher the melting point of the metal, the **higher the temperature** needed for annealing. **Note:** During this process the stresses in the metal are relieved. This process of heating and cooling a metal is done to make it tougher and less brittle.

Remember:

- **Cold work** *(also called strain hardening or work hardening)* is the deformation of a metal at **room temperature,** in contrast to the effect of working at a higher temperature, such as in forging. An **example** of cold work would be the bending of a wire back and forth rapidly between the fingers.

- **Burnishing** is somewhat related to polishing in that the surface is drawn or moved. **However,** instead of using many tiny particles, only one large point is employed. If a round steel point is rubbed over the margin of a gold inlay *(made from a Type I or II gold alloy)*, the metal may be moved so that any small discrepancy between the inlay and the tooth can be closed.

OPERATIVE

Gold

Essential properties of a **Class V** cavity prepared for **direct filling gold** include all of the following **except:**

- Rounded internal line and point angles
- Small retentive undercuts placed in the axio-occlusal and axio-gingival line angles
- Mesial and distal walls that flair and meet the cavosurface at a 90° angle
- An axial wall that is convex and follows the external contour of the tooth .5 mm into dentin

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- **Rounded internal line and point angles**

Class V cavity preparation for direct filling gold:

- The **outline form** is usually either **trapezoidal** *(most popular)* or **kidney-shaped.** The axial wall is placed .5 mm into dentin *(this will make the occlusal wall slightly deeper than the gingival wall because there is a thicker layer of enamel making up the occlusal wall).* The mesial and distal walls are placed at the line angles of the tooth.

Remember: For any Class V prep *(whether for amalgam, composite or direct filling gold)*, the outline form is determined by the **extension of the carious lesion.**

- The **retention form** is attained by sharp internal line and point angles *(axio-gingival and axio-occlusal).*
- The **resistance form** is provided by flat mesial and distal walls and a convex axial wall which parallels the external surface of the tooth.

Note: The axial wall is **convex** in a mesiodistal direction in order to **conserve** tooth structure and **minimize** pulpal irritation.

Important: The rubber dam **is essential** to prevent contamination of the gold with saliva. A cervical clamp usually is necessary to retract the gingiva *(#212 ivory clamp).* The hole that is to be punched in the rubber dam for the **tooth that is being restored** should be located **facial** to the normal alignment with the adjacent teeth.

OPERATIVE

Gold

All of the following statements concerning **direct gold** are true **except:**

- It is the **most nearly permanent** of all restorative materials
- It provides **good adaptation** to the cavity walls
- Its **coefficient of thermal expansion** is close to that of tooth structure
- It has a **high tensile strength** *(edge strength)*
- It **will** corrode

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- **It will corrode**

***This is **false;** direct filling gold **will not corrode.**

Direct gold is considered by many authorities of operative dentistry to be the finest and most desirable restorative material. However, the additional skill required, the demand for close attention to detail and the limitation in versatility are drawbacks of the technique which have lessened its popularity, resulting in a utilization rate much below what the material merits. **Disadvantages** of direct gold include: poor esthetics, a demanding technique, high cost and a high coefficient of thermal conductivity *(12 times that of amalgam).*

Indications for direct gold:
- **Ideal lesion** → no more than 1.0 to 2.0 mm into dentin and of minimal outline form
- **Ideal pulp** → at least 2 mm of dentin between restoration and pulp. Also, pulp should be vital with no history of trauma or tooth sensitivity
- **Ideal periodontium** → no mobility of tooth, no inflammation or degenerative processes are present

Note: The amount of force needed to compact direct gold is influenced most by the **surface area of the condenser.** A decrease in diameter of a condenser point will produce a proportional increase in energy concentration. For this reason, good concentration with less force and trauma can be accomplished with a small point. **During condensation,** the surface hardness, tensile strength and yield strength of direct gold increase.

Gold

Different microstructure has been described for gold foil, mat gold and powered gold. It has been demonstrated at the microscopic level that the:

- Void spaces remain in any compacted gold
- Gold foil is more porous than any other form of gold
- Crystalline golds can be packed densely more readily than gold foil
- Proper compaction can remove all voids from commercially available direct filling golds

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• **Void spaces remain in any compacted gold**

Mat gold *(crystalline gold)* is formed by electrolytic precipitation yielding a crystalline structure resembling trees or links of chain. **It is used for bulk filling of cavities.** The flow and adaptation of mat gold is **not as good** as gold foil or powdered gold.

Gold foil is the **traditional and oldest type of gold.** It is formed by rolling and beating gold into thin sheets. This process causes the elongation of grains, giving a fibrous appearance. Gold of this type is available in sheets, cylinders and pellets. Gold foil is used for bulk filling as well as a finishing veneer for mat gold.

Powdered gold *(granular gold)* is used to describe gold that is formed by atomizing. Most of the granules in this material have an overall spherical shape. This type of gold has **three advantages:**

→ It can be placed in a very short period of time
→ It is more dense than foil and therefore is easier to manipulate and condense, and saves time
→ A gold foil veneer is not required

Notes:

1. There are always microscopic voids in any compacted gold, no matter which type is used. The cause of porosities or pits in a gold foil restoration is related to **improper condensing technique** and the use of oversized pellets.
2. Cohesion of direct gold at room temperature is an example of the principle of **atomic attraction.**

The number one indication for the use of **direct filling gold** is:

- The large Class II lesion
- The small initial Class III lesion
- The small Class II lesion
- The large Class III lesion

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- **The small initial Class III lesion**

Reconciling permanency and esthetics is the key limitation and challenge to this classification. Lesions on the distal surfaces of all anteriors are relatively invisible and present less of an esthetic concern than mesial lesions. In most cases, the distal restoration in a canine is not visible.

Class III cavity preparation for direct filling gold:

- The **outline form** is best described as a horizontal slot. The incipient lesion and subsequent preparation are usually positioned gingival to the contact area. The preparation has a flat labial wall and straight, parallel incisal and gingival walls. The axial wall is flat and there is no lingual wall.
- The **retention form** is attained by sharp internal anatomy with well-defined line angles and point angles. Retentive grooves are placed along the inciso-axial and gingivo-axial line angles.
- The **resistance form** is provided by flat walls and a flat, well-supported labial wall.

Note: The most important factor in securing **adaptation of gold foil** to all parts of the preparation is the **direction** in which the force is applied *(along with a small condenser point).*

Generally, for the average size wax pattern, sprue formers smaller in diameter than approximately:

- 1.0 mm are contraindicated
- 1.5 mm are contraindicated
- 1.75 mm are contraindicated
- 2.0 mm are contraindicated

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• **1.5 mm are contraindicated**

The purpose of the sprue former or sprue pin, as it is usually called, is to provide an ingate or sprue in the investment through which the **molten alloy can reach the mold** after the wax has been eliminated.

The size of the sprue former depends to a considerable extent on the type and size of the pattern, the type of the casting machine to be used and the dimensions of the flask or ring in which the casting is to be made. **Generally,** however, for the average size pattern, sprue formers smaller in diameter than approximately 1.5 mm are contraindicated. If a sprue is too small, the molten metal freezes completely in this area first, and localized shrinkage porosity results. The general rule for sprue pin diameter when using a centrifugal type of casting machine is that the diameter of the sprue pin should be **equal to or greater than** the thickest portion of the pattern.

As a general rule, it is desirable to attach the sprue at the **point of greatest bulk** in the pattern. There is less chance of distortion upon attaching the sprue, and the molten metal is more apt to remain liquid in this area until the entire mold is filled. The **direction** of the sprue former is also important. It should never be attached at a right angle to a broad flat surface of the mold. The entering hot metal impinges the mold surface at this point to cause **turbulence** of the metal, which, in turn, creates a shrinkage void or suck-back porosity. When the same pattern is sprued at an **angle of 45°** to the proximal wall, a satisfactory casting is obtained.

Dental wax patterns *(i.e., inlays, onlays, crowns)* should be invested as soon as possible after fabrication to minimize change in the shape caused by:

- Reduced flow
- Drying-out of the wax
- Relaxation of internal stress
- Continued expansion of the wax
- All of the above

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• **Relaxation of internal stress**

The three types of inlay waxes **differ** in terms of melting point and flow:

1. **Type A** → hard or low-flow wax that is rarely used except in some indirect technique
2. **Type B** → medium-flow wax that is used in some direct techniques.
3. **Type C** → soft or high-flow wax that is used in indirect techniques for the construction of inlays, onlays, and full crowns.

The essential ingredients of a successful inlay wax are paraffin wax, gum dammar and carnauba wax with some coloring material. **Paraffin wax** is generally the main ingredient, usually in a concentration of 40-60%. **Gum dammar** is added to the paraffin to improve the smoothness in the molding. It also increases the toughness of the wax. **Carnauba wax** is quite hard and tends to decrease the flow of a wax.

Note: However the pattern is prepared, it should be an accurate reproduction of the missing tooth structure. The casting can be **no more accurate** than the wax pattern. The wax pattern should be invested as soon as possible after fabrication in order to minimize changes in shaped caused by relaxation of the internal stresses in the wax.

Which component of gypsum bonded investments provides the thermal expansion for the investment?

- The binder
- The refractory filler
- Modifiers
- None of the above

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• **The refractory filler**

Main components of gypsum bonded investments:

1. A **refractory** filler which is a form of silicon dioxide *(SiO_2)*, such as quartz or cristobalite comprises 60 to 65% of the investment. These two compounds have different crystal structures and therefore have different thermal expansion coefficients. **Key point:** This refractory filler regulates and provides the **thermal expansion** for the investment.

2. The **binder** *(which is a gypsum matrix of α – calcium hemihydrate)* comprises 30 to 35% of the investment. This is the material that hardens after being mixed with the liquid and thus holds the investment together *(adds strength).* The actual or effective setting expansion is dependent upon the gypsum content plus the water / powder ratio. **Key point:** Using a **thinner mix** *(which contains more water)* of a gypsum-bonded investment will decrease the setting expansion, increase the setting time, increase the porosity of the set material, and **ultimately weaken the set material.**

3. **Modifiers** are added to modify various physical properties of the investment. These modifiers include magnesium oxide, sodium chloride, boric acid, graphite, or potassium sulfate.

Dental investments serve three important functions:

1. A **detailed reproduction** of anatomical form.
2. **Enough strength to withstand** the heat of burnout and the actual casting of the molten metal
3. **Compensation expansion** equal to the alloy solidification shrinkage.

Which of the following are **indications** for a cast gold onlay?

- **Restoration of large lesions:**
  - involving more than **one-third intercuspal dimension**
  - extensive loss of supporting structure where **at least half of the clinical crown remains**
  - loss of cusp(s) with **at least 1 mm of dentin supporting remaining cusps**
- **Restoration of ideal occlusion** in cases of drifting, hypo and hyper eruption, etc.
- **Restoration of optimum contour and proximal contact**
- **Restoration of brittle teeth** *(endodontically treated)*
- **Restoration of a tooth as an abutment for removable prosthesis,** creating ideal guiding planes, rest seats and undercuts
- **Restoration of teeth to meet patient preference for gold**
- All of the above

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• **All of the above**

The main advantage of the onlay is that it can **permanently restore and reinforce a tooth by a conservative technique.** Although conservation of tooth structure is desirable, such action is offset by a lack of retention. It has been shown that conservative onlays have inferior retention compared to full crowns. This is due to the **crown's greater axial surface area.**

Retentive features → parallelism of vertical surface *(axial walls)* is the primary retentive feature of an onlay preparation. **Sharp point and line angles** add to retention.

Auxillary retentive features include a box or a groove. These features may be indicated where inadequate surface area of vertical walls is present. **A box offers a greater increase in surface area, thus greater retention than a groove,** but is also more costly in terms of lost tooth structure.

The location of the gingival margin in the preparation of proximal surfaces is influenced by the **amount of retention required, the need to extend gingivally to clear the contact area and convenience form.**

Which of the following should be **checked first** when a casting that fits on its die cannot be seated on the tooth in the mouth?

- The occlusal contacts
- The taper of the preparation
- The proximal contacts
- The impression used to pour the cast

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• **The proximal contacts**

If a casting fails to completely seat on a prepared tooth, one should first check for residual temporary cement or other debris on the prepared tooth. Once all the temporary cement and other debris are removed, the **first step** in fitting the casting in the mouth is to **adjust the proximal contact areas.**

Very important points:

1. **When seating a casting,** the initial interferences are usually the proximal contacts. Complete seating of the restoration can be verified by an x-ray and a sharp explorer used at the gold tooth margin.

2. **When seating cast gold restorations,** the occlusion of the restoration should be to the same degree that teeth contact in that quadrant and on the opposite side *(use shim stock to check occlusion).*

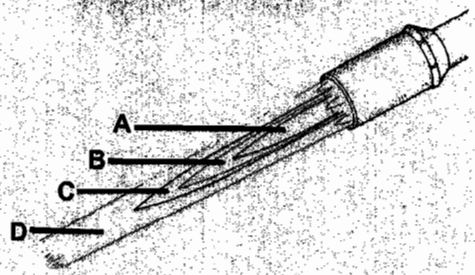
3. **Initially,** if a cast restoration is in **hyperocclusion,** the patient will complain of **cold sensitivity and pressure in the tooth.** If the restoration is **not adjusted,** the tooth will become **very cold sensitive, show signs of mobility and there might be recession of the facial gingival tissue.**

OPERATIVE

Gold

What letter represents the hottest area in the flame below?

- A
- B
- C
- D



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• C

A → The first cone is called the **mixing zone.** It is cool and colorless. The air and gas are mixed before combustion in this zone.

B → The next cone, which is greenish-blue and surrounds the inner cone, is the **combustion zone** in which partial combustion takes place. This is an **oxidizing zone.**

C → The next cone, which has a dim blue tip, is the **reducing zone.** This is the hottest area in the flame and is the **only part of the flame** that should be used to heat the alloy.

D → The outer area is the **oxidizing zone.** This is where final combustion between the gas and surrounding air takes place.

With a little practice, the proper zone in contact with the metal can be readily detected by the condition of the metal surface. When the **reducing zone** is in contact, the surface of the alloy is **bright and mirror-like.** When the **oxidizing portion** of the flame is in contact with the metal, there is a dull film of **"dross"** developed over the surface *("dross" is defined as scum on molten metal).*

Which of the following **methods of investing** is more dependable in the prevention of surface nodules on a casting?

- Hand investing
- Vacuum investing
- Both produce similar results

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• **Vacuum investing**

These defects, or nodules, are caused by the **collection of air bubbles** during the investing. The **best way** to help eliminate these defects is to subject the water-investment mixture to a **vacuum** during the investing procedure in order to remove the air bubbles.

The porosity of the investment is definitely **reduced** by vacuum investing, presumably because of the increased density obtained. As a result, the texture of the surface of the casting is somewhat smoother, with better reproduction of fine detail. The **compressive strength** of the investment is increased slightly by the vacuum investment *(the investment will not fracture as easy).*

Not all of the air is removed by the vacuum treatment. The amount removed depends somewhat upon the consistency of the mix. The **more viscous** the mix, the **more air bubbles** remain in the investment. **However,** a thick mix is usually necessary because of the desired shrinkage compensation and because of the poor surface texture that is obtained with a thin mix.

The minimal reduction of **working cusps** for protection from the forces of mastication should be?

- 2.5 to 3 mm for **both** amalgam and cast gold restorations
- 1.5 to 2.0 mm for **both** amalgam and cast gold restorations
- 2 mm for **amalgam** and 1 mm for **cast gold** restorations
- 2.5 to 3 mm for **amalgam** and 1.5 mm for **cast gold** restorations

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• **2.5 to 3 mm for amalgam and 1.5 mm for cast gold restorations**

Remember:

Working cusps *(functional cusps)*
- Max. teeth → lingual
- Mand. teeth → buccal

Non-working cusps *(non-supporting)*
- Max. teeth → buccal
- Mand. teeth → lingual

Minimal Occlusal Reduction			
Cusp	**Amalgam**	**Cast Gold**	**Metal-Ceramic**
Working	2.5 – 3.0 mm	1.5 mm	1.5 – 2.0 mm
Non-working	2.0 mm	1.0 mm	1.5 – 2.0 mm

Notes:

1. For non-working cusps the **minimal reduction for amalgam is 2.0 mm** while forming a flattened surface *(this provides resistance form)* and for cast gold it is 1.0 mm.
2. **For metal-ceramic restorations:** facial and lingual reduction → 1.5 mm.
3. The difference between tooth preparation for a metal-ceramic restoration and a porcelain jacket crown is mostly related to the configuration of the finishing line or margin → chamfer or bevel for metal-ceramic restorations and a butt joint for porcelain jacket crowns.

Important: The **most effective means** for verifying adequate occlusal clearance is a **wax bite chew-in.**

Which **grasp** used with hand instruments allows for the **greatest** intricacy or delicacy of touch?

- The modified pen grasp
- The inverted pen grasp
- The palm and thumb grasp
- The modified palm and thumb grasp

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• **The modified pen grasp**

***The modified pen grasp is also the **most common.**

With this grasp the middle finger, index finger, and thumb all rest on the handle close to the junction of the handle and the shank. The middle finger rests on the shank and the thumb and index finger are opposite each other on the handle.

The **inverted pen grasp** is very seldom used, however, sometimes it is used for cavity preps utilizing the lingual approach on anterior teeth.

The **palm and thumb grasp** is the most powerful grasp and is most effectively used on the maxillary arch. It is similar to the grasp used for holding a knife while paring the skin from an apple.

The **modified palm and thumb grasp** allows much of the power of the palm and thumb grasp but also permits more delicate control. It works best when you can rest the thumb on the tooth being restored or on the adjacent tooth. It also works best on the maxillary arch.

The **most important** design characteristic of a bur blade is:

- The clearance face
- The rake face
- The edge angle
- The clearance angle
- The rake angle

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• **The rake angle**

***Remember:** The rake angle = the angle made between the line connecting the edge of the blade to the axis of the bur and the rake face. This angle may be positive or negative.

Generally speaking, **soft** materials such as acrylics are cut most effectively with **positive** rake angle burs; whereas extremely **hard and brittle materials** *(such as amalgam)* are best cut with **negative** rake angle burs. A rake angle is said to be negative when the rake face is ahead of the radius *(from the cutting edge to the axis of the bur)*. A negative rake angle minimizes fractures of the cutting edge that helps to increase the bur life. Increasing the edge angle reinforces the cutting edge of the bur and reduces the likelihood of the blade to fracture.

Carbide burs used for cutting tooth structure generally have slight **negative** rake angles and **edge angles** of approximately 90°. **To be most effective,** these burs should be rotating rapidly **before** contacting the tooth.

The **clearance angle** is also another important factor in blade design. The clearance angle serves to eliminate friction between the clearance face and the new tooth structure exposed by the cutting edge. **The greater the clearance angle, the less friction.**

All of the following **influence tooth temperature** during a cutting procedure:

- Diameter and sharpness of the bur
- Bur / tooth contact time
- Type of coolant used *(**water is best**, air may dehydrate the tooth or cause the tooth to be hypersensitive by drawing odontoblasts into the dentinal tubules)*
- Amount of force applied to the bur

The **clearance angle** of a bur blade is:

- The angle made between the line connecting the edge of the blade to the axis of the bur and the rake face
- The angle formed between the rake face and the clearance face
- The angle formed between the clearance face and a tangent to the path of rotation

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- **The angle formed between the clearance face and a tangent to the path of rotation**

Each bur blade has two sides:

1. The **rake face** *(toward the direction of cutting)*
2. The **clearance face**

- The **rake face** is that surface *(side)* of the blade, which makes contact with the tooth surface and faces in the direction of bur rotation.
- The **clearance face** is that surface *(side)* of the blade that faces away from the direction of bur rotation.

Each bur blade has three important angles:

1. The **rake angle**
2. The **edge angle**
3. The **clearance angle**

- The **rake angle** is the angle made between the line connecting the edge of the blade to the axis of the bur and the rake face. This angle may be positive or negative.
- The **edge angle** is the angle formed between the rake face and clearance face.
- The **clearance angle** is the angle formed between the clearance face and a tangent to the path of rotation.

Note: For **most effective cutting,** a bur should be rotating rapidly **before** contacting the tooth.

What is the blade width of a cutting instrument with the following formula: **10 - 85 - 8 -14**

- 10 mm
- 1.0 mm
- 0.85 mm
- 0.80 mm

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- **1.0 mm**

Cutting instruments have **formulas** describing the dimensions and angles of the working end:

- The **first number** indicates the **width of the blade** in tenths of a millimeter → 1.0 mm *(10 x .1)* for the example on the front of the card.
- The **second number** indicates the **primary cutting edge angle** in centigrades → 85 for the example on the front of the card.
- The **third number** indicates the **blade length** in millimeters → 8 mm for the example on the front of the card.
- The **fourth number** indicates the **blade angle** in centigrades → 14 for the example on the front of the card.

***A nib is not a major part of a hand cutting instrument. It is the **working end of a non-cutting instrument** *(i.e., a burnisher, condenser, etc.)*. **Note:** It corresponds to the blade of a hand cutting instrument.

Three major parts of a hand-cutting instrument::

1. The **handle** is that part of the instrument held or grasped during activation of the blade. **Types:** single ended or double ended.
2. The **shank** serves to connect the blade to the handle. **Types:** straight or angled *(mon-angle, bi-angle, or triple-angle)*, meaning one, two or three angles in the shank. **Note:** Proper balance of the instrument is accomplished by angling the shank of the instrument so that the cutting edge of the blade is within 2 mm of the long axis of the handle. In order to keep the blade within 2 mm of the long axis, the shank of the instrument is angled.
3. The **blade** is the working end of the instrument and is connected to the handle by the shank. Blades are of many designs and sizes, depending on the function they are to perform.

OPERATIVE

Chisels are used primarily to cut:

- Cementum
- Dentin
- Enamel
- Amalgam

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• **Enamel**

Chisels may be grouped as:

1. **Straight, slightly curved, or bin-angle** → primarily used for planing or cleaving enamel. Characterized by a blade that terminates in a cutting edge formed by a one sided bevel.

2. **Enamel hatchets** → are chisel-bladed instruments with the cutting edge in the plane of the handle. They come paired left and right.

3. **Gingival margin trimmers** → are similar in design to the enamel hatchet except it has a curved blade and an angled cutting edge. They are primarily used for beveling gingival margins. Among other uses for these instruments is the rounding or beveling of the **axiopulpal line angle** of Class II preparations ***(very important).***

Match the type of **excavator** on the left with the **main purpose** on the right.

• Hoes	Sharpening line angles and convenience points for gold foil preps
• Angle formers	Preparing retentive areas on anterior teeth
• Ordinary hatchet	Remove carious dentin and sometimes carve amalgam
• Spoons	Class III and V direct gold preps

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- **There are four subdivisions of excavators:**

1. The **hoe** excavator → has the cutting edge of the blade perpendicular to the axis of the handle. It is commonly used in **Class III and V preps for direct gold.**

2. The **angle former** → has the cutting edge at an angle *(other than 90°)* to the blade. It is used for **sharpening line angles and is especially useful to form convenience points for gold foil preps.**

3. An **ordinary hatchet** excavator → has the cutting edge of the blade directed in the same plane of the handle and is bibeveled. **Used primarily on anterior teeth for preparing retentive areas.**

4. A **spoon** excavator → has a curved blade with a rounded cutting edge. **It is used to remove carious dentin and sometimes to carve amalgam. Note:** These can be sharpened with handpiece stones.

Which of the following instruments is designed to **most effectively** plane the enamel of the facial and lingual walls of a Class II amalgam preparation?

- A gingival margin trimmer
- A straight chisel
- An enamel hatchet
- A spoon excavator

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• **An enamel hatchet**

***The **enamel hatchet** is the only instrument that will allow the dentist to have proper access to the margins and that will impart the proper cavosurface angle to the margins.

The **number of bevels** that make up the cutting edge can classify hand cutting instruments. **For example,** enamel hatchets and chisels have single bevels, whereas ordinary hatchets *(for example excavators)* have two bevels and are called bibeveled.

Dental hand cutting instruments are angled to:

- Provide better manipulative control
- Produce a better distribution of force
- Increase efficiency
- Establish proper balance when in use

Instruments used to trim restorative materials rather than for cutting tooth structure:

- **Knives** *(finishing, amalgam, or gold)* → used for trimming excess filling material on the facial and lingual.
- **Files** → also used to trim excess filling material, especially at the gingival margins.
- **Discoid-cleoid** → used principally for carving occlusal anatomy in unset amalgam restorations.

All of the following are parts of dental burs **except:**

- Shank
- Shoulder
- Head
- Neck

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• **Shoulder**

Parts of burs:

1. **Shank** → the part that fits into handpiece. The three most common types are straight, latch-type angle and friction-grip angle.
2. **Neck** → the intermediate portion of a bur that connects the head to the shank. Its main function is to transmit rotational and translational forces to the head.
3. **Head** → the working part of the bur, the cutting edges of which perform the desired shaping of tooth structure.

Types of burs → **steel** *(used mostly for finishing procedures)* → **carbide** *(used for cavity preparation; perform best at high speeds).*
Shapes of burs → refers to the contour of the head. The basic head shapes are round, inverted cone, pear, straight fissure and tapered fissure. **Note:** Within a given series of burs, the **smaller numbers** represent small burs; the **larger numbers,** large burs.

Important: The **greater** the number of cutting blades on a bur results in **less efficient** cutting but a **smoother surface** *(polishing burs are of this type).* A **lesser** number of blades on a bur results in **more efficient** cutting but a **rougher surface.** Crosscut fissure burs at high speed or low speed are of this type.

Recent modifications in bur design:

1. Reduced use of crosscut burs → non-crosscut burs are now popular
2. Carbide fissure burs with extended heads
3. Rounding of the sharp tip corners

There are several types of bleaching products available for use at home, which can either be dispensed by a dentist or purchased over-the-counter. Currently, only dentist-dispensed home-use _____ tray-applied gels carry the ADA Seal.

- 30% carbamide peroxide
- 15% hydrogen peroxide
- 20% hydrogen peroxide
- 10% carbamide peroxide

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- **10% carbamide peroxide**

Two methods of bleaching:

1. **"In office"**→ most use a light-activated solution of 35% peroxide in 4-10 minute cycles. This procedure is called "chairside bleaching" and may require more than one office visit. Each visit may take from 30 minutes to one hour.
2. **"At home"**→ the active ingredient contained in all of the at home tooth whiteners which have earned the ADA's seal, and the compound which has been evaluated in the vast majority of at home bleaching studies, is **carbamide peroxide** at a concentration of 10%. The active ingredient found in most over-the-counter at home bleaching products is not carbamide peroxide but instead hydrogen peroxide.

Note: Bleaching can affect the color of dentin and enamel. **Extrinsic stains** respond best to vital beaching. Response is best with yellow stain followed by brown and orange. The worst response is from gray stains *(tetracycline staining)*.

Other ways to lighten vital teeth:

- **Direct composites** → useful for tetracycline staining.
- **Laboratory-fabricated porcelain veneers** → useful when the shape, size and arrangement of teeth are esthetically unacceptable.
- **Full-coverage crowns** *(most invasive and costly)* → may be all-ceramic or porcelain fused to gold.

OPERATIVE

Misc.

All of the following are zones in four-handed dentistry **except:**

- Operator zone
- Assistant zone
- Transfer zone
- Grasping zone
- Static zone

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• **Grasping zone**

The operator and the assistant should concentrate on positioning themselves in work circles. The dentist's work circle should allow easy and unobstructed access to the patient's mouth. The assistant's work circle should include all instruments and supplies needed for the intended operation, also allowing access to the transfer zone to bring the necessary items to the dentist. When viewed from above with the patient's head in the 12:00 o'clock position, the right handed dentist will operate in an area from 8:00 to 11:00 o'clock.This area is the **operator's zone.** Nothing should be in this area that would interfere with the free movement of the dentist. The area from 11:00 to 2:00 o'clock is called the **static zone.** This area is reserved for the mobile cabinet and nitrous oxide apparatus. The area from 2:00 to 5:00 o'clock is the **assistant's zone.** Although the assistant will not move as much as the dentist, nothing should be positioned in this area that would hamper the assistant's free access to the oral cavity, mobile cabinet and dental unit. The area form 5:00 to 8:00 o'clock is the **transfer zone.** This area is reserved for the transfer of instruments, medicaments, and supplies to the dentist. Also, the dental unit should be positioned within this arc.

Other considerations for an efficient four-handed dental delivery system:

- **Concerning the transfer of instruments** → the hand instrument to be transferred to the dentist is held by the assistant between the **thumb and the forefinger.**
- **Equipment selection** → whatever equipment is used it should be compatible for dentist and assistant.The position of the chairside assistant should be **higher than the dentist.**

Note: Venting the suction exhaust to the building exterior can reduce health hazards to the office staff from the central suction unit.

Medications such as diuretics, sedatives, hypnotics, antihistamines, antihypertensives, antipsychotics, antidepressants, and appetite suppressants have a (an) _____ which can cause xerostomia.

- Cholinergic affect
- Antiadrenergic affect
- Anticholinergic affect
- Adrenergic affect

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- **Anticholinergic affect**

***Xerostomia** is the #1 most common adverse side effect of medications.

Other etiologic factors for xerostomia include:

- Radiation therapy to head and neck
- Salivary gland surgery
- Autoimmune disorders such HIV infections, systemic lupus erythematosus, rheumatoid arthritis, and Sjogren's Syndrome
- Endocrine disorders such as diabetes and hyperthyroidism

Treatment for xerostomia:

- Consider stopping offending medication
- Commercial saliva substitute
- Fluoride Supplementation
- Scrupulous dental care is essential

Anticholinergic drugs *(which block receptor sites for acetylcholine)* decrease salivary flow and respiratory secretions during surgery. **Examples include:** atropine, scopolamine, methantheline and propantheline bromide.

Don't forget: Local anesthetics aid in reducing the flow of saliva during operative procedures by **reducing sensitivity and anxiety** during tooth preparation.

Remember: Cholinergic agents actually increase secretions, a cholinesterase inhibitor would also increase secretions because it would reduce the metabolism of acetylcholine.

When two proximal surfaces diverge from an area of contact, an embrasure is formed

- Facially only
- Lingually only
- Cervically and occlusally only
- Lingually, facially, occlusally and cervically

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• **Lingually, facially, occlusally and cervically**

There are four embrasures for every contact area:

1. Buccal *(or facial)*
2. Lingual *(usually larger than the facial)*
3. Occlusal *(or incisal)*
4. Cervical *(or gingival)* **Note:** In **posterior teeth,** the gingival tissue fills this embrasure. Normally it is **"col"** shaped when viewed in a faciolingual cross section.

Functions of embrasures:

1. Make a **spillway** for food during mastication
2. Make the teeth more **self-cleansing**
3. **Protect** the gingival tissue from undue frictional trauma, but at the same time provide the proper degree of stimulation to the tissue.

A **contact area** is an area in which the mesial and distal surfaces of adjacent teeth in the same arch make contact. A **contact point** is a point at which teeth of the opposing arches meet or touch in occlusion or closure.

The **height of contour** refers to the thickest portion or point of greatest circumference of the tooth when viewed form the incisal or occlusal surface. Its functions include forming the contact area on the mesial and distal surfaces and protecting the gingiva surrounding the tooth.

All of the following are **radiographic signs** of trauma from occlusion **except:**

- Hypercementosis
- Root resorption
- Periodontal pockets
- Alteration of the lamina dura
- Widening of the periodontal ligament space

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- **Periodontal pockets**

***Remember:** Radiographs **cannot determine** periodontal pocketing.

Some common **clinical signs of trauma** from occlusion include:

- Increased tooth mobility is the most common clinical sign
- Thermal sensitivity *(cold)* → presumably, this sensitivity is due to venous hyperemia of the tooth
- Attrition of the enamel
- Recession of the facial gingival tissue

Remember: Whenever a restoration is done, the **occlusion has to be right.** The degree of contact on the restoration should be to the **same degree** that teeth contact in that quadrant and on the opposite site.

The **radiograph of choice** for evaluating root surfaces, supporting bone and the periodontal ligament is the **periapical film.**

The primary purpose (s) of the proximal contact relationships between adjacent teeth in the dental arches is:

- To prevent periodontal pocket formation
- Twofold → this relationship serves both to stabilize the dental arches by the combined support of the individual teeth and to prevent the impingement of food material on interseptal tissues between the teeth
- To maintain the faciolingual dimension of the teeth
- To form the interproximal papillae

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- **Twofold → this relationship serves both to stabilize the dental arches by the combined support of the individual teeth and to prevent the impingement of food material on interseptal tissues between the teeth**

The proximal contact area functions to:

1. **Support** neighboring teeth *(stabilizes the dental arch)*
2. **Prevent** food particles from entering the interproximal areas
3. **Protect** the periodontium
4. **Form** embrasures

The loss of proximal contact between teeth may result in periodontal disease, malocclusion, food impaction, or drifting of teeth.

Remember: When viewed from the facial, all **premolars** have their contacts at the **junction of the occlusal and middle third.** From this same view, molars have a proximal contact located in the middle third. From the **occlusal view**, all posterior teeth have contacts, which are located **slightly buccal of the middle third** *(mesial and distal)*. This creates a wide lingual and a narrow facial embrasure.

OPERATIVE

A cold stimulus applied to a tooth will produce **no response** if the tooth:

- Has a periodontal pocket
- Has a hyperemic pulp
- Is non-vital
- Has acute pulpitis

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• **Is non-vital**

Pulpal necrosis is the death of the pulp. A tooth affected with a necrotic pulp may have no painful symptoms. It may appear discolored. The **EPT** *(electric pulp tester)* will be of value because there will be **no response** at any current level. The tooth sometimes responds to heat, **but will not** respond to cold. Treatment is root canal or extraction.

Note: Acute apical periodontitis is characterized by pain, which is commonly triggered by chewing or percussion. AAP *(acute apical periodontitis)* alone is **not indicative** of an irreversible pulpitis. It is indicative that the apical tissues are irritated, which may be associated with an otherwise vital pulp with a potentially reversible pulpitis. In the **absence** of acute pain, a negative EPT test or a frank apical radiolucency, a carious tooth with sensitivity to percussion **may** respond to caries control *(temporary filling)*. If it doesn't respond to a sedative filling, root canal is indicated.

Pulpal Diagnosis	Cold response	Treatment
Normal	Not delayed or very short linger	None needed
Rev. pulpitis	Lingers less than 10-15 seconds	Remove causative agent
Irreversible pulp	Lingers longer than 15 seconds	RCT or EXT
Necrotic pulp	No response	RCT or EXT

Direct pulp capping is the dressing of an exposed pulp with the aim of maintaining pulp vitality. Mechanical exposures _____ than carious exposures to be successfully capped.

- Are less likely
- Are more likely
- Are just as likely
- None of the above, the success of direct pulp capping has nothing to do with whether the exposure is mechanical or carious

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• **Are more likely**

Direct pulp capping involves the prompt application of a setting calcium hydroxide cement to a "pin-point" *(less than 1 mm in diameter)*, well isolated traumatic pulpal exposure. This procedure may be expected, in most instances, to stimulate the formation of a reparative **"dentin bridge"** over the exposure site and to preserve the underlying pulpal tissue in a healthy condition. **Note:** Direct pulp capping is especially successful in **immature teeth.**

Favorable factors for direct pulp capping include: the visual evidence of uninflamed *(pink)* pulp tissue, the absence of copious hemorrhage through the exposure, no previous symptoms of pulpitis, a small non-carious exposure *(a mechanical pulp exposure*), and a clean cavity uncontaminated with saliva.

The **failure** of this direct pulp capping procedure would be indicated by symptoms of pulpitis at any time or the lack of a vital response after several weeks or months.

The following adverse responses may occur following direct pulp capping procedures:

- Physical or microbial insult to the pulp may result in persistent inflammatory changes, which may culminate in partial or complete pulpal necrosis.
- Regulation of the mineralization processes involved in dentin bridge formation may become deranged, resulting in extensive calcification and obliteration of the pulp canal space by mineralized tissue.
- **Very rarely,** the differentiation of odontoclasts may be induced with the development of internal resorptive lesions.

Remember: Direct pulp capping **should not** be attempted on teeth with a history of pain, sensitivity to percussion or periapical radiolucencies *(root canal therapy may be indicated).*

The ideal amount of time from placing an **indirect pulp cap** until reopening the tooth to remove the remaining decay is:

- 7 - 10 days
- 2 - 3 weeks
- 1 month
- 3 - 4 months
- 1 year

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- **3 – 4 months**

Two types of pulp capping procedures:

1. **Indirect pulp cap** → a calcium hydroxide base is placed on a thin layer of **questionable dentin** remaining over the pulp. It is performed when a carious exposure is anticipated. After a 3-4 month waiting period, the tooth is reopened and the remaining decay is removed. During the waiting period, it is hoped that there will be **secondary dentin formation,** allowing complete removal of the decay without pulp exposure.

   Classic example: A radiograph of a first molar shows gross decay that may involve a horn of the dental pulp. The ideal treatment would be to do an indirect pulp cap and place a sedative filling *(IRM)*. If tooth remains asymptomatic, in 3-4 months you can re-enter the tooth and remove all decay with subsequent placement of a permanent filling. **Note:** If this patient had pain in the tooth *(aggravated by heat and tender to percussion),* and excavation of the carious lesion revealed exposure of the pulp horn **without evidence** of vital tissue, the emergency treatment **pending** eventual root canal therapy is to place a small cotton pellet dampened with eugenol over the exposure and seal the cavity with a temporary material *(IRM).*

   Rationale for indirect pulp capping → there are three dentinal layers in a carious lesion: **(1)** a necrotic, soft, brown dentin outer layer, teeming with bacteria; **(2)** a firmer, discolored dentin layer with fewer bacteria; and **(3)** a hard, discolored dentin deep layer with a minimal amount of bacteria invasion.

2. **Direct pulp cap** → a calcium hydroxide base is placed directly on a **pulpal exposure.**

Pulpal pain, either spontaneous or elicited by an irritant, that **lingers** more than 10 to 15 seconds suggests:

- Pulpal necrosis
- Pulpal hyperemia
- Irreversible pulpitis
- Acute apical periodontitis

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• **Irreversible pulpitis** → is sometimes called **"acute pulpitis"**

Irreversible pulpitis is an acute inflammation of the dental pulp characterized by intermittent spasms of pain, which become continuous. In the early stages, it may appear as a very severe hyperemia. As the condition continues, the pain may be described as a **gnawing or dull throbbing.** The pain is generally increased by heat and relieved by cold. The treatment **accepted by most clinicians** is pulp removal *(root canal therapy).* **Note:** The tooth is usually percussion positive.

At first, the pain is initiated and sustained only by the stimulus *(this is reversible pulpitis).* Later, as the pulpal swelling spreads from the initial area of damage or irritation to the rest of the pulpal tissue in the chamber, the pain initiated by the stimulus becomes more prolonged *(this is irreversible pulpitis).* If enough pulpal tissue becomes damaged, the pain may initiate or persist without any stimulus at all. At the same time, the degenerative inflammation of the pulp may reach down the entire length of the root or roots and begin to cause the apical PDL to become inflamed *(acute apical periodontitis → AAP).* Now the patient may have not only a throbbing toothache but also pressure sensitivity *(to the pressure of chewing or percussion).* This stage marks a later point in the pulpal degenerative timeline when the tooth is the "hottest" and usually the most difficult to get numb.

Important: Sometimes it is hard to distinguish between **reversible** and **irreversible** pulpitis, in which case **caries control** *(the placement of a temporary filling)* is a conservative approach toward making the final diagnosis. If a tooth responds well to this temporary filling, then the need for root canal therapy at this time is ruled out.

Remember: Reversible pulpitis or pulpal hyperemia is merely the engorgement of the pulpal vessels with blood. Once the causative agent *(i.e., bacteria or a restoration in hyperocclusion)* is removed or adjusted, the pulp will most likely return to normal.

A **short, painful** response to cold suggests:

- Pulpal necrosis
- Irreversible pulpitis
- Pulpal hyperemia
- Acute apical periodontitis

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• **Pulpal hyperemia**

***Hyperemia of the pulp is an **excessive accumulation** of blood in the pulp resulting in vascular congestion.

Pulpal hyperemia is sometimes called **"reversible pulpitis"** and may be caused by physical, chemical or bacterial insult. Following restoration placement, teeth often become hyperemic and are **sensitive to cold** for a few days. The pain is **not spontaneous** and **does not last longer** than approximately 10 seconds after the stimulus is removed. It is this fact, its short duration and low intensity, which distinguishes it from the pain of acute pulpitis *("irreversible pulpitis")*. **Remember:** Hyperemic teeth respond on a lower level of current on the EPT *(electric pulp tester)* than a normal tooth.

Treatment: if possible, the source *(e.g., high restoration)* should be removed. If indicated, a sedative restoration can be useful. If due to deep caries, an indirect pulp cap should be used only in permanent teeth and when pulp pathology is believed to be reversible *(e.g., no periapical pathology, no lingering spontaneous pain that might be worse overnight and stimulated pain of short duration only).* **Important:** Pulpal hyperemia caused by **bacterial insult** is a limited inflammation of the pulp. The tooth **can recover** if the caries is eliminated by timely operative treatment.

When the pulp becomes **severely inflamed** as indicated by a thermal stimulus producing pain that lasts long after the stimulus is removed *(longer than 15 seconds)*, this suggests **"irreversible pulpitis".** The pulp is unlikely to recover after removing the caries.
Remember: The **most effective** way to reduce injury to the pulp during tooth preparation is to **use adequate irrigation** to avoid heating of the dentin.

The **retention** of a pin:

- Decreases as the diameter of the pin increases
- Increases as the diameter of the pin increases
- Increases as the diameter of the pin decreases
- The retention of a pin has nothing to do with the diameter of the pin

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- **Increases as the diameter of the pin increases**

The **largest pin** that can safely be placed should be selected in any situation. As a rule, **one pin per missing axial line angle** should be used. The **optimal placement** is at the line angles or corners of the tooth, where the tooth / root mass is greatest and the risks of perforation into the pulp or furcation are minimal.

Types of Pins:
- Cemented
- Friction-lock
- Self-threading

Advantages:
- More conservative and less time involved than castings
- Enhances retention form *(adds walls)* and is an economical alternative to castings

Disadvantages:
- Can cause dentin crazing
- Microleakage can occur at pin channel
- Pins weaken amalgam alloy
- Placement can result in pulpal exposure, perforation and fracture of the tooth

Notes:

1. The function of a pin is to **retain** the restorative material. It **does not** increase the strength of the restorative material.
2. When placing pins in **endodontically** treated teeth, use only **self-threaded pins** or **cemented pins** *(not friction-locked pins)*.

The **most retentive** style of pins is:

- Cemented pins
- Friction-locked pins
- Self-threaded pins

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• **Self-threaded pins**

Self-threaded pin systems *(for example, TMS, Whaledent)* use holes sized just under the screw diameter. The **elasticity** *(resiliency)* **of the dentin** functions to retain the screwed pin. This system comes with a self-limiting drill of **optimal 2 mm depth** and self-shearing pins that guard against overtightening. This type of pin system is the **most frequently used** of the three types of pins.

The TMS *(Thread-mate system)* system has four sizes of pins *(regular, minim, minikin and minuta)*. They are available in titanium or stainless steel plated with gold.
Cemented pins are serrated stainless steel pins that are cemented into pinholes that are **larger** than the diameter of the pin.

Self-threading	Drill Size	Pin Diameter
Regular	0.027"	0.031"
Minim	0.021"	0.024"
Minikin	0.017"	0.019"
Minuta	0.0135"	0.015"

Friction-locked pins are tapped into pinholes that are smaller than the diameter of the pin. They are retained by the elasticity of the dentin.

Note: The increased strength of the latest dentin / enamel bonding agents, coupled with the revived use of retentive slots, pot-holes, grooves and channels, has led to a reduction in the use of pins. Examples of **dental adhesives** include: **Amalgambond Plus, All Bond 2, DenTastic and Easybond .** These systems allow adhesion to preconditioned substrate with the added benefits of retention and sealing of the restoration and a stronger total cohesive mass to support all remaining cuspal segments of the tooth.

Regarding the use of pins, all of the following statements are true **except:**

- Use one pin per missing axial line angle, cusp, or marginal ridge, up to a maximum of four
- Use large-diameter pins whenever possible
- Use the minimum number of pins compatible with adequate retention *(pins weaken amalgam)*
- Pins should extend 2 mm into dentin and restorative material
- Keep at least .5 to 1 mm of dentin between the pin and the DEJ
- Pins should be placed away from furcation areas and parallel to the external tooth surface
- Coating of pins with adhesion promoters such as Panavia and 4-META materials improves fracture resistance of composite and amalgam cores
- Pins are bent to make them parallel or to increase their retentiveness

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• **Pins are bent to make them parallel or to increase their retentiveness**

***This is **false;** pins **are not** to be bent to make them parallel or to increase their retentiveness. Occasionally, bending a pin may be necessary to allow for condensation of amalgam occlusogingivally. When pins require bending, a **bending tool must be used.** A hand instrument *(e.g., an amalgam condenser or spoon excavator)* should not be used.

The **main advantage** of pins is to improve the **retention** of large restorations. Unfortunately, pin retention techniques are **not without disadvantages.** Pins are known to **weaken the restorative material** into which they intrude. If placed by force, they can create stresses that cause crazing of the tooth structure. They may provide an additional deep path for microleakage. If placed in close proximity to the pulp, they may aggravate an existing pulp problem or create one. The use of pins may be **contraindicated** in young teeth with very large pulps and in teeth with reversible pulp pathology, which might be aggravated by instrumentation. Placement is always influenced by the limitations of access and vision.

Remember: Cusps to be restored with dental amalgam should be reduced by **2 mm** while forming a flat surface *(perpendicular to the occlusal forces).*

Note: After restoring a tooth, make sure **you check the occlusion very carefully.** If a restoration is left in supra-occlusion, the patient will return complaining of discomfort when **biting,** usually with no other symptoms.

The optimal interpin distance depends on the size of the pin to be used. The minimal interpin distance is _____ for the Minikin pin and _____ for the Minim pin.

- 0.5 mm, 2mm
- 1.0 mm, 3 mm
- 3 mm, 5 mm
- 5 mm, 7 mm

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- **3 mm, 5 mm**

***Maximal interpin distance results in **lower levels of stress** in dentin.

Ideally, pins should be placed 1 to 1.5 mm inside the cavosurface margin and at least .5 mm inside the dentinoenamel junction *(DEJ)*, if present. Placement of the pin channel **at least .5 mm away** from the DEJ helps prevent crazing or complete fracture of the remaining enamel. **Note:** The optimal depth of the **pinhole** into the dentin is **2 mm.**

The rule of thumb: Pins should be **2 mm** into dentin, **2 mm** within amalgam, and **1 mm** from the DEJ *(to be safe)* with no bends in the pins. **Important:** The **twist drill** used to prepare the pin channels must be angled so that it remains in dentin only. The **channel** should be prepared **parallel to** the external surface of the tooth.

When pins are placed nearer the occlusal surface, as in cuspal coverage areas, the pins should project only minimally into the restorative material *(2mm for amalgam)*; long pins near an area of occlusal loading will significantly weaken the amalgam; additionally, the purpose of the pin in cuspal coverage areas is to bind the cusp to the restoration and to resist lateral displacement with occlusal function.

Remember: If when attempting to drill a pin hole the drill enters a **vital pulp** chamber, the proper treatment is to allow the bleeding to stop, dry with a sterile paper point and place calcium hydroxide in the hole. Proceed with a better location for a pin hole. If a pin channel **perforates** the external surface of the tooth and all factors are favorable, a pin can be placed provided there is no extension beyond the surface of the tooth.

The **two** most frequently quoted **disadvantages** of using the rubber dam are:

- Time consumption
- Patient objection
- Cost
- Staff allergies to material

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- **Time consumption**
- **Patient objection**

***However**, if you become proficient in placing the rubber dam and explain to patients the **advantages** of using it, these claims will be eliminated.

Advantages of using the rubber dam:
- Dry, clean field
- Access and visibility
- Improved properties of dental materials
- Protection of the patient and the operator
- Operating efficiency

The following conditions may preclude the use of the rubber dam:
- Severely tilted teeth
- Some third molars
- Teeth that are not erupted sufficiently
- Some respiratory problems such as asthma or severe colds in which breathing through the nose is difficult

Remember: The use of a rubber dam is the **standard of care** when performing endodontics.

After placing the rubber dam which isolated the six mandibular anterior teeth, the dentist observed an unusual amount of wrinkling of the rubber dam between the isolated teeth. This wrinkling is the result of:

- The holes that were punched were too small
- The holes that were punched were too close together
- The holes that were punched were too far apart
- Crowding and overlapping of the anterior teeth

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- **The holes that were punched were too far apart**

Five functions of rubber dam isolation:

1. **Retracts** soft tissue, such as lips, cheeks and tongue
2. **Provides** for clean, dry field
3. **Protects** the patient by eliminating the possibility of the swallowing debris or instruments. Protects the dentist somewhat by isolating him / her from possible infectious conditions in the patient's mouth.
4. **Provides** for maximum physical properties of materials. **For example:** The rubber dam provides a dry field, which is essential for placement of amalgam restorations as well as cements. **Remember,** the cements that are placed under dry conditions have maximum strength. Also a dry field **prevents** delayed expansion of amalgam.
5. **Saves time** → rubber dam saves time due to the fact that the operator can work more efficiently in a clean, dry field where visibility is not impaired.

In order for a rubber dam clamp to be **stable,** all four points of the jaws of the clamp must contact the tooth gingival to the height of contour. They should not extend beyond the line angles to prevent impingement of the interdental papilla and possible interference with placement of a wedge.

Note: A frequent cause of interdental papillae protruding from beneath the rubber dam is holes punched **too close together.**

Which **rubber dam frame** provides **more retraction** of the soft tissues?

- Woodbury
- Young's
- Both are the same

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- **Woodbury**

***However, the **Young's frame is most popular** → the U-shaped metal frame.

Important points about using the rubber dam:

- Apply lubricant to the lips and corners of the patient's mouth.
- Plot the hole on the rubber dam. **Always isolate a minimum of three teeth.** Punch the appropriate size hole for a particular tooth. **For a tooth bearing a clamp, the hole should be one size larger than those without a clamp.**
- An appropriate clamp is selected that will fit the most distal tooth to be isolated. The dam may either be stretched over the clamp with the clamp in place on the tooth; or the clamp may be carried with the dam and placed on the tooth in one step.
- Once the dam is placed, it is secured with either a Woodbury or Young's holder *(frame).*
- The rubber dam is inverted into the gingival sulcus using floss and / or a blast of air and an instrument such as a plastic instrument → this will prevent seepage of saliva.

Removal of the rubber dam → very important: Removal is the reverse of application, **except** all ligatures *(interdental septum of dam)* **must be cut and removed** before the dam is removed.

All of the following statements concerning **sealants** are true **except:**

- Sealants are highly effective in preventing pit and fissure caries
- When sealants are applied correctly, there is a decreased development of new carious lesions and a decreased progression of pre-existing lesions
- A close correlation exists between the retention of sealants and their effectiveness
- The effectiveness of sealants appears to be equal whether applied by dentists, dental hygienists, or dental assistants, provided that they have received appropriate training
- High viscosity sealants wet acid-etched tooth surfaces the best
- Dental sealants complement fluoride's role, which is more effective for smooth surface caries
- Dental sealants are underutilized

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• **High viscosity sealants wet acid-etched tooth surfaces the best**

***This is **false; low viscosity** sealants wet acid-etched tooth surfaces the best.

The placement of sealants is a **highly effective** means of preventing pit and fissure caries. It is safe. It is currently underused in both private and public dental health care delivery systems.
Note: The substantial reductions in dental decay that have occurred in the young population of the United States are due, for the most part, to the **use of systemic and topical fluorides.** The control of smooth surface caries that is provided by fluorides is of critical importance to the additional effectiveness of sealants.

Important: Sealants need **micro-mechanical retention.** The surfaces should be cleaned with a prophylaxis brush or rubber cup and pumice with water. When the teeth are effectively isolated from saliva contamination, the surfaces are dried and **acid-etched** by the application of a 30 to 50% phosphoric acid solution for one minute. The solution should be gently agitated during the application. This is then washed away and dried leaving a frosty-appearing etched surface.

Miscellaneous information concerning sealants:

1. The properties of sealants are closer to those of **unfilled direct resins** than to those of filled resins *(composites).*
2. Sealants are weak compared to filled resins *(composites).* The strength of a sealant is sacrificed in order to make it flow into the pits and fissures *(the viscosity needs to be low enough to flow into the pits and fissures).*
3. The most likely result of **inadvertently** sealing a small carious lesion in the occlusal surface of a tooth is that the **caries would be arrested.**
4. Research indicates that **pit and fissure sealants** are retained best on maxillary and mandibular bicuspids. **However**, the first molars *(max. and mand.)* benefit the most from sealants.

The **major factor** for selecting teeth for sealant application is:

- Age
- Caries risk
- Family history
- Health status

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• **Caries risk**

The success of a sealant is highly dependent upon obtaining and maintaining an intimate adaptation of the sealant to the tooth surface and thereby hopefully sealing it. Research has demonstrated that caries protection is **100% in pits and fissures that remain completely sealed.** Curing of sealant materials occurs one of two ways. Some sealants are chemically cured through a process called **autopolymerization.** These materials are dispensed as two components. As soon as the components are mixed the polymerization begins. The curing is complete in approximately 60 seconds. Other sealant material is cured with **visible light.** When using the light cured materials, it is very important the curing light is of high quality and is tested frequently for the value of the light emitted. The retention rates for chemically cured and light cured sealants have been compared in numerous studies and offer similar results.

Components of pit and fissure sealants:

- Monomer → **BIS-GMA** *(may be diluted with TEGDMA to reduce viscosity)*
- Initiator → benzoyl peroxide *(in self-cured)* and **diketone** *(in light-cured)*
- Accelerator → amine *(in self-cured)*
- Opaque filler → small amounts of TiO2 *(titanium oxide)* are added to make appearance slightly different from the occlusal enamel.

Sealant materials can be unfilled, filled with an opaquer, clear, and colored. There appears to be no difference in the retention rate. The filled materials are often easier to see and monitor, but the clear materials allow the operator to continue to see the filled fissures. Sometimes operators prefer the colored sealants to make monitoring retention easier. The newest type of sealant material available is fluoride releasing. The fluoride is released from the sealant after polymerization. The clinical significance of this has not yet been determined. The fluoride release is thought to be at the base of the sealed groove, which provides a fluoride rich layer available when remineralization of an incipient lesion takes place.

All of the following statements concerning **fluoride** are true **except:**

- The substantial reductions in dental decay that have occurred in the young population in the United States are due, for the most part, to the use of systemic and topical fluorides
- Fluoride reduces caries by increasing the enamel hardness
- Fluoride converts hydroxyapatite into fluorapatite by the substitution of the OH ion for the fluoride ion
- The fluoride ion decreases the solubility of the hydroxyapatite crystal and at the same time increases the size of the crystal itself

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• **Fluoride reduces caries by increasing the enamel hardness**

***This is **false;** fluoride **does not** make the enamel harder, **but reduces** its rate of solubility.

Mechanisms of action for fluoride in caries inhibition:

1. The topical effect of constant infusion of a low concentration of fluoride into the oral cavity is thought to **increase remineralization of enamel.**
2. Fluoride **inhibits glycolysis** in which sugar is converted to acid by bacteria. Specifically, fluoride ion inhibits the enzymatic production of **glucosyltransferase.**
3. The **incorporation of fluoride** into the enamel hydroxyapatite crystal. Fluoride ions replace the hydroxyl radicals of the hydroxyapatite crystals in the enamel, producing **fluorapatite.** This form of enamel is less soluble in catabolic acids produced by oral bacteria.
4. Recent research suggests a **bacteriocidal action** → topical fluoride may also prevent caries by directly interfering with the growth and metabolism of cariogenic bacteria — organisms such as Streptococcus mutans that produce acids responsible for decay. **Key point:** Fluoride inhibits the acid production that causes decay.

Notes:

1. The **concentration of fluoride** in the body fluids is regulated by an equilibrium relationship between bone and urinary excretion.
2. Fluoride ion is **easily exchanged** for hydroxyl ion in the lattice structure of enamel because the fluoride ion is slightly smaller than the hydroxyl ion, and has a greater affinity for the hydroxyapatite crystal than does the hydroxyl ion.

All of the following facts concerning **fluoride** are true **except:**

- Most fluoride is absorbed in the small intestine and excreted through the kidneys
- Fluorosis may result from excessive fluoride consumed during the mineralization stage of tooth development and can occur in permanent and deciduous teeth
- Fluoride passes the placental barrier slowly
- Fluoride is deposited in calcified tissues *(i.e., bones)*
- At 1.0 ppm fluoride is tasteless, colorless, and odorless
- Many communities cannot afford the high cost of fluoridated water systems
- The uptake of fluoride on teeth depends on the amount of either ingested or delivered fluoride in contact with the tooth throughout the day

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- **Many communities cannot afford the high cost of fluoridated water systems**

***This is **false;** the estimated cost in 1989 per person per year was 51 cents. However, costs have been shown to vary from 12 cents to $5.41 per person per year.

In relation to teeth, fluoride is characterized by the following:

- Its concentration increases in the **external layer of enamel throughout life.**
- Its concentration **increases during topical application,** but decreases for a few days after treatment.
- Increasing the fluoride content in the **external layers of a tooth** increases the resistance of the enamel to demineralization.
- Fluoride **uptake is greater in enamel** than in dentin or cementum.

Remember: The optimal concentration of fluoride in community drinking water depends upon the **average air temperature** and the **average water consumption.** For temperate climates it is **1 ppm,** for warmer and colder climates, the amount can be adjusted from **0.7 to 1.2 ppm,** respectively.

Notes:

1. In communities **without** fluoridated water supplies, the most cost-effective method of delivering fluoride to **6-12** year old children is through **school water fluoridation** *(as opposed to fluoride tablets, brushing with a fluoride gel or rinsing with fluorid mouthrinse).*
2. The **most effective** means of increasing the fluoride content in the external layers of teeth is the **daily** application of 1.23% acidulated phosphate fluoride in fitted trays for four minutes. Obviously this is not realistic, since we do not routinely do "daily" applications.

The **optimum fluoride concentration** in community drinking water is:

- 0.1 ppm
- 0.12 ppm
- 1.0 ppm
- 10.0 ppm

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• **1.0 ppm** *(1 part per million)*

Water fluoridation involves the addition of a fluoride containing compound, to a water supply, to obtain an optimal level of fluoride ion, which would best prevent the occurrence of dental caries. Community water supply systems add fluoride to water supplies in one of three forms: sodium silicofluoride, hydrofluorosilicic acid or sodium fluoride. The U.S. Public Health Service sets the optimum fluoride level at 0.7 to 1.2 ppm. For **temperate climates the optimum is 1.0 ppm,** due to the fact that people tend to drink more in warm weather, warmer weather dosage levels are much lower than those of colder weather *(for warmer or colder climates the amount is adjusted from 0.7 to 1.2 ppm).*

One of the most effective means of reducing caries activity in the dentition is by decreasing the solubility of the enamel to the acid attack of the microflora. This demineralization starts when the pH reaches 5.5.

The **main anticariogenic effect of fluoride** is by enhancing remineralization. In second place is its role in decreasing demineralization by increasing the resistance of the outer surface of the tooth, and lastly, the least understood property of fluoride are in its antimicrobial properties.

Fluoride's **main effect** occurs after the tooth has erupted above the gingiva *(called post-eruptive).* This topical effect happens when small amounts of fluoride are maintained in the mouth in saliva and dental plaque. Fluoride works by stopping or even reversing the tooth decay process. It keeps the tooth enamel strong and solid by preventing the loss of *(and enhancing the re-attachment of)* important minerals from the tooth enamel. Fluoridation of community water has been credited with reducing tooth decay by 50% - 60% in the United States since World War II. More recent estimates of this effect show decay reduction at 18% - 40%, which reflects that even in communities that are not optimally fluoridated, people are receiving some benefits from other sources *(e.g., bottled beverages, toothpaste).*

All of the following are **components** of sodium fluoride paste that can be used to treat root sensitivity **except:**

- Sodium fluoride
- Kaolin
- Glycerin
- Eugenol

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• **Eugenol**

***Sodium fluoride, kaolin and glycerin are present in the paste **in equal parts.**

The rationale of desensitization procedures is not fully understood. Some techniques may depend on denaturation of the superficial ends of Tomes' fibers or of nerve endings in dentin. Other procedures are designed to deposit an insoluble substance on the ends of the fibers or nerves to act as a barrier to stimuli. Still others are designed to stimulate secondary dentin formation thus insulating the pulp from external stimuli.

Remember: The **most accepted theory** to explain the unusual sensitivity and response of exposed root surfaces to various stimuli is the **hydrodynamic theory.** This theory postulates that the pain results form indirect innervation caused by **dentinal fluid movement in the tubules,** which stimulates mechanoreceptors near the predentin.

Technique: After cleaning the hypersensitive areas, rub the paste into the exposed root surfaces with a porte-polisher and orangewood stick or a rubber cup for 1 to 5 minutes. Satisfactory results are usually obtained. No caustic effects on the gingiva or mucosa result from contact with the paste; **however,** it is toxic if accidentally ingested.

Which of the following types and concentrations of fluoride should be recommended to a head and neck cancer patient for home-care custom tray use?

- 1.23% acidulated phosphate fluoride and 1% neutral sodium fluoride
- 0.4% stannous fluoride and 1.23% acidulated phosphate fluoride
- 0.4% stannous fluoride and 1% neutral sodium fluoride
- 1.23% acidulated phosphate fluoride, 0.4% stannous fluoride and 1% neutral sodium fluoride

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- **0.4% stannous fluoride and 1% neutral sodium fluoride**

The gel contains either **1.0% sodium fluoride** or **0.4% stannous fluoride.** For maximum benefit, the gel must be in direct contact with the teeth. Fluorides are recommended to protect these patients from post-irradiation caries. **Remember two important points: (1)** The fluoride found in commercial toothpastes is not adequate for people who have had head and neck radiation; and **(2)** These patients must continue to use the fluoride gel as directed for the rest of their life to protect their teeth from rampant decay.

Instructions for patient: The trays containing the fluoride are placed over the teeth for a prescribed period of time *(usually 10 minutes)*, and he / she may not eat or drink for at least 30 minutes. Usually this is done at night after toothbrushing and just before going to bed.

The **daily use of fluoride gel in custom trays** at home is indicated in the following situations:

- Rampant enamel or root caries in any age group
- Xerostomia
- Head and neck radiation therapy
- For use on abutment teeth under an overdenture
- Hypersensitive root surfaces

The most effective and least objectionable topical fluoride is:

- 0.2% Sodium fluoride
- Stannous flouride
- Acidulated phosphate fluoride *(APF)*
- 2% Sodium fluoride

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• **Acidulated phosphate fluoride *(APF)***

Important:

1. Acidulated phosphate fluoride *(APF)* is the most commonly applied in-office fluoride treatment.
2. The combination of 1.23% sodium fluoride and 1 M orthophosphoric acid results in acidulated phosphate fluoride. It is very stable in solution.
3. The most common recommended over-the-counter fluoride is 0.05% sodium fluoride.
4. Stannous fluoride *(8% F)* has a very bitter, metallic taste, may burn the mucosa, and has a short shelf life.
5. The tin ion in stannous fluoride may be responsible for staining the teeth, it may be beneficial for arresting root caries.

Fluoride Agents for Professional Application			
	NaF	APF	SnF_2
Concentration	Solution 2%	Gel or solution 1.23%	Solution 8%
Fluoride ion %	0.91%	1.23%	1.95%
ppm fluoride	9,040 ppm	12,300 ppm	19,360 ppm
mg Fl/ml	9.04	12.0	19.36
Efficacy	29%	28%	32%
Taste	Bland	Bitter without flavoring	Astringent
Tooth discoloration	None	None	Brown
Gingival reaction	None	None	Occasional

Match up the **fluoride concentration** with the appropriate **type of professionally applied topical fluoride.**

Topical Fluoride	Concentration
Sodium fluoride *(NaF)*	8.0%
Acidulated phosphate fluoride *(APF)*	2.0%
Stannous fluoride *(SnF_2)*	1.23%

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- **Sodium fluoride → 2.0%**
- **Acidulated phosphate fluoride → 1.23%**
- **Stannous fluoride → 8.0%**

Fluorapatite is the **most stable** reaction product of a topical application of fluoride.

Fluoride Concentrations Found in Other Compounds			
Type of Fluoride	**Dentifrice**	**Brush-on-Gel**	**Oral Rinse**
Sodium fluoride *(NaF)*	0.24%	1.1%	0.05% daily–swallow 0.02% weekly–expectorate
Sodium Monofluorophosphate *($NaPO_3F$)*	0.76	Not available	Not available
Acidulated phosphate fluoride *(APF)*	Not available	0.5%	0.04% daily–swallow or expectorate
Stannous fluoride *(SnF_2)*	Not available	0.04%	0.1%

A 15-year old female has lived in a **non-fluoridated** area all of her life. Which of the following is most likely to occur in this female when she moves to a community where the drinking water naturally contains 6 ppm of fluoride?

- A 50% reduction in dental caries
- Moderate dental fluorosis
- An increase in the amount of fluoride stored in her bones
- Gastrointestinal problems

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• **An increase in the amount of fluoride stored in her bones**

Moderate fluorosis **will not occur** since by age 15 all of her dentition has undergone complete enamel calcification *(with the possible exception of the third molars).*

There are **three types** of fluoride used in today's public drinking water supplies: sodium fluoride, sodium silicofluoride and hydrofluorosilicic acid. The first two types of fluoride are typically supplied in bulk, crystalline form and are regularly used at water treatment plants. Hydrofluorosilicic acid is supplied as an aqueous solution *(55 gallon drums)* and is commonly used at ground water well houses.

The use of the hydrofluorosilicic acid is advantageous because of the relatively inexpensive equipment required as compared with the equipment typically used with powered compounds. The hydrofluorosilicic acid is added directly to the water by a chemical dosing system typically consisting of a chemical feed pump, analyzer / controller, injector, drum scale and a 55 gallon drum of the acid solution. Dilution of the acid prior to adding to the water is not recommended because of the hazards involved in handling the acid and the errors associated with dilution.

Note: When fluoride concentrations in excess of 1.5 to 3.0 mg/L are consumed, a chemical combination can occur within the tooth enamel, resulting in mottled and permanently discolored teeth. Concentrations in excess of 4 mg/L can be toxic.

The pH of **acidulated phosphate fluoride gels** is in which of the following ranges?

- 1 to 4
- 4 to 7
- 7 to 10
- 10 to 12

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• **1 to 4**

Important:
• The pH of **acidulated phosphate fluoride** *(APF)* is approximately 3.0 to 3.5 *(acidic)*
• The pH of **sodium fluoride** *(NaF)* is approximately 9.2 *(basic)*
• The pH of **stannous fluoride** *(SnF_2)* is approximately 2.1 to 2.3 *(acidic)*

Note: Acidulated phosphate fluoride is **contraindicated on porcelain and composite restorations** due to the fact that it has been shown to cause pitting and etching of these materials. Acidulated fluorides should be **avoided on implant patients → may corrode the surface of titanium implants.**

When painting fluoride on, it is very important to isolate the teeth with cotton rolls. When using fluoride trays, cotton rolls may be placed in the premolar areas to increase patient comfort and help keep the fluoride in place. Patients are asked not to brush, rinse, eat or drink 30 minutes after a fluoride treatment so that the fluoride is left undisturbed and is able to continue reacting with the hydroxyapatite for some time after the initial application. Fluoride treatments should be applied for four minutes, although there are now some one-minute products that are being marketed.

How many **milligrams of fluoride** are contained in an 8.2-ounce tube of toothpaste sold in the United States containing approximately 1100 ppm F^- ?

- 130 mg
- 232 mg
- 350 mg
- 400 mg

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• **232 mg**

Note: For tubes sold in other countries the concentrations of fluoride varies alot, and so these calculations would be incorrect.

Either sodium fluoride or sodium monofluorophosphate contains approximately **1.0 mg** of fluoride per gram of toothpaste. To solve the question, convert ounces to grams: **8.2 ounces x 28.35 = 232 grams** *(approximately)*. Since there is approximately 1.0 mg of fluoride per gram of toothpaste, the answer is **232 milligrams** of fluoride.

For an adult, the lethal dose of fluoride is between 2.5 and 10 g, with the average lethal dose being 4 - 5g. Death has occurred in infants with as little as 0.25 g *(250 mg)*. Death is likely in a chid who ingests more than 15 mgF/kg body weight *(for example, 5 teaspoons of APF gel for a 44 lb. child)*, so it is really weight dependent. Most fluoride is rapidly absorbed by the small intestine and stomach *(an acidic pH is required to facilitate this diffusion process)*. The currently accepted estimate for the minimum lethal dose of fluoride is 5 mgF/kg *(i.e. 5 milligrams of fluoride for each kilogram of bodyweight)*. This is called the Probable Toxic Dose or PTD. The PTD, 5.0 mgF/kg, is defined as the dose of ingested fluoride that should trigger immediate therapeutic intervention and hospitalization because of the likelihood of serious toxic consequences.

Fluorides currently **used in dentifrices** sold over-the-counter in the United States:
• Sodium fluoride
• Sodium monofluorophosphate
***Stannous fluoride is **not used** in any currently approved dentifrice in the United States

Some **abrasives** used in dentifrices:
• Silica
• Calcium carbonate
• Dicalcium phosphate *(may inactivate the fluoride ion)*
• Dicalcium phosphate dihydrate

If a topical fluoride is to be used in conjunction with a pit and fissure sealant, which sequence is used?

- Fluoride before sealant
- Fluoride after sealant
- Fluoride should not be used
- It really dose not make any difference

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• **Fluoride after sealant**

When a topical fluoride is used, it must be applied either before the conditioner *(acid etchant)* or after the sealant. When cleaning the tooth prior to application, no glycerine containing products can be used. Applying fluoride after the conditioner will decrease the bond strength.

Technique for the application of sealants:

- Gross debridement of enamel surfaces — use plain flour or pumice. **Do not use** anything that contains fluoride. It will **inhibit** etching.
- Rinse thoroughly
- Isolate teeth either with **rubber dam or cotton roll**
- Dry isolated teeth thoroughly
- Acid etch teeth with **phosphoric acid** *(30-50% concentration)*. Apply etchant by gentle dabbing of the enamel surface. Recommended etching time for permanent teeth is **one minute;** for primary teeth it is **two minutes.**
- Rinse and dry teeth thoroughly. Properly etched surface will appear **dull and chalky.**
- Apply sealant *(procedure differs with method of polymerization)*
- Evaluate results, floss tooth, **check occlusion**

OPERATIVE

S / FI

Studies have shown that incipient carious lesions _____ after sealant placement.

- Progressively get bigger
- Are arrested
- Spread rapidly into the interproximal areas
- Remain the same

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• **Are arrested**

Studies have carefully demonstrated that once an incipient carious lesion is sealed, the caries is stopped for most intensive purposes. Since caries is caused by aerobic microorganisms, once sealed over with a sealant an anaerobic environment is created in which the microorganisms become static or die and there is no further action because there is no metabolite to reach the organisms, there is no oxygen for them to exist in their environment, therefore they die.

Notes:

1. **Sealants act as a physical barrier** in preventing bacteria from accumulating in the pits and fissures of the teeth.
2. **Care should be taken not** to mix the sealant resin too vigorously prior to placement or to over-manipulate the sealant resin upon placement. Either of these errors could incorporate air into the sealant resin, resulting in a void in the surface of the sealant.
3. **As long as a sealant** remains **intact**, decay will **not** develop underneath it.
4. **The following factors** may influence which teeth are candidates for sealants: the presence of interproximal decay, patient age and how caries prone the patient is.
5. **Sealants should be placed** right after the tooth has fully erupted, before the decay process has had a chance to begin. For permanent molar placement, this would usually be around age 6 and 12, give or take 6 months.
6. **Although sealants are most commonly placed** on permanent molars, they may be placed on deciduous teeth in the following instances: the presence of deep pits and fissures, a very caries prone mouth and a tooth that is not likely to be exfoliated any time soon.
7. The most common reason for sealant failure **is salivary contamination,** usually due to inadequate isolation.

OPERATIVE

Terms

Deformations over time in response to a constant stress is called:

- Creep
- Modulus of elasticity
- Resilience
- Brittleness

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• **Creep** → or strain relaxation

Materials which are relatively weak or which are relatively close to their melting temperature are more susceptible to creep. Dental wax deforms *(creeps)* under its own weight over short periods of time. Traditional dental amalgam restorations are involved in intraoral creep.

Brittleness is generally considered to be the opposite of toughness. A brittle material is apt to fracture at or near its proportional limit. A brittle material has a high compressive strength **but** a low tensile strength *(e.g., amalgam)*. **Note:** This is why amalgam preparations **do not** have beveled margins *(they need butt joints)*.

The **modulus of elasticity** is a measure of the **stiffness or rigidity** of a material *(it is the ratio of stress to the strain below the elastic limit)*. **Important:** The higher the modulus of elasticity, the stiffer, or more rigid, the material and the less strain it exhibits for a given stress.

Resilience is the energy that a material can absorb **before** the onset of any plastic deformation.

Remember: Toughness is the property of being **difficult to break.** It is affected by the yield strength, percent elongation, and the modulus of elasticity.

OPERATIVE Terms

Loading of a restoration beyond the material's elastic limit produces:

- Stress relaxation
- Plastic deformation only
- Elastic deformation only
- Both elastic and plastic deformation

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• **Both elastic and plastic deformation**

The **elastic limit** is the greatest stress to which a material can be subjected, such that it will return to its original dimensions when the forces are released. Up to the elastic limit only elastic deformation is involved, but beyond that, there is a combination of elastic and plastic deformation, with the plastic portion increasing and the elastic portion decreasing up to the breaking point.

An example would be if a small tensile stress is induced in a wire, the resulting strain might be such that the wire **will return** to its original length when the load is removed. If the load is increased progressively in small increments and then released after each addition of stress, a stress value finally will be found at which the wire **does not** return to its original length after the load is removed. In such **a case,** the wire is said to have been stressed beyond its elastic limit.

The **proportional limit** is the greatest stress, which may be produced in a material such that the stress is directly proportional to the strain. A material that has a high proportional limit compared to one with a lower proportional limit also has more **resistance to permanent deformation.**

Although the two terms, **elastic limit** and **proportional limit,** are defined differently, their magnitudes are so nearly the same that for all practical purposes the terms can often be **used interchangeably.**

Note: The **yield strength** represents a stress **slightly higher** than the **proportional limit.**

In general, ductility _____ in temperature, whereas malleability _____ in temperature.

- increases with increase, decreases with increase
- decreases with increase, increases with increase
- None of the above

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• **decreases with increase, increases with increase**

Ductility is the ability of a metal to easily be worked into desired shapes *(for example, the ability to form a wire from a metal).* These materials undergo extensive plastic deformation prior to fracture *(in tension).* Ductility is dependent upon **plasticity and tensile strength. Note:** Ductility is usually expressed in terms of the **percent elongation;** the higher the value, the more ductile the alloy.

A related term is **malleability,** which describes a metal being able to be hammered *(compression)* into a thin sheet without rupture. It is also dependent on plasticity, but is not as dependent upon tensile strength as is ductility.

Gold is the most ductile and malleable metal, and silver is second. Of the metals of interest to the dentist, platinum ranks third in ductility and copper ranks third in malleability.

An enamel bonding agent which bonds together etched enamel with dental composite is a classic:

- Dental cohesive joint
- Dental adhesive joint
- Dental absorptive joint

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• **Dental adhesive joint**

Adhesion is a process of solid and / or liquid interaction of one material *(adhesive or adherent)* with another *(adherend)* at a single interface. Most instances of **dental adhesion** are also called **dental bonding.** A pit and fissure sealant bonded to etched enamel is a case of dental adhesion.

An **adhesive joint** is the result of interactions of a layer of intermediate material *(adhesive or adherant)* with two surfaces *(adherends)* producing two adhesive interfaces. Examples include orthodontic bracket bonding resin, enamel bonding system for a composite resin, and a bonded porcelain veneer.

There are two principal types of adhesion:

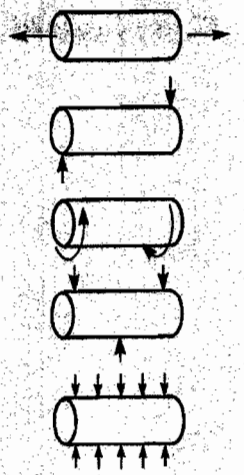
1. **Physical forces** → called van der Waals forces
2. **Chemical forces** → called chemisorption

Adhesive potential can be predicted by measuring the spreading or **wetting** of the adhesive over the surface of the substrate. This is done by determining the contact angle of the drop of adhesive as it spreads out. **The smaller the angle, the greater the wetting and thus the potential for adhesion.**

OPERATIVE Terms

Match the following mechanical properties of loading terms with their correct pictures.

- Shear
- Flexion
- Torsion
- Compression
- Diametral compression / tension
- Tension



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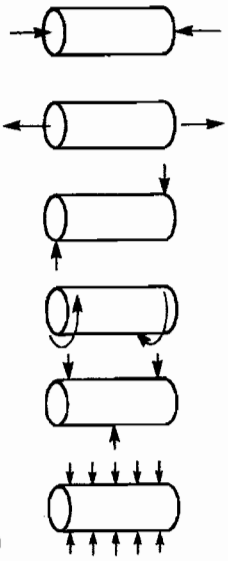
- **Compression**
- **Tension**
- **Shear**
- **Torsion**
- **Flexion**
- **Diametral compression / tension**



***Strain** is the actual change in shape or deformation that accompanies any stress.

Which of the following is produced by secondary odontoblasts in response to irritants?

- Primary dentin
- Secondary dentin
- Tertiary dentin
- Sclerotic dentin

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• **Tertiary dentin** → also known as reparative or reactive dentin

Throughout life, the dentin will respond to environmental changes *(normal wear, caries, operative procedures, etc.)* These changes initiate the **deposition of tertiary dentin** which is formed by **replacement odontoblasts** *(termed secondary odontoblasts)*. This reparative dentin will be limited to the site of irritation. The composition of **reparative and secondary dentin** is basically the same *(reparative dentin is more irregular)* and they differ only in location of deposition.

If the environmental insult is strong enough, it will kill the odontoblast and its tubular process, leaving the tubule empty. If there is a collection of empty tubules, they are referred to as **dead tracts.** In time, these tubules *(dead tracts)* will calcify. The term used to describe the tubules that become calcified is **sclerotic dentin.**

Primary dentin is the dentin forming the **initial shape** of the tooth. It is deposited **before** completion of the apical foramen.

Secondary dentin is dentin that is formed **after completion** of the apical foramen. It is formed at a slower rate than primary dentin as functional stresses are placed on a tooth. Secondary dentin is a regular and somewhat uniform layer of dentin around the pulp cavity.

Note: The junction between **primary and secondary** dentin is characterized by a sharp change in the direction of dentinal tubules.

OPERATIVE

Terms

Which of the following is defined as "that form the cavity preparation takes to **aid the operator** in preparing, placing, or finishing the restoration"?

- Retention form
- Resistance form
- Convenience form

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• **Convenience form**

Retention form is that form the cavity takes to **resist dislodgement** or **displacement** of the restoration. For example, the buccal and lingual walls of a **Class I** amalgam preparation converge occlusally; thus preventing dislodgement of the amalgam. For Class II restorations, this resistance to dislodgement is provided by the occlusal dovetail and retention grooves in the **proximoaxial line angles.**

Resistance form is that form the cavity walls take to resist the forces of mastication. Resistance form **prevents fracture** of the restoration and the tooth.

Examples: Severely undermined cusps should be reduced so as to prevent fracture. The axiopulpal line angle in Class II amalgam preparation is **rounded or beveled** so as to reduce concentration of stresses and thus prevent fracture of the brittle amalgam. Also, proper angulation of cavity walls *(converging)* and pulpal and gingival walls that are perpendicular to occlusal forces *(flat walls at right angles to the long axis of the tooth)* help achieve resistance form.

Important: The most common cause of fracture at the isthmus of a Class II amalgam restoration is **inadequate depth at the isthmus area** *(must be adequate depth to obtain resistance form).* Most detrimental to the strength of a posterior tooth in a cavity preparation is an increase in **faciolingual width.**

Remember: G.V. Black, is known as the father of modern dentistry. He is known for his principles of tooth preparations, in which he outlines the proper methods to prepare teeth for fillings. The phrase, **"extension for prevention"** is still famous in the dental community today and represents Black's idea that dentists should follow preventive measures to aid patients from developing tooth decay. Further, he organized a classification system with 5 categories for different patterns of tooth decay which is still in use today. Since that time, only one more category *(Class VI)* has been added to his classification system.

OPERATIVE Terms

The **outline form** of a cavity preparation is defined as:

- "That form the cavity takes to **resist** the forces of mastication"
- "That form the cavity takes to **resist** dislodgement or displacement of the restoration"
- "The shape or form of the cavity on the **surface** of the tooth"
- "The shape or form of the preparation after carious dentin has been excavated"
- "The shape or form the preparation assumes after the retention form has been completed"

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• **"The shape or form of the cavity on the surface of the tooth"**

Two important points should be remembered in obtaining the outline form:

1. In **general,** the margins should be placed in areas of lessened caries susceptibility. This principle is called **extension for prevention** *(phrased by G.V. Black)*
   ***TODAY → **modification of principle:** extension determined by extent of **DECAY and RESTORATIVE material.**

2. In general, all **undermined enamel** *(which is enamel not supported by sound dentin)* should be removed.

***The above two points are **influenced** by:
- → the **lateral spread** of decay at the DEJ
- → the **type of restorative material** to be used
- → the **tooth** and its relative position in the arch

Important: Extension *(for prevention)* can be restricted in patients with very low caries susceptibility.

Remember: If when establishing the **ideal outline form,** caries remains on any of the walls of preparation, the next step is to **extend the outline** form before excavating any caries.

OPERATIVE Terms

The brief but sharp electrical sensation one can receive when two dissimilar metals come into contact in the mouth is called:

- Electromagnetic pulse
- Alternating current corrosion
- Electrolyte explosion
- Galvanic shock

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• **Galvanic shock**

An example of this phenomenon: An amalgam restoration is placed on the occlusal surface of a lower tooth directly opposing a gold inlay in an upper tooth. Because both restorations are wet with saliva, an electric couple exists, with a difference in potential between the dissimilar restorations. When the two fillings are brought into contact, the potential is short-circuited through the two alloys. **The result is sharp pain.** Such post-operative pain **usually occurs immediately after insertion of a new restoration** and generally it gradually subsides and disappears in a few days.

The amount of electricity involved in galvanic shock can range up to 1.0 microamperes and 500 millivolts.

Match the dental material on the left with the appropriate Coefficient of Thermal Expansion *(ppm/c x 10)* on the right.

• Unfilled resins	14.4
• Composite resins	11.4
• Amalgam	81 - 92
• Direct gold	22 - 28
• Tooth	28 - 35

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Material	Coefficient of Thermal Expansion *(ppm/c x 10)*
Tooth	11.4
Direct Gold	14.4
Amalgam	22-28
Composite	28-35
Unfilled Resins	81-92

The coefficient of thermal expansion is a measure of the **tendency of a material to change** in shape when it is subjected to temperature changes *(for example, when eating or drinking hot or cold items)*. A possible break in the marginal seal of any restoration becomes imminent when there is a marked difference in the coefficient of expansion between the tooth and the restorative material. The closer the coefficient is to the tooth, the better *(direct gold is best)*. If the coefficient of thermal expansion is referenced to a single dimension, it is called the linear coefficient of thermal expansion *(LCTE)*. The LCTE is expressed in units of ppm / °C.

One of the consequences of thermal expansion and contraction differences between a restorative material and adjacent tooth structure is **percolation,** which is defined as the cyclic ingress and egress of fluids at the restoration margins. The possibility of **recurrent decay** at the margins **increases with increased percolation.**

OPERATIVE

Terms

Which cavities can involve **any teeth,** anterior or posterior?

- Class I
- Class II
- Class III
- Class IV
- Class V
- Class VI

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• **Class I , Class V & Class VI**

Cavity classification → standardized methods of recording the need for restoration exist to facilitate communication among clinicians, researchers, and dental educators. The most commonly accepted means of classifying cavities is by the names of the surfaces involved. Cavity type is classified further based on the type of treatment and anatomical area involved. This classification, developed by Dr. G. V. Black in 1908, is designated by Roman numerals as Class I, Class II, Class III, Class IV, Class V, and Class VI *(this is the only category that has been added to his original classification system)*. **Note:** It is important to remember that the classification relates to location and not size of the cavity.

- **Class I** cavities → involve the pits and fissures, while all other classifications involve smooth surfaces of the teeth.
- **Class II** cavities → involve the proximal surfaces and occlusal surfaces of premolars and molars.
- **Class III** cavities → involve the proximal surfaces of incisors and canines that do not involve the incisal angle.
- **Class IV** cavities → are located on the proximal surface of incisors and canines and do involve the incisal angle.
- **Class V** cavities → are on the facial or lingual surface of all teeth and do not involve a pit or fissure.
- **Class VI** cavities → are on the incisal edges of anterior teeth or on the occlusal cusp heights of posterior teeth.

Remember: The **best method** for definitive detection of incipient carious lesions on the interproximal surfaces of posterior teeth *(distal surface of canines through molars)* is by **bite-wing radiographs.**

OPERATIVE

Amal

Which Class III lesions **should not be filled** with composite resin?

- Mesial-lingual of canines
- Distal-lingual of laterals
- Mesial-lingual of centrals
- Distal-lingual of canines

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- **Distal-lingual of canines**

Composite resin **is not recommended** for Class III lesions on the distal-lingual aspect of canines *(use either amalgam or direct gold).* Composite material will not maintain the mesiodistal dimension of the tooth. **Note:** This may not be entirely true today due to the fact that there are much better wear resistant resins than in the past, **however,** for National Board questions composite is not recommended for this situation.

A lingual approach is made when preparing a Class III dental amalgam preparation for the distal of a canine because a lingual approach preserves the esthetic value of the facial surface.

Remember:
- A **bite-wing radiograph** is the best method to diagnose incipient carious lesions on the distal surface of canine teeth.
- A **diagnostic aid** to be used as a last resort to confirm the presence of a carious lesion on the proximal surface of an anterior tooth is mechanical separation *(usually with a wedge).*

Rule of thumb: When two teeth adjacent to each other have Class III lesions, the dentist should **prepare the larger one first** and **fill the smaller one first.** Access to the preps and shade matching are easier when you do both at the same appointment.

The ideal amount of dentin required between an amalgam restoration and the pulp for insulation is:

- 0.5 mm
- 1.0 mm
- 2.0 mm
- 3.0 mm

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• **2.0 mm** → 1.0 - 1.5 mm is acceptable but not ideal

***Amalgam is a **poor thermal insulator;** this is why a base of either calcium hydroxide or zinc oxide eugenol is placed under most amalgam restorations *(to provide thermal protection).*

Comparison of Materials			
Characteristic	**Direct Gold**	**Amalgam**	**Composite**
Close adaptation to cavity walls	Very Good	Good	Good
Coefficient of thermal expansion similar to tooth structure	Very Good	Good	Poor
Chemically acceptable to hard and soft tissues	Very Good	Good	Poor
Absence of irritation to gingiva	Very Good	Good	Poor
High edge strength	Very Good	Poor	Poor
Non-corroding in oral fluids	Very Good	Fair	N/A
Insolubility in oral fluids	Excellent	Excellent	Good
Clinical longevity	Very Good	Good	Fair
High abrasion resistance	Good	Good	Fair
Permanently restores M-D dimension	Good	Very Good	Fair
Withstands masticatory forces	Fair	Good	Poor
Versatility in use	Fair	Good	Poor
Imitates natural tooth color	Fair	Poor	Very Good
Ease of manipulation and placement	Poor	Good	Very Good
Thermal and electrical insulator	Very Good	Poor	Good

The **amount of mercury** remaining in a set amalgam restoration is related to:

- How much silver-tin matrix is in the amalgam before condensation
- How much zinc is in the amalgam alloy
- How much of the mercury-rich matrix is left in the amalgam after condensation
- How much tin is in the amalgam alloy

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• How much of the mercury-rich matrix is left in the amalgam after condensation

The **amount of mercury** remaining in a set amalgam restoration is related to how much of the mercury-rich matrix is left in the amalgam after condensation. **The key is to minimize the amount of matrix which forms during the reaction.** By condensing the amalgam mixture in the cavity preparation, the mercury-rich matrix will come to the surface and be removed by subsequent condensing and carving. The final amalgam restoration will be composed of mostly residual alloy and very little of the mercury-rich matrix.

The amount of mercury remaining in dental amalgam after condensation **directly affects:** the porosity of the restoration, the compressive strength of the restoration, the corrosive resistance of the restoration and the surface finish of the restoration.

Mercury is used to initiate the reaction with the alloy. Although an amalgam restoration is nontoxic, mercury is poisonous. Free mercury, in the form of vapor or liquid droplets, represents a significant health hazard in the dental office. The greatest potential hazard of chronic mercury toxicity comes from inhalation of mercury vapor. The vaporization is most likely to occur during condensation of the amalgam *(always use high speed suction).*
Note: Mercury hypersensitivity *(allergy)* is very rare *(1 in 100 million).*

Important point concerning condensation pressure: The area of the condenser point and the force exerted on it by the operator govern the condensation pressure. **The smaller the condenser point, the greater is the pressure exerted on the amalgam.** By doubling the diameter of the condenser point and doubling the hand pressure applied to the instrument, the operator actually exerts less condensation pressure.

The position of the **gingival margin** of a Class II amalgam restoration is dictated primarily by:

- Aesthetics
- The tooth being restored
- The extent of the carious lesion
- The thickness of the enamel

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- **The extent of the carious lesion**

***The gingival margin should clear the contact area to allow for adequate finishing of the enamel margins and placement of a matrix band.

Comparison of Conservative Class II Amalgam and Inlay Preparation		
Characteristic	**Class II Inlay**	**Class II Amalgam**
Occlusal isthmus width	1/3 intercuspal (Max)	1/3 intercuspal (Max)
Inclination of walls with respect to the occlusal surface	Diverge 2-5 degrees occlusally	Buccal and lingual walls converge approximately parallel to the external surface
Depth into dentin	0.5 mm	0.5 mm
Internal line angles	Sharp	Slightly rounded
Axio-pulpal line angle	Blunted or rounded	Blunted or rounded
Proximal retention form	Rectangular box Reverse bevel	Retentive grooves
Gingival wall	Form a reverse bevel at the axial gingival line angle, into the gingival wall	Gingivo-axial line angle slightly rounded

Which of the following factors tends to **reduce the setting expansion** of amalgam?

- Increasing the amount of mercury in the mix
- Increasing the silver content of alloy
- Increasing the trituration time
- Increasing the zinc content of the alloy

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• **Increasing the trituration time**

The **longer** the trituration time, **the smaller** the setting expansion. If the trituration is carried to the extent that the amalgam appears shiny and wet, the **strength will be maximal** and the smooth, carved surface will retain its luster long after polishing. A properly triturated amalgam appears **shiny, wet, smooth and homogenous.**

Inadequate trituration results in an amalgam mix that has a low strength and a rough surface that **will accelerate** corrosion. An undertriturated amalgam mixture will appear **dry** and **grainy.** Condensing this mixture will result in poor adaptation to the walls of the preparation, lacrimation between condensed increments, and reduced strength.

Remember: An **overtriturated** mix is **preferable** to an **undertriturated** mix.

Notes:

1. The discolored, corroded, superficial layer frequently seen on the surface of a dental amalgam restoration is most likely a **sulfide.**
2. When carving an amalgam restoration, be sure to trim the margins with a sharp instrument that rests on tooth structure *(this will prevent* **"ditching"** *the margins).*
3. If an amalgam **"chips"** when you are carving it, the reason for this is that the amalgam was condensed after its working time had elapsed.

The site of **two** retention grooves commonly used in a Class V amalgam are:

- Incisoaxial line angle
- Mesioaxial line angle
- Distoaxial line angle
- Gingivoaxial line angle

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- **Incisoaxial line angle**
- **Gingivoaxial line angle**

The **retention form** for a Class V amalgam preparation is provided by the gingival retention groove placed along the gingivoaxial line angle and the incisal retention groove placed along the incisoaxial line angle.

The **outline form** for the classical Class V amalgam preparation is a **deformed trapezoid** *(sometimes called "kidney-shaped")*. The outline form **is determined** by the location and size of the carious area.

The **non-parallel** mesial and distal walls of the preparation are **straight and parallel** to, **but never extending beyond,** the transitional line angles. The direction of these walls is determined by the direction of the enamel walls *(as is the decay pattern)*.

The occlusal and gingival walls of the preparation should be gently curved arcs as determined by the **contour of the free margin** of the gingival tissue. **Note:** These arcs should be as **parallel** to each other as possible.

Important points:

1. The **occlusal arc** will normally be the **longer** of the two.
2. The **gingival margin** will normally be at, or slightly below, the margin of the free gingiva.
3. For **incipient lesions,** the axial wall should be uniformly deep into dentin.

Delayed expansion of amalgam restorations is associated with which **two** factors?

- Insufficient trituration and condensation
- High residual mercury
- The contamination of the amalgam by moisture during trituration and condensation
- The failure to use a cavity varnish

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- **Insufficient trituration and condensation**
- **The contamination of the amalgam by moisture during trituration and condensation** → ***Important:** This is unquestionably the principal cause of failures.

If moisture is incorporated into an alloy that contains zinc, the water reacts with the zinc to produce hydrogen gas. The resulting pressure from the liberated gas produces severe expansion of the amalgam. This results in the following **clinical manifestations:**

- The amalgam protrudes from the cavity preparation
- Post operative pain
- Excessive corrosion

Important points to remember regarding amalgam:

1. The compressive strength is **greatly reduced** when amalgam is contaminated with moisture.
2. Amalgam has a coefficient of thermal expansion **approximately twice** that of tooth structure, thus, percolation does occur during temperature changes.
3. The tensile strength of amalgam is about **one-fifth to one-eighth** of its compressive strength → that is why enamel is needed to support amalgam at the margins of restorations. It is more abrasion-resistant than composite resin or unfilled resin.

How is **high mercury content** generally manifested in the clinical amalgam restoration?

- By tissue irritation adjacent to the restoration
- By severe marginal breakdown
- By delayed expansion
- By increased thermal shock resulting from hot and cold foods

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- **By severe marginal breakdown**

The most important consideration in the strength of the amalgam is the mercury content. If the mercury content **exceeds 55%,** a dramatic loss in strength results. Amalgam restorations that contain mercury levels of about 55% exhibit a **high incidence** of marginal breakdown, fracture, corrosion, and the surface finish of the restoration is not good.

Factors which influence the final mercury content of a restoration:

- Original mercury-alloy ratio → specific to each product but generally less than 1:1 so that amalgam contains 43% to 50% mercury
- Amount of trituration
- Condensation pressure and time involved in carrying out condensation

Removing the mercury-rich matrix by **proper condensation and carving** produces a stronger and more corrosion-resistant amalgam because it minimizes the formation of the matrix phases of amalgam, which are the least desirable parts of the set material.

Which phase of the amalgam reaction is prone to corrosion in clinical restorations?

- Gamma
- Gamma-one
- Gamma-two
- They all have the same resistance to corrosion

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• **Gamma-two**

The reaction that occurs between the alloy particles and the mercury can be summarized as follows:

Silver-tin alloy	+	Mercury	→	Silver-tin alloy	+	Silver-mercury	+	Tin-mercury
Ag_3Sn		Hg		Ag_3Sn		Ag_2Hg_3		Sn_3Hg
(gamma)				**(gamma)**		**(gamma-one)**		**(gamma-two)**

Gamma is the unreacted alloy. It is the strongest and corrodes the least. Forms 30% of volume of set amalgam. **Gamma-one** is the matrix for unreacted alloy and is the second strongest phase. Forms 60% of volume of set amalgam. **Gamma-two** is the weakest and softest phase. It is also the **most susceptible** to corrosion in the mouth. Forms 10% of volume of set amalgam. Volume decreases with time due to corrosion.

Notes:

1. **Smaller particle size** results in higher strength, lower flow, and better carvability.
2. **Spherical amalgams high in copper** usually have the best tensile and compressive characteristics.
3. **Copper contents over 6%** *("high-copper" alloys)* eliminate the gamma-two phase by forming a copper-tin phase resulting in superior properties.
4. Amalgam has a coefficient of thermal expansion **approximately twice** that of tooth structure.
5. The tensile strength of amalgam is about **one-eighth** of its compressive strength.

The _____line angle is beveled to reduce concentration of stresses when preparing a Class II amalgam preparation.

- Axiobuccal
- Axiolingual
- Axiopulpal
- None of the above

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- **Axiopulpal**

General properties of Class II amalgam preparations:

- All walls should meet the surface of the tooth at a 90° angle *(butt joint)*.
- Occlusal dovetail → provides resistance to proximal displacement.
- Pulpal floor should be flat → same as Class I.
- The buccal and lingual walls of the proximal section should converge occlusally → the extension of these walls is determined primarily by the position of the adjacent teeth in relation to the tooth being restored.
- The buccal, lingual, and gingival walls should be extended into the embrasures enough to allow easy cleaning → areas of lessened caries susceptibility.
- The axiopulpal line angle is beveled to reduce concentration of stresses → resistance form.
- Retention grooves are placed in the axiobuccal and axiolingual line angles and extended to the height of the axial wall → resistance to dislodgement.
- Lingually, it is often necessary to create a reverse curve in the outline. A reverse curve is a curve put into the buccal or lingual wall so the wall meets the external surface of the tooth at a 90° angle.

Note: Class II amalgam preps have independent retention and resistance form for both the proximal box and occlusal portion of the preparation.

Beveling the gingival **cavosurface margin** of the proximal box of a Class II amalgam preparation on a permanent tooth:

- Should result in a long bevel
- Is contraindicated because of the low edge strength of amalgam
- Should remove unsupported enamel which may fracture
- Is unnecessary since the tooth structure in this area is strong

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- **Should remove unsupported enamel which may fracture**

***The gingival cavosurface margin is beveled **only** if it is in the enamel. Beveling is not necessary if the gingival margin is within cementum.

The gingival cavosurface margin should be beveled to **remove any** unsupported enamel. The bevel is usually placed with a gingival margin trimmer. This gingival margin must be below any existing contact with the adjacent tooth in order to allow proper finishing of the gingival margin.

Note: The bevel is no steeper than necessary to ensure full-length enamel rods forming the gingival margin and is no wider than the enamel.

Remember: Enamel rods in the gingival third of the **primary teeth** extend occlusally from the DEJ, **eliminating the need** in Class II preparations for the gingival bevel that is required in permanent teeth.

Note: Primary molar teeth have marked **cervical constriction.** Therefore, when preparing the proximal portion of a Class II cavity prep, a satisfactory gingival seat may be difficult to obtain if the prep extends too deeply gingivally.

Which tooth requires special attention when preparing the **occlusal aspect** for a restoration?

- Maxillary **first** bicuspid
- Mandibular **first** bicuspid
- Maxillary **second** bicuspid
- Mandibular **second** bicuspid

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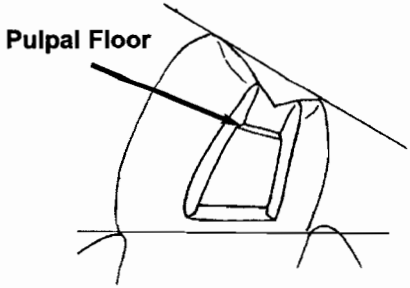
• **Mandibular first bicuspid**

The bur should be **tilted lingually** to prevent encroachment on the **facial pulp horn** and also to maintain dentinal support of the lingual cusp. The pulpal floor should be parallel to the occlusal plane of the tooth faciolingually.



Note: Pulpal floor slopes to coincide with the slope *(height)* of the cusps.

Remember: The area of the tooth that is most sensitive during cavity preparation is DEJ.

The **mesial and distal walls** of a Class I amalgam preparation should diverge slightly towards the occlusal surface to:

- Provide convenience form
- Provide resistance form
- Afford support for the mesial and distal marginal ridges
- Make condensing of the amalgam easier

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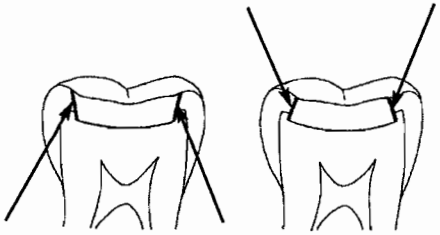
• **Afford support for the mesial and distal marginal ridges** *(see picture below)*

***This slight occlusal divergence prevents undermining the marginal ridges of their dentin support.



DIVERGING (Correct)

CONVERGING (Incorrect)

Notes:

1. This **divergence** of the mesial and distal walls holds true for Class I preparations for direct filling gold and gold inlays as well.
2. For **premolars** the distance from the margin of the mesial and distal wall to the proximal surface must not be less than 1.6 mm. For **molars** this minimal distance is 2 mm.

When is the **matrix band removed** from the tooth?

- Prior to the final carving of the restoration
- As soon as the amalgam has been condensed into the prep
- After the final carving of the restoration
- None of the above

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- **Prior to the final carving of the restoration**

Important points to remember regarding matrix bands:

- The reason for placing the matrix for a Class II amalgam restoration to protrude above the cavity preparation is to **allow for overfilling,** thus enhancing adequate cavosurface coverage.

- Contact areas are always carefully restored in all restorations in order to **protect** the gingival tissue. The matrix band should be burnished into contact with adjacent teeth → this will help assure contact.

- One of the most difficult teeth to adapt the matrix band to is the **mesial of a maxillary first premolar** → due to its developmental depression *(concavity in the cervical third of the mesial surface of the crown).*

Important: The wedging action between the teeth should provide enough separation to **compensate for the thickness of the matrix band.** This will ensure a positive contact relationship after the matrix is removed following the condensation and initial carving of the amalgam.

The diagonal slot opening on the Tofflemire matrix retainer *(also called the Universal matrix system)* is always placed facing the gingiva. **This:**

- Permits easy separation of the retainer from the band in an occlusal direction
- Allows for better contour of band to tooth
- Allows for easier wedge placement
- Is less harmful on the gingiva

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- **Permits easy separation of the retainer from the band in an occlusal direction**

In addition, the **larger circumference** of the matrix band is always placed toward the occlusal surface of the tooth. This accommodates the **larger tooth circumference** at the contact level.

The primary function of the matrix **is to restore anatomical contours and contact areas.** Other functions include providing a rigid wall to condense filling material against, preventing excess filling material from going subgingivally, and to some extent, limiting moisture contamination during condensation. A properly placed wedge will also protect the gingival tissue and help reduce moisture leakage into the cavity preparation.

Wedges are **inserted** from the facial or lingual embrasure, whichever is larger, slightly gingival to the gingival margin. The wedging action between the teeth should provide enough separation to **compensate for the thickness** of the matrix band.

Important: When placing a matrix band for a Class II amalgam restoration, the gingivo-occlusal width of the band should be trimmed to be **at least 1 mm greater** than the expected marginal ridge height.

What is the proper amount of time to wait until an amalgam restoration can be **finished and polished?**

- 2 hours
- 12 hours
- 24–48 hours
- Makes no difference

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- **24–48 hours**

The final finish of the amalgam restoration should **not be done** until after the amalgam is fully set. It should be delayed for at least 24 hours after condensation and preferably longer. By waiting you can be assured that the reactions between the alloy and mercury will have been completed and a more corrosion-resistant surface will be created.

Amalgam restorations should be finished and polished for three major reasons: **(1)** to reduce marginal discrepancies and to create a more hygienic restoration, **(2)** to reduce marginal breakdown which will reduce the chance of recurrent decay, and **(3)** to prevent tarnishing and to improve the appearance of the restoration.

Heat generation must be avoided. The use of dry polishing powders and discs can easily raise the surface temperature above the 60°C *(140°F)* danger point. Thus, a wet abrasive powder in a paste form is the agent of choice. **Note:** Heat will not only damage the pulp but also draws mercury to the surface of the restoration and an inferior restoration will result.

Final polishing may be accomplished using a rubber cup with flour of pumice followed by a high-luster agent, such as tin oxide.

Remember: When checking the occlusion on a newly condensed amalgam restoration, the marks left by the articulating paper should be of the same intensity as other markings in the same quadrant.

All of the following are true concerning a **Class V amalgam** preparation **except:**

- The **outline form** is determined primarily by the location of the free gingival margin
- The mesial, distal, gingival and incisal walls of the cavity preparation **diverge outward**
- The **retention form** is provided by the gingival retention groove along the gingivoaxial line angle and an incisal retention groove along the incisoaxial line angle
- A **cervical clamp** is usually necessary to retract gingival tissues

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- **The outline form is determined primarily by the location of the free gingival margin**

***This is **false;** the outline form is determined primarily by the **location and size of the carious lesion.**

Notes:

1. The Class V amalgam restoration is used to restore lesions from **caries, erosion and abrasion.**
2. Care should be taken not to **"ditch"** the cementum when finishing and polishing.
3. Occasionally you will notice that the gingival tissue has receded apically from the gingival margin of a Class V restoration that was previously polished. This may be related to irreversible tissue changes caused by inadvertently traumatizing the tissue when the restoration was being polished. **Key point** → **Be careful.**

Which of the following statements in reference to amalgam **is false?**

- Increased trituration time will **increase** compressive strength and **decrease** setting expansion
- A **decrease** in particle size will **decrease** compressive strength and increase setting expansion
- **Increased** condensation pressure will **increase** compressive strength and **decrease** setting expansion

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- **A decrease in particle size will decrease compressive strength and increase setting expansion**

***The **opposite** is true.

Dimensional change:

- **Setting Expansion**
  Most amalgam restorations show slight setting expansion, but not of clinical significance.
  The **more free mercury,** the more setting expansion *(and vice versa).*
  - The greater the time of trituration, the less the expansion.
  - The greater the pressure used in condensation, the less the expansion.
  - The smaller the particle size, the less the expansion.

- **Strength**
  Amalgam is brittle, but possesses **good compressive strength.** The most important consideration in the strength of the amalgam is the mercury content. Mercury content above 55% will cause a marked decrease in strength *(should be within 45-53% by weight).*
  - Higher condensation pressure increases strength.
  - The smaller the particle, the more strength.
  - The longer the trituration time, the more strength.
  - The fewer voids, the more strength.

An occlusal cavosurface bevel is contraindicated in the cavity preparation for an amalgam restoration. It is important to provide a _____cavosurface angle.

- 45°
- 60°
- 90°
- 115°

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• **90°**

Clinical experience has established that this butt joint margin of enamel and amalgam is the strongest. Amalgam is a brittle material with low edge strength and tends to chip under occlusal stress if its angle at the margins is less than 80° to 90°.

Comparison of the Cavosurface Margins for a Conservative Class II Amalgam and an Inlay Preparation		
Characteristic	**Class II Inlay**	**Class II Amalgam**
Occlusal cavosurface margin	**Beveled** to result in 40^0 marginal metal	90^0 with external surface
Proximal cavosurface margins	Outer planes carried into cleansable area *(provides access for finishing margins)*. Beveled to result in 40^0 marginal metal	90^0 angle with external surface
Gingival cavosurface margin	Beveled to result in 40^0 marginal metal	90^0 angle with external surface

Which component of dental amalgam prevents oxidation of the other metals while the alloy is being prepared by the manufacturer?

- Silver
- Copper
- Tin
- Zinc

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- **Zinc**

Constituents in Amalgam

Basic constituents:

- Silver (Ag) 40-70% → decreases setting time, increases the setting expansion and also increases the strength.
- **Tin** *(Sn)* 25-27% → decreases expansion, decreases strength, and increases setting time. **Note:** Influences the amalgam in an opposite manner to silver.
- **Copper** *(Cu)* 6% or less → ties up tin reducing gamma-2 formation, increases strength, reduces tarnish and corrosion, reduces creep *(reduces marginal deterioration).* **Note:** New alloys called **"high copper"** contain 9 - 30% copper. These alloys have less marginal breakdown and are less likely to corrode.
- **Mercury** *(Hg)* 3% max. → activates reaction with other alloy particles to produce different phases.

Other constituents:

- **Zinc** *(Zn)* 1% or less → used in manufacturing, decreases oxidation of other elements *(sacrificial anode).*
- **Palladium** *(Pd)* 1% or less → reduces corrosion.
- **Indium** *(In)* 1% or less → decreases surface tension *(reduces amount of mercury necessary and reduces emitted mercury vapor)*, reduces creep and marginal breakdown, and increases strength. ***Must be used in admixed alloys.

High copper and **low mercury** content of an amalgam restoration will tend to:

- Increase creep
- Decrease creep
- Will not effect creep

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• **Decrease creep**

Creep *(time-dependent deformation or strain relaxation)* is the deformation with time in response to a constant stress. It has been implicated as one of the **main causes for marginal fracture** of amalgam restorations.

Notes:
1. The higher the creep, the greater the degree of marginal deterioration.
2. Creep is time-dependent.

High copper and low mercury content of an amalgam restoration will tend to **decrease creep.** Altering the trituration time and condensation pressure can change the creep rate of an amalgam restoration:
- Both **undertrituration** and **overtrituration** tend to **increase** the creep rate.
- If there is a **delay** between trituration and condensation, the creep rate **increases.**
- Increasing the condensation pressure **decreases** the creep rate *(this will also decrease the final mercury content of the restoration).*

The marginal leakage of an amalgam restoration **decreases** as the restoration ages. Corrosion products are helpful in reducing marginal leakage around amalgam restorations. These corrosion products, such as **tin oxide** and **tin sulfide,** accumulate in the gap between the restoration and the tooth, thus providing an excellent seal.

OPERATIVE

Amal

The principal purpose of trituration is to:

- Dissolve all of the alloy particles in the mercury
- Coat the alloy particles with mercury
- Reduce the size of the crystals as rapidly as they form
- Reduce the size of the original alloy particles as much as possible

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• **Coat the alloy particles with mercury**

The object of trituration is to bring about an amalgamation of the mercury and alloy. Each individual alloy particle is coated with a slight film of oxide that prevents penetration by the mercury. During trituration this film is rubbed off and the clean metal is then readily attacked by the mercury.

Silver Alloys for Dental Amalgams:

Low copper alloys → 6% or less, traditional alloy
• Comminuted *(Irregular, filing, or lathe-cut)*
• Spherical particles

High copper alloys → 10-30% most common, corrosive resistant
• Spherical → sets faster and attains final mechanical properties more rapidly
• Comminuted can have zinc or be zinc free and also can be fine cut or microcut
• Combination *(admix)* mixture of spherical and comminuted particles

Dispersed phase alloy was the original admix alloy, mixture of comminuted traditional silver alloy and spherical particles of silver-copper eutectic alloy. Most commonly used alloy today.

Eutectic alloy made up of elements that when solid are insoluble and form a layered structure.

Note: Once amalgamation occurs, no free *(unreacted)* mercury is associated with the amalgam restoration. Has no toxic properties. However, if the amalgam is heated beyond 80°C, liquid mercury can form on the surface of the amalgam and its vapor presents a health hazard.

You are conducting an oral health education program for expectant parents. You remind the parents that because the local water supply is fluoridated at <0.3 ppm, the recommended supplemental dietary fluoride dosage for a child aged 6 months to 3 years is:

- 0.25 mg / day
- 0.50 mg / day
- 1.0 mg / day
- No fluoride supplement required

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• **0.25 mg / day**

Supplemental Fluoride Dosage Schedule *(in mg F per day)* According to fluoride Concentration of Drinking water			
	Concentration of Fluoride in the Water *(ppm)*		
Age in Years	**<0.3 ppm**	**0.3 ppm to 0.6 ppm**	**0.6 ppm**
Birth – 6 months	None	None	None
6 months – 3 yrs	0.25 mg	None	None
3 – 6 yrs	0.50	0.25	None
6 – 16 yrs	1.0 mg	0.50 mg	None

PHARMACOLOGY

Legend

Major Topic	Abbreviation	Major Topic	Abbreviation
Adrenergics / Anti-Adrenergics	Adren	Barbiturates	Barb
Anesthetics	Anesth	Cancer Chemotherapy	Cancer / Chemo
Anti-Anxiety Agents	Anti-Anx	Cardiovascular	Cv
Anti-Convulsants *(Epileptics)*	Anti-Convul	Cholinergic / Anti-Cholinergic	Chol
Anti-Depressants	Anti-Depres	Corticosteroids	Corticosteroids
Anti-Histamine	Anti-Hist	Diuretics	Diuretics
Anti-Infectives	Anti-Infect	Hypoglycemic Agents	Hypoglycemics
Anti-Psychotics	Anti-Psych	Miscellaneous	Misc.
Aspirin /Acetaminophen / NSAID's	Asp / Acet / NSAID's	Narcotic Analgesics	Narc Analg
		Terms / Definitions	Trms / Def

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True or False

Amphetamines are sympathomimetic amines that have a stimulating effect on both the central and peripheral nervous system.

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- **True**

Amphetamines **increase** systolic and diastolic blood pressures and act as weak bronchodilators and respiratory stimulants. They have a **high potential for abuse**, resulting in tolerance, psychological dependence, and severe social disability. Amphetamines pass readily into the CNS and cause a rapid release of norepinephrine in the brain. Abuse causes extreme violence and paranoid psychotic behavior.

Therapeutic **uses** of amphetamines:

- **Attention Deficit Hyperactivity Disorder** *(ADHD; hyperkinesis)* – dextroamphetamine *(Dexedrine)* and a mix of dextroamphetamine with amphetamine *(Adderall)*; *(Adderall XR)* sometimes used instead of methylphenidate *(Ritalin)*.
- **Narcolepsy** – dextroamphetamine *(Dexedrine)* is used to prevent daytime sleepiness in these patients.
- **Weight loss** – phentermine *(Ionamin)* – structurally similar to dextroamphetamine.

Remember: Sympathomimetic agents *(both indirect and direct)* mimic the effects of stimulation of organs and structures of the sympathetic nervous system.

The following effects result from what type of pharmacological agent: an **alpha adrenergic receptor blocker** *(alpha blocker)* or a **beta adrenergic receptor blocker** *(beta blocker)*?

- Tachycardia
- Lower blood pressure
- Vasodilation
- Orthostatic hypotension

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- **Alpha adrenergic blocker** *(alpha blocker)*

Examples of alpha blockers used in medicine:

- **Doxazosin** *(Cardura)* – selective for α_1 – used to treat hypertension
- **Prazosin** *(Minipress)* – selective for α_1 – used to treat hypertension
- **Terazosin** *(Hytrin)* – selective for α_1 – manage mild / moderate hypertension; treatment of benign prostate hyperplasia *(BPH)*

Alpha 1 blockers **cause** orthostatic hypotension, also known as **postural hypotension**. This is a fainting spell which occurs because of a rapid fall in blood pressure when moving from the supine to the upright position, as in getting rapidly out of the dental chair. The symptoms are similar to simple fainting, however the condition is related to positioning.

A drug that acts in a way **opposite** to the sympathetic nervous system is called a:

- Sympathomimetic
- Sympatholytic
- Sympathetic amine
- Adrenergic agent

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- **Sympatholytic**

***Also called **anti-adrenergic.**

Four kinds of sympatholytics: **Note:** They are all used to **treat hypertension.**

1. **Beta-adrenergic blockers**
   - Propranolol – blocks **both** $beta_1$ and $beta_2$ receptors
   - Timolol – blocks **both** $beta_1$ and $beta_2$ receptors
   - Nadolol – blocks **both** $beta_1$ and $beta_2$ receptors
   - Metoprolol – blocks $beta_1$ receptors
   - Atenolol – blocks $beta_1$ receptors
2. **Alpha-adrenergic blockers**
   - Tolazoline – blocks $alpha_2$ receptors
   - Phentolamine – blocks **both** $alpha_1$ and $alpha_2$ receptors
   - Phenoxybenzamine – blocks **both** $alpha_1$ and $alpha_2$ receptors
   - Prazosin – blocks $alpha_1$ receptors
3. **Centrally acting agents**
   - Clonidine)
   - Guanfacine) Inhibit adrenergic nerve transmission
   - Guanabenz) through actions within CNS
   - Methyldopa)
4. **Neuronal depleting agents**
   - Reserpine) Deplete catecholamine *(NE)* and serotonin
   - Guanethidine) stores from adrenergic terminals and in the brain

PHARMACOLOGY

Adren

What is the **pharmacologic** category of the following drugs?

- Epinephrine *(Adrenalin)*
- Phenylephrine *(Neo-Synephrine)*
- Albuterol *(Proventil; Ventolin)*
- Isoproterenol

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- **Adrenergic agonists**

Note: The adrenergic receptors are divided into the subtypes of alpha 1(α_1), alpha 2 (α_2), beta 1 (β_1) and beta 2 (β_2).

Therapeutic uses of adrenergic agonists:

- **Control superficial hemorrhage** – α_1 adrenergic agonist – vasoconstrictor – epinephrine *(Adrenalin)*
- **Allergic shock** *(anaphylaxis)* – α_1 adrenergic agonist – vasoconstrictor – epinephrine and β_2 adrenergic agonist – relaxation of bronchial smooth muscle *(dilation of airway)* – epinephrine
- **Nasal decongestant** – α_1 adrenergic agonist – vasoconstrictor – phenylephrine *(Neo-Synephrine)*
- **Bronchial relaxation, airway dilation** *(asthma)* – β_2 adrenergic agonists – albuterol *(Proventil; Ventolin)*
- **Cardiac stimulation** – β_1 adrenergic agonist – isoproterenol

Adrenaline *(epinephrine)* stimulates:

- Alpha$_1$ receptors only
- Beta$_1$ receptors only
- Both alpha$_{1,2}$ and beta$_{1,2}$ receptors
- Alpha$_1$ and beta$_1$ receptors only

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- **Both alpha$_{1,2}$ and beta$_{1,2}$ receptors**

Adrenergic Receptor Type	Characteristics	Neurotransmitters that Combine with Receptors
Alpha$_1$	Most common alpha receptor	Norepinephrine or Epinephrine
Alpha$_2$	Less common alpha receptor	Norepinephrine or Epinephrine
Beta$_1$	Less common beta receptor *(found on cells in heart)*	Norepinephrine or Epinephrine
Beta$_2$	Most common beta receptor	Epinephrine (*Norepinephrine generally combines either weakly or not at all)*

Remember: Alpha receptor responses are **predominantly excitatory** in nature, while **beta receptor** responses are **excitatory in nature in the heart and inhibitory elsewhere.**

All of the following drugs are **indirect-acting** adrenergic agonists, **except:**

- Tyramine
- Amphetamine
- Epinephrine
- Methamphetamine
- Hydroxyamphetamine

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- **Epinephrine**

Two types of adrenergic agonists:

1. **Direct-acting**: are those that interact directly with the alpha or beta receptors.
2. **Indirect-acting**: are those that cause the release of stored norepinephrine at the post-ganglionic nerve endings to produce their effects.

Direct-acting adrenergic agonists may be receptor selective or receptor non-selective:

- Phenylepherine = $alpha_1$ selective agonist
- Clonidine = $alpha_2$ selective agonist
- Dobutamine = $beta_1$ selective agonist
- Terbutaline = $beta_2$ selective agonist
- Albuterol = $beta_2$ selective agonist
- Epinephrine = $alpha_{1,2}$ and $beta_{1,2}$ agonist
- Norepinephrine = $alpha_{1,2}$ and $beta_1$ agonist
- Isoproterenol = $beta_{1,2}$ agonist

Remember: Some literature refers to **direct and indirect-acting adrenergic agonists** as direct and indirect-acting sympathomimetic agents. These terms are almost always synonymous. They are agents that bring about tissue responses resembling those produced by stimulation of the sympathetic nervous system.

All of the following drugs are **alpha blockers except:**

- Doxazosin *(Cardura)*
- Phenoxybenzamine hydrochloride *(Dibenzyline)*
- Phentolamine hydrochloride *(Regitine)*
- Prazosin *(Minipress)*
- Propranolol *(Inderal)*
- Terazosin *(Hytrin)*
- Tolazoline *(Priscoline)*

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- **Propranolol** *(Inderal)* ***Propranolol is a beta blocker *(specifically a* **nonselective beta blocker)**

Alpha blockers are medications that act by competitively inhibiting the action of catecholamines at the alpha receptor site. **They act on blood vessels, causing them to relax.** Alpha blockers are commonly used to reduce high blood pressure and to treat an enlarged prostate. There are **two types** of alpha blockers, which are classified according to the alpha receptor that they block *(alpha$_1$ or alpha$_2$)*.

- **Selective alpha antagonists:** only **block alpha$_1$ receptors** and are more commonly used to treat cardiac conditions *(hypertension)* and benign prostatic hyperplasia.
- **Nonselective alpha antagonists**: block both types of receptors and are generally not used for cardiac conditions because blocking both receptors can cause **tachycardia** *(rapid heart beat)* and **palpitations** *(pounding heart beat)*. They are used in the presurgical management of pheochromocytoma and sometimes in treating Raynaud's phenomenon.

Some examples of alpha blockers:

- Doxazosin – **selective alpha$_1$** blocker. Among the alpha$_1$ blockers it is the **preferred agent** for hypertension due to a **longer duration of action.**
- Terazosin – **selective alpha$_1$** blocker. Used to treat benign prostatic hypertrophy.
- Prazosin – **selective alpha$_1$** blocker. It is rarely used to treat hypertension due to unwanted adverse reactions.
- Tolazoline – **selective alpha$_2$** blocker. It is used to treat persistent pulmonary hypertension of the newborn.
- Phenoxybenzamine and phentolamine – both are **nonselective alpha$_1$ and alpha$_2$** blockers. They are used in the presurgical management of pheochromocytoma.

***Aside from **hypotension**, which is a major effect of alpha-blockade, alpha-blockers cause relatively few adverse reactions.

The heart is generally considered to have **predominantly** which type of **adrenergic receptors**?

- Alpha 1 (α_1) receptors
- Beta 1 (β_1) receptors
- Alpha 2 (α_2) receptors
- Beta 2 (β_2) receptors

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- **Beta 1 (β_1) receptors**

Receptor Types in the Sympathetic (Adrenergic) System		
Receptor Type	**Prominent Effector Organs**	**Response to Receptor Activation**
α_1	• Arterioles in skin, mucosa, viscera, and kidney *(resistance vessels)* • Veins	• Constriction • Constriction
α_2	• Presynaptic nerve endings • Postsynaptic in CNS	• Inhibit NE release • Decreased sympathetic tone
β_1	• Heart	• Increased heart rate • Increased force of contraction
β_2	• Arterioles *(and arteries in skeletal muscle)* • Bronchial and uterine smooth muscle	• Dilation • Relaxation

Alpha adrenergic receptors are located on:

- Vascular smooth muscle
- Presynaptic nerve terminals
- Blood platelets
- Fat cells
- Neurons in the CNS
- All of the above

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• **All of the above**

There are two main types of adrenergic receptors:

1. **Alpha** *(α)* **receptors** – responsible for most of the **excitatory** effects such as vasoconstriction and contraction of the uterus and spleen.
2. **Beta** *(ß)* **receptors** – responsible for most of the **inhibitory** effects such as vasodilation and relaxation of respiratory smooth muscle.

Note: Two important exceptions to the above: some alpha receptors mediate relaxation of gastrointestinal smooth muscle, and some beta receptors mediate increases in the force and rate of contractions of the heart.

Alpha receptors fall into two groups:

1. **Postjunctional alpha$_1$ adrenergic receptors** are found in radial smooth muscle of the iris, arteries, arterioles, and veins; in the GI tract.
2. **Prejunctional alpha$_2$ adrenergic receptors** mediate the inhibition of release of norepinephrine.

Beta receptors fall into two groups:

1. **Postjunctional beta$_1$ adrenergic receptors** are found in the myocardium *(heart)*, the intestinal tract smooth muscle, and adipose tissue.
2. **Postjunctional beta$_2$ adrenergic receptors** are found in bronchiolar and vascular smooth muscle.

True or False

Carbidopa, a drug used in the treatment of Parkinson's disease, only works when combine with levodopa.

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• **True**

Levodopa is used in the treatment of Parkinson's disease to replenish the brain's supply of **dopamine**, the neurotransmitter that is deficient in this disorder.

Administering **carbidopa in combination with levodopa** *(Sinemet)* reduces the required dose of levodopa by about 75%. When levodopa is given alone, much of the dose is metabolized before the drug reaches the brain. Therefore, large doses are required, and these are apt to cause unwanted side-effects. Carbidopa inhibits the peripheral decarboxylation of levodopa. This action simultaneously reduces the likelihood of peripheral side effects and allows more levodopa to reach the brain. Since carbidopa does not cross the blood-brain barrier, the levodopa in the brain is converted there to dopamine. Thus, co-administration of carbidopa plus levodopa in the form of **Sinemet** allows a significant reduction of levodopa dosage without reducing the desired effects.

Factors influencing **hepatic drug metabolism** include which of the following?

- Microsomal enzyme inhibition
- Microsomal enzyme induction
- Plasma protein binding
- Genetic factors
- Pathology
- All of the above

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- **All of the above**

Factors Influencing Hepatic Drug Metabolism

Microsomal enyzme inhibition → many drugs and environmental agents can inhibit many of the CYP isoforms of the P-450 microsomal drug metabolizing system. Thus many drugs which ordinarily metabolized by the particular CYP inhibited will not be effectively metabolized and will achieve higher than expected blood levels.

Microsomal enzyme induction → agents which induce higher levels of the microsomal drug metabolizing enzymes may cause a more rapid metabolism of other drugs thus resulting in lower than expected blood levels of a drug.

Plasma protein binding → drugs highly bound to plasma proteins will not enter the liver to be metabolized, thus resulting in a longer plasma half-life of the drug.

Genetic factors → There is individual variance through genetic factors which contribute to differing rates of drug metabolism in the hepatic microsomal enzyme system.

Pathology → Hepatic impairment and liver disease most often will result in impairment of the microsomal drug metabolizing system. This most often results in elevated levels of unmetabolized drug.

PHARMACOLOGY

Adren

All of the following neurons are **cholinergic except:**

- Preganglionic sympathetic neurons
- Preganglionic parasympathetic neurons
- Postganglionic sympathetic neurons
- Postganglionic parasympathetic neurons

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• **Postganglionic sympathetic neurons**

The autonomic nervous system has **cholinergic neurons** that secrete **acetylcholine** and **adrenergic neurons** that secrete **norepinephrine**. Preganglionic neurons of both divisions *(sympathetic and parasympathetic)* are **cholinergic**, as are the postganglionic neurons of the parasympathetic branch. Postganglionic neurons of the sympathetic branch are usually **adrenergic**. **One exception** is the sympathetic postganglionic neurons that innervate the sweat glands (**they are cholinergic**=*secrete acetylcholine*).

Note: Adrenergic blocking agents block the effect of impulses transmitted by the adrenergic **postganglionic neurons** of the sympathetic branch.

Remember: Alpha and beta adrenergic blocking agents act by **competitive inhibition** of postjunctional adrenergic receptors.

PHARMACOLOGY

Adren

Which of the following drugs is a **bronchodilator?**

- Epinephrine
- Albuterol *(Proventil)*
- Salmeterol *(Serevent)*
- Metaproterenol *(Alupent)*
- Aminophylline
- All of the above

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• **All of the above**

Asthma is a respiratory disorder characterized by recurring episodes of paroxysmal dyspnea, wheezing on expiration, coughing, and viscous mucoid bronchial secretions. The episodes may be precipitated by inhalation of allergens or pollutants, infection, vigorous exercise, or emotional stress.

Management of an **acute** asthma attack:

Epinephrine, albuterol, salmeterol and metaproterenol are **beta 2 adrenergic receptor agonists**. Thus they stimulate the beta receptors in the airway to cause bronchodilation. They are used to help reverse an acute asthmatic attack. They are taken via aerosol inhalers and nebulizer.

Aminophylline is an example of a **theophylline compound**. Theophylline compounds are administered orally as bronchodilators in reversible airway obstruction due to asthma or COPD *(chronic obstructive pulmonary disease)*. These drugs relax bronchial smooth muscle to improve airway function.

PHARMACOLOGY

Adren

What substance produces all of the following **physiologic** actions?

- Constrict arteriolar blood vessels *(vasoconstriction)*
- Relaxes bronchial smooth muscle *(bronchodilation)*
- Decrease blood volume in nasal tissues
- Causes a hypertensive response
- Produces physiological actions opposite to that of histamine

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- **Epinephrine**

Therapeutic **indications** for epinephrine:

- To alleviate symptoms of an **acute** asthma attack
- To treat bronchospasm associated with hypotension, as in anaphylaxis
- To treat hypersensitivity reactions
- **Agent of choice for anaphylactic reactions** *(given sublingually or subcutaneously)*
- It is added to local anesthetics as a vasoconstrictor **to prolong the activity of the local anesthetic solutions**, by decreasing the rate of diffusion and absorption from the injection site
- To restore cardiac activity in cardiac arrest
- To relieve congestion of the nose, sinuses, and throat

Common side effects: Headaches, agitation *(anxiety)*, and tachycardia.

Important: Epinephrine should be used with caution in patients with high blood pressure, hyperthyroidism, etc. These patients may have an increased sensitivity to epinephrine.

True or False

All of the following are **desirable effects** of epinephrine that make it the agent of choice for treating an anaphylactic reaction.

- It has **vasopressor** activity
- It has **bronchodilator** properties
- It has a **rapid onset of action**

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• **True**

Epinephrine is the **agent of choice** for treating an anaphylactic reaction because of its stimulatory effects on both alpha and beta adrenergic receptors. Alpha receptor stimulation results in a vasopressor response *(elevation of blood pressure)*. Beta receptor stimulation results in air way dilation and increased cardiac output. In this way, epinephrine counteracts the vascular effects of histamine-related anaphylaxis.

Note: Epinephrine is administered either intravenously, sublingually, subcutaneously or intramuscularly. There is a very rapid onset of action when given by these routes.

Other therapeutic **indications** for the use of epinephrine:

- To control superficial hemorrhage
- As a component of local anesthetic solutions to prolong duration of anesthesia
- Glaucoma → reduces internal eye pressure
- Asthma → bronchodilator properties

The average onset time for drug action after **subcutaneous administration:**

- Immediate
- 5 minutes
- 15 minutes
- 30 minutes

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- **15 minutes**

Routes of Drug Administration

- **Oral route:** It takes approximately 30 minutes for onset of drug effect after drug is swallowed. Oral route allows for use of many different dosage forms including tablets, capsules and liquids.

- **Intramuscular injection:** Onset of action of drugs injected into muscle occurs rapidly *(approx. 5 minutes)* because of high blood flow through the muscles.

- **Subcutaneous injection:** Onset of action of drugs injected under the skin takes about 15 minutes.

- **Inhalation:** Gases such as nitrous oxide are absorbed rapidly through the lungs and gain access to the general circulation within 5 minutes.

- **Topical:** This route includes ointments and creams applied to the skin and mucous membranes. It is not intended for systemic drug administration.

- **Patch delivery:** Skin patches release drug into the blood stream over a 12 to 24 hour period. It is not intended for rapid administration of drug.

"Epinephrine reversal" is a predictable result of the use of epinephrine in a patient who has received a *(an)*:

- Beta-blocker
- Alpha-blocker
- Adrenergic agonist
- All of the above

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• **Alpha-blocker**

Explanation of answer: One of the best known effects of the alpha-receptor blocking agents *(antiadrenergic)* is their ability to reverse the pressor action of adrenaline *(epinephrine)*. In the absence of blocking agents, epinephrine and norepinephrine both cause the blood pressure to rise. After the alpha-receptors have been blocked by an alpha blocker, the pressor effect of norepinephrine is reduced or abolished while epinephrine brings about a fall in blood pressure.

This is because **epinephrine** stimulates both alpha and beta-receptors in the cardiovascular system but norepinephrine only stimulates alpha-receptors *(NE lacks beta$_2$ effects)*. After blockage of the alpha-receptors, only the **beta-receptors can be stimulated.**

Note: A **pressor** response *(produces an increase in blood pressure)* is mediated by alpha-receptors and a **depressor** response *(produces a decrease in blood pressure)* is mediated by beta$_2$ receptors.

Of the following, which is a clinically significant adverse reaction due to **Metoprolol?**

- Drowsiness
- Hallucinations
- Arthralgia
- Dry mouth

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• **Drowsiness**

Metoprolol *(Lopressor, Toprol-XL)* is a **competitive, beta$_1$ selective blocker** *(cardioselective)*, which is most similar to Atenolol. It is used in the treatment of hypertension, acute angina pectoris and may be helpful after a heart attack.

Atenolol *(Tenormin)* is a **competitive, beta$_1$ selective blocker** *(cardioselective)*, with a long plasma half life *(long duration of action)*. It is used in the treatment of hypertension and chronic angina pectoris. Due to its low lipid solubility, Atenolol is renally eliminated, minimally metabolized, and has a low potential for causing CNS side effects compared to lipid soluble beta blockers *(e.g., Propranolol)*.

***Metoprolol and Atenolol** are both longer-acting and more predictable than Propranolol in producing therapeutic plasma levels. Because they are **beta$_1$ selective blockers**, they are also safer to use in patients with a history of **asthma or bronchitis.**

Acebutolol *(Sectral)* is a **beta$_1$ selective blocker** *(cardioselective)*. It is used to treat hypertension and to control **ventricular arrhythmias**. It has a low lipid solubility, which reduces its likelihood of producing adverse CNS effects. It also has mild **intrinsic sympathomimetic activity** *(partial agonist activity at beta$_2$ receptors)* similar to Pindolol.

Important points:

1. With all **"selective"** beta blockers, selectivity for the beta$_1$ receptor **is lost at high doses.** As the dose is increased they also block **the beta$_2$** receptors, thereby having effects on bronchial smooth muscle.
2. The **most common side effects** of beta blockers are weakness and drowsiness.

If norepinephrine or epinephrine were to stimulate or combine with the **alpha** receptors in the eye, which response would you **expect?**

- Miosis *(contraction of the pupil)*
- Mydriasis *(dilation of the pupil)*
- Neither of the above; norepinephrine and epinephrine do not stimulate or combine with alpha receptors in the eye

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• **Mydriasis *(dilation of the pupil)***

Organ / Tissue	Receptor Type	Response to Adrenergic Agonists
Heart	β_1 β_1 β_1 β_1	Increases conduction **velocity** Increases contraction **force** Increases contraction **rate** Increases cardiac **output**
Arterioles	α_1 β_2	Constricts cerebral arterioles Dilates skeletal muscle arterioles
Eye	α_1	Contracts sphincter muscle, producing mydriasis
Lung	β_2	Relaxes tracheal and bronchial muscles
Intestine	α, β α	Decreases peristalsis Contracts sphincter
Urinary bladder	α_1 β_1	Contracts trigone and sphincter muscles Relaxes detrusor muscle
Uterus	α β	Excites uterine contractions Inhibits uterine contractions

All of the following are **centrally acting antihypertensives except:**

- Methyldopa *(Aldomet)*
- Clonidine *(Catapres)*
- Tolazoline *(Priscoline)*
- Guanabenz *(Wytensin)*
- Guanfacine *(Tenex)*

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• **Tolazoline *(Priscoline)***

***Tolazoline is a parenteral antihypertensive agent. Tolazoline's actions are caused by a direct peripheral vasodilation, with **moderate alpha$_2$ adrenergic blocking** activity. The **centrally acting** antihypertensives are **alpha$_2$ selective agonists**. They reduce blood pressure by reducing cardiac output or vascular resistance *(or both)* to some degree

Methyldopa *(Aldomet)* is most effective when combined with a diuretic. It produces a **false transmitter** *(alpha-methylnorepinephrine)* which **replaces norepinephrine** in the vesicular storage sites and is released by the nerve impulse. It is of most benefit in treating hypertension in those patients with renal damage. **Adverse side effects** include: **Cardiovascular** *(orthostatic hypotension, bradycardia)*, **CNS** *(sedation and fever)*, **GI** *(colitis)*, and possible hepatitis and cirrhosis.

Clonidine *(Catapres)* is used in combination with a thiazide diuretic and hydralazine. It is an alpha$_2$ selective agonist that works by controlling nerve impulses along certain nerve pathways. As a result, it relaxes blood vessels so that blood passes through them more easily. This helps to lower blood pressure *(it reduces heart rate, cardiac output, and total peripheral resistance)*.

Guanfacine *(Tenex)* and **Guanabenz** *(Wytensin)* both stimulate central alpha$_2$ adrenergic receptors, thereby inhibiting sympathetic nervous system outflow, reducing peripheral vascular resistance and are used either alone or with a thiazide diuretic.

The **amide** local anesthetics are metabolized primarily in the:

- Lungs
- Plasma
- Liver
- Kidney

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• **Liver**

The **amide-type** local anesthetics that are used in dentistry and metabolized by the liver include:

- **Lidocaine** *(Xylocaine)*
- **Mepivacaine** *(Carbocaine)*
- **Prilocaine** *(Citanest)*
- **Bupivacaine** *(Marcaine)*

These anesthetics are metabolized by the hepatic microsomal enzyme system. The products formed do not have anesthetic actions and are excreted from the body by way of the kidney. These agents should be used with caution or not at all in patients with compromised liver function.

Note: Bupivacaine has the **longest duration of action** of any dental local anesthetic presently available.

Dental Anesthetic Preparations and Average Duration by Route

Product	Infiltration	Inferior Alveolar Block
Carbocaine 3% *(mepivacaine)*	20 minutes	40 minutes
Citanest Plain 4% *(prilocaine)*	20 minutes	2.5 hours
Citanest Forte w/ epinephrine *(prilocaine 4% w/ epinephrine 1:200,000)*	2.25 hours	3 hours
Lidocaine 2% w/ epinephrine *(1:100,000)*	60 minutes	90 minutes
Marcaine 0.5% w/ epinephrine *(bupivacaine w/ epinephrine 1:200,000)*	60 minutes	5-7 hours
Articaine 4% w/ epinephrine *(1:100,000)*	60 minutes	60 minutes

What are some **adverse side effects** of local anesthetics?

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Adverse effects caused by local anesthetics are categorized into **two major** groups:
1. Effects resulting from **toxicity**
2. Effects resulting from **allergy**

Toxicities:

- Too much anesthetic in the bloodstream can cause toxicities to the central nervous system *(CNS)* and cardiovascular system.
- The **CNS** effects include: restlessness, stimulation, tremors, convulsive seizures followed by CNS depression, slowed respiration and even coma.
- The **cardiovascular** effects include: bradycardia and reduction of cardiac output.

Allergy:

- Hypersensitivities or allergic reactions to local anesthetics, particularly from the amides, are rare. These reactions are manifested as dermatological reactions and edema at the injection site.
- Asthmatic wheezing syndromes have occurred in response to local anesthetic injections.
- Allergic reactions are **more prevalent** with the ester-type rather than the amide-type anesthetics.
- **Ester** local anesthetic allergic manifestations include: nasolabial swelling, itching, and oral mucosal swelling.

The **urinary elimination** of drugs is controlled by:

- Glomerular filtration
- Tubular reabsorption
- Active transport
- All of the above

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- **All of the above**

- **Glomerular filtration →** all drugs are filtered through the glomerulus to enter the renal tubules. The amount of drug varies according to the degree of plasma protein binding, and bound drugs are not subjected to filtration.

- **Tubular reabsorption →** once they enter the renal tubules, drugs may be reabsorbed back into the blood stream through the renal tubular cells. Reabsorption favors the highly lipid soluble agents; the converse is that highly polar compounds are not effectively reabsorbed and are effectively excreted from the renal tubules.

- **Active transport →** the rate of renal elimination also depends on whether active transport into or out of the tubular fluid occurs.

Other excretory pathways for drugs:

— The **gastrointestinal tract** excretes some drugs through the feces. This is not as prevalent as urinary excretion.
— Most drugs can be detected in saliva after administration, but the **salivary glands** are not considered a route of drug excretion since the drug is re-swallowed along with the saliva.
— **Lungs** excrete volatile compounds that were originally inhaled into the system. Nitrous oxide and the volatile general anesthetics are excreted by this route.
— Some drugs are excreted in the sweat, but this route accounts for only a small percentage of drug excretion.

Mefloquine *(Lariam)* belongs to a class of drugs which is useful in treating which of the following conditions?

- Malaria
- AIDS
- Hepatitis
- Cancer
- Chlamydia

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- **Malaria**

Mefloquine *(Lariam)* has been shown to be effective in treating malaria caused by **Plasmodium falciparum, P. vivax. malariae and P. ovale.**

Other antimalarial drugs are:

- **Chloroquin** *(Aralen)*
- **Combination of atovaquone and proguanil** *(Malarone)*
- **Halofantrine**
- **Quinine**
- **Combination of sulfadoxine and pyrimethamine** *(Fansidar)*

Which statement best describes the **mechanism of action** of local anesthetics on the nerve axon?

- Decreases sodium uptake through sodium channels of the axon
- Increases potassium outflow from inside to outside the nerve
- Increases the membrane's permeability to sodium
- Increases the excitability of the nerve axon

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- **Decreases sodium uptake through sodium channels of the axon**

When a local anesthetic solution is injected near the nerve, the solution interferes with the uptake of sodium from outside to inside the nerve. The local anesthetic blocks the specific sodium channels thereby blocking the sodium uptake. This decreases the nerve excitability below a critical level and nerve impulses fail to propagate along the axon. Since axons carry pain sensations, these sensations will not be carried and a blockage of pain results.

Local anesthetics **reversibly block** nerve impulse conduction and produce reversible loss of sensation at their administered site.

Local anesthetics have **no effects** on potassium at the nerve axon.

Small, nonmyelinated nerve fibers, which conduct pain and temperature sensations, are affected first, followed by touch, proprioception, and skeletal muscle tone.

Which form of a local anesthetic can **readily penetrate tissue membranes?**

- Ionized form
- Nonionized free-base form
- Both Ionized and nonionized forms

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• **Nonionized free-base form**

Local anesthetic free bases are fat-soluble *(lipophilic)* drugs. They are converted to their water-soluble *(hydrophilic)* hydrochloride salts to allow preparation of an injectable solution. In solution an equilibrium is established between the ionized and the nonionized forms of the local anesthetic. The proportion of the drug in the ionized form depends on the pKa of the drug and the pH of the solution. **At the usual solution pH of 6.0 or lower,** most local anesthetics are almost completely in the ionized form.

This is important because only the **nonionized free-base** form of the drug can readily penetrate tissue membranes. Local anesthesia can only be obtained if sufficient free-base is available. The lower the pKa of the drug and the higher the pH of the solution **or injected tissues**, the **more free-base** will be available.

Once the local anesthetic is injected the buffering capacity and **pH of the tissues** *(normally 7.4)* shifts the equilibrium in favor of free-base formation. At physiologic pH of 7.4 approximately 5-20% of the local anesthetic is in free-base form which is enough to penetrate and cause anesthesia. **If infection or inflammation is present,** the pH of the tissues may be **acidic** and there is a significant reduction in the concentration of the free-base form. In this situation, the local anesthetic may not be effective.

Key point of all this: The potential action of all local anesthetics depends on the ability of the anesthetic salt to **liberate the free-base.**

Note: If you inject lidocaine *(pKa = 7.8)* into tissue that has a pH of 7.8, the lidocaine will exist in an equal mixture of ionized and nonionized forms which will be more than enough to produce anesthesia.

The only **nonbarbiturate sedative-hypnotic agent** that is indicated in the practice of dentistry is:

- Pentobarbital
- Secobarbital
- Chloral hydrate
- Meperidine

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• **Chloral hydrate**

Traditionally chloral hydrate has been used orally in the **preoperative management of the anxious pediatric dental patient.** It has an unpleasant odor and a bitter, caustic taste, which can be partially masked in a flavored syrup. Chloral hydrate is rapidly absorbed after oral administration with an onset of 15 to 30 minutes. Its duration of action is about 4 hours.

For use in children, chloral hydrate is available as a 500 mg / 5 ml solution. The usual dose for a child is 50 mg / kg up to a maximum of 1g. **Important:** Children will often enter a period of **excitement and irritability** before becoming sedated. It affects brain centers that control wakefulness and alertness. Chloral hydrate **does not relieve pain.**

Notes:

1. Chloral hydrate is a prodrug and is metabolized to the active metabolite, trichloroethanol.
2. Chloral hydrate's metabolite *(trichloroethanol)* may displace warfarin from its protein binding sites resulting in an increase in the hypoprothrombinemic response to warfarin.
3. Sedative effects and/or respiratory depression with chloral hydrate may be additive with other CNS depressants; monitor for increased effect; includes ethanol, antidepressants, narcotic analgesics, and benzodiazepines.

Ester-type local anesthetics are metabolized where?

- Liver
- Lungs
- Plasma
- Kidneys

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• **Plasma**

Ester-type local anesthetics all have an ester grouping within their chemical structure. An ester grouping is essentially a bridge or link containing the $-COOCH_2-$ configuration.

Amide-type local anesthetics all have an amide grouping within their chemical structure. An amide grouping is essentially a bridge or link containing the $-CONHCH_2-$ configuration.

Ester-type local anesthetics are **mainly available** as topical anesthetics and include benzocaine, tetracaine, and dibucaine. They are also available as medical anesthetic preparations such as propoxycaine *(Ravocaine)*. Ester-type local anesthetics are no longer available as dental anesthetic injectable preparations because of their relatively high incidence of allergy. Only the amides are presently available as dental injectable local anesthetic agents. These include lidocaine, mepivacaine, prilocaine, bupivacaine, and articaine.

Ester-type local anesthetics are **metabolized** by the plasma enzyme **plasma cholinesterase.** Another name for this enzyme is pseudocholinesterase. This enzyme splits the ester linkage within the chemical structure rendering the anesthetic ineffective. **Procaine** *(Novocaine)* was one of the original ester-type anesthetics. When procaine is metabolized by plasma cholinesterase, a highly allergic compound called **para-aminobenzoic acid** *(PABA for short)* is formed. Many patients developed an allergy to PABA.

Which of the following is a contraindication or precaution to the use of **Prilocaine?**

- Biliary tract disease
- Type II diabetes
- Rheumatoid arthritis
- Hepatic disease

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- **Hepatic disease**

***Remember:** All amides are metabolized **primarily in the liver**, and the metabolites are then renally excreted.

Prilocaine *(Citanest)* is a local anesthetic of the amide class, used for nerve block, epidural and regional anesthesia. It has an intermediate duration of action and is longer acting than Lidocaine. Prilocaine produces less vasodilation than do equal amounts of Lidocaine *(it is somewhat less potent than Lidocaine)*. It is available as a 4% solution with or without epinephrine, which prolongs the anesthetic effect.

Prilocaine is metabolized to orthotoluidine, a product that can produce **methemoglobinemia**, a condition that is characterized by increased levels of methemoglobin in the blood. **Note:** This methemoglobin is less effective than hemoglobin in carrying oxygen in the blood.

Prilocaine is about one-half as toxic as Lidocaine, but since methemoglobinemia is a possible reaction, Prilocaine is **not used** for patients with hypoxic conditions of any kind.

PHARMACOLOGY

Anesth

Which **local anesthetic** has no place in the routine practice of dentistry?

- Articaine
- Cocaine
- Lidocaine
- Bupivacaine
- Prilocaine

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• **Cocaine**

Cocaine is a naturally occurring ester of benzoic acid and was the **first local anesthetic** used in dentistry and medicine. It is potent and extremely toxic, and it is the only local anesthetic that causes **definite vasoconstriction.** Cocaine is commercially available in a variety of forms and is **applied to mucous membranes of the oral, laryngeal, and nasal cavities for use as a topical anesthetic.** Cocaine causes significant euphoria, and abuse can lead to a physical dependence. Despite being an excellent local anesthetic, the risk of abuse and the intense local vasoconstriction it produces prevent cocaine from being more widely used clinically. **Important: Cocaine has no place in the routine practice of dentistry.**

The pharmacology of cocaine is unique among local anesthetics in that the drug **inhibits the uptake of catecholamines** by adrenergic nerve terminals. It therefore potentiates the action of endogenously released and exogenously administered sympathomimetic amines such as dobutamine, dopamine, or epinephrine. It increases the risk of developing cardiac arrhythmias and hypertension. *(i.e., cocaine increases the pressor activity of these sympathomimetic amines).*

Local anesthetics theoretically should be **less effective** in acutely inflamed tissue than in normal tissue because in inflamed tissue what happens?

- The pH rises, thus inactivating the anesthetic
- The pH rises, thus decreasing available free base
- The pH decreases, thus decreasing available free base
- The pH remains the same, the extracellular fluid dilutes the anesthetic

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- **The pH decreases, thus decreasing available free base**

At body pH *(7.4)*, a local anesthetic when infiltrated, will chemically exist as a portion which is ionized *(has a proton attached)* and as a portion which is non-ionized *(has no proton attached)*. **Note:** A proton is nothing more than a hydrogen *(H+)* atom. That portion which is ionized has difficulty penetrating the nerve and will not be effective. That portion which is non-ionized will penetrate the nerve to cause anesthesia. That portion which is non-ionized is also known as the free base. The more proportion of the anesthetic which is in the free base form, the more effective it will be.

When tissue conditions are **normal** *(pH 7.4)*, approximately **10-20%** portion of an infiltrated local anesthetic is in the form of the free base *(non-ionized form)*. This is enough to penetrate the nerve to cause anesthesia. When tissues are **acidic**, as in the case of tissue infection, less free base portion exists and more ionized portion is present. There is not enough free base form to penetrate the nerve to cause anesthesia. Therefore, the local anesthetic when infiltrated to the tissue site is not effective at the normal anesthetic doses.

A dental anesthetic carpule contains 1.8 ml of a 2% solution of lidocaine with 1:100,000 epinephrine. How much lidocaine and epinephrine does the carpule contain?

- 3.6 mg lidocaine and 0.18 mg epinephrine
- 3.6 mg lidocaine and 0.018 mg epinephrine
- 36 mg lidocaine and 0.18 mg epinephrine
- 36 mg lidocaine and 0.018 mg epinephrine

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- **36 mg of lidocaine and 0.018 mg of epinephrine**

Key to question: 1 ml of a 2% solution of lidocaine with 1:100,000 epinephrine contains 20 mg of lidocaine and 0.010 mg of epinephrine.

A dental carpule contains 1.8 ml solution. Therefore, 1.8 ml of 2% solution of lidocaine with 1:100,000 epinephrine contains 36 mg of lidocaine and 0.018 mg epinephrine.

Epinephrine *(vasoconstrictor)* is included in local anesthetics for the following reasons:

- It prolongs the duration of action
- It reduces toxicity
- It reduces the rate of vascular absorption
- It provides a hemostatic effect to reduce bleeding at injection site

The maximal recommended adult dose of **Lidocaine is 300 mg.** How many milliliters of 2% Lidocaine need to be given to reach this level?

- 7.5 milliliters
- 10 milliliters
- 15 milliliters
- 20 milliliters

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- **15 milliliters**

Important information to solve questions of this type:

- There are **20 mg** of Lidocaine in every milliliter of **2%** Lidocaine
- **300 mg** *(maximal recommended dose)* ÷ **20 mg** *(in every mL)* = **15 mLs**
- 20 mg x **1.8 mL** *(in every carpule)* = 36 mg / 1 carpule
- 300 mg ÷ 36 mg = **8.3 carpules** *(8 carpules of 2% Lidocaine can be used)*

Maximum Recommended Doses of Local Anesthetic

Drug	Maximal Recommended Dose		Concentration Used (%)	Approximate Number of Carpules
	mg	ml		
Lidocaine *(Xylocaine)*	300	15	2	8.3
Mepivacaine *(Carbocaine)*	300	10	3	5.6
Prilocaine *(Citanest)*	400	10	4	5.6
Bupivacaine *(Marcaine)*	90	18	0.5	10

The maximum recommended dose of a local anesthetic that can be administered to a **child** < 10 years of age is determined by:

- Age
- Weight
- Height
- Gender

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• **Weight (mg / kg)**

For Lidocaine *(2%)* w/wo epinephrine, a dosage of **4.4 mg / kg should not be exceeded** *(maximum is 300 mg).*

Maximum Recommended Doses of Lidocaine for Children < 10 Years		
Patient Weight (kg / lb)	**Mg**	**No. of Carpules**
10 / 23	44	1.2
15 / 34.5	66	1.8
20 / 46	88	2.4
25 / 57.5	100	2.6

Note: 1 kg = 2.3 lbs

2% = **20 mg / mL x 1.8 mL** / *1 carpule* = **36 mg** / *1 carpule*

Local anesthetics **depress** which of the following nerve fibers **first?**

- **Large** myelinated fibers
- **Small** unmyelinated fibers
- **Small** myelinated fibers
- **Large** unmyelinated fibers

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• **Small unmyelinated fibers** *(these conduct pain and temperature)*

They depress the **large myelinated fibers last**. The small nerves have the greater surface-volume ratio *(this accounts for the rapid onset of action).*

Clinically the general order of loss of function is as follows:

1. Pain
2. Temperature
3. Touch
4. Proprioception
5. Skeletal muscle tone

Note: Pain threshold refers to the lowest level of pain a patient will detect.

Remember: Local anesthetics **reversibly block nerve impulse conduction and produce the reversible loss of sensation** at their administration site. They **do not** produce a loss of consciousness. They appear to become incorporated within the nerve membrane or to bind to specific membrane sodium ion channels, restricting sodium permeability in response to partial depolarization.

PHARMACOLOGY

Anti-Anx

All of the following drugs are considered to be what **type** of drugs?

- Chlordiazepoxide *(Librium)*
- Diazepam *(Valium)*
- Flurazepam *(Dalmane)*
- Midazolam *(Versed)*
- Alprazolam *(Xanax)*
- Lorazepam *(Ativan)*
- Triazolam *(Halcion)*

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• **Tranquilizers *(Benzodiazepines)***

The benzodiazepines are used as oral preparations to alleviate anxiety and to induce sleep, and intravenously to cause conscious-sedation for outpatient surgery. Benzodiazepines appear to produce their calming effects by depressing the limbic system and reticular formation through potentiation of the inhibitor neurotransmitter gamma-amino-butyric acid *(GABA)*, which is found in the brain. **Tolerance** and **physical dependence** can occur with prolonged high dosage. They seem to be much safer than barbiturates. Other useful properties include being an anti-convulsant and a skeletal muscle relaxant.

Adverse effects:

- **CNS** effects – fatigue, slurred speech, drowsiness / sleepiness and confusion *(do not drive a car)*
- **GI** effects – dry mouth, nausea
- **Other** effects – hypotension, muscle relaxation

Benzodiazepines **used orally** as tranquilizers: **chlordiazepoxide** *(Librium)*, **diazepam** *(Valium)*, **alprazolam** *(Xanax)* and **lorazepam** *(Ativan)*. Those used as **hypnotics** to overcome insomnia: **flurazepam** *(Dalmane)* and **triazolam** *(Halcion)*. **Midazolam** *(Versed)* comes as a liquid for pre-operative sedation in children and as injectable for IV conscious sedation.

Note: Benzodiazepines should never be taken with any form of alcohol. Serious potentiation of the sedative effects of each will occur leading to unexpected inebriation and respiratory depression.

The most common adverse effects associated with the **benzodiazepines** include all of the following **except:**

- CNS depression *(drowsiness and sedation)*
- GI disturbances *(nausea, vomiting and diarrhea)*
- Confusion
- Respiratory depression
- Disorientation
- Ataxia

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• **Respiratory depression**

The benzodiazepines have clinically useful antianxiety, sedative-hypnotic, anticonvulsant and skeletal muscle relaxant properties. They exert their main effect on central GABA-nergic neurons. With normal dosing, the benzodiazepines have little effect on respiratory systems in healthy individuals.

The benzodiazepines *(especially diazepam, lorazepam and midazolam)* are important adjuncts in the practice of anesthesiology. They may be used as preoperative sedatives and induction agents, as well as supplemental agents for the maintenance of anesthesia. **Important:** The benzodiazepines, barbiturates and narcotic analgesics **all produce sedation and have the ability to produce dependence.**

For the **treatment of insomnia** due to anxiety flurazepam, temazepam and triazolam are usually prescribed.

Note: The term **tranquilizer** refers to a drug that promotes tranquility by calming, soothing, quieting or pacifying **without sedating or depressant effects.** Antipsychotic agents are considered to be **major tranquilizers** and antianxiety agents *(benzodiazepines)* are considered to be **minor tranquilizers.**

Important: Flumazenil *(Mazicon)*, a benzodiazepine antagonist, may be used to reverse the residual effects of benzodiazepines in the event of an overdose.

Buspirone *(BuSpar)* is classified is a minor tranquilizer which works by what mechanism?

- GABAmimetic
- Diminishes serotonergic activity
- Cholinomimetic
- Diminishes glutamate activity

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- **Diminishes serotonergic activity**

Buspirone is an orally administered anxiolytic *(antianxiety agent)* that is structurally and pharmacologically distinct from all other anxiolytics including the benzodiazepines and barbiturates. Buspirone also differs from other antianxiety agents in that it does not possess anticonvulsant or muscle relaxant properties, **does not** impair psychomotor function and **does not** cause sedation *(lacks CNS depressant activity)* or physical dependence.

Buspirone has a number of special characteristics:

- **Slow onset of action** *(as long as two weeks).*
- **Different mechanism of action** than either the barbiturates or benzodiazepines. Most likely acts as a partial agonist on certain 5-hydroxytryptamine *(serotonin)* receptors, and diminishes serotonergic action.
- **Generally fewer side effects;** less sedation than benzodiazepines.
- **Low abuse potential → Remember:** The benzodiazepines and barbiturates have abuse potential and may cause dependence.
- **It possesses no hypnotic or anticonvulsant properties** *(benzodiazepines and barbiturates do).*

Side effects are limited to restlessness, dizziness, headache, nausea, diarrhea, and paresthesias. However, **tardive dyskinesia** is possible with long-term therapy.

Which of the following side effects are most common with the use of **Diazepam** *(Valium)*?

- Rash, itch
- Mouth, throat ulcers
- Drowsiness, fatigue
- Difficulty with urination

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• **Drowsiness, fatigue**

Diazepam is prescribed in the treatment of anxiety, nervous tension, muscle spasm, and as an anticonvulsant. It is preferred to a barbiturate as an antianxiety because it has less addiction potential and produces less profound CNS depression. **Contraindications to use:** Acute narrow angle glaucoma and psychoses.

It is the agent of choice **to reverse status epilepticus** induced by a local anesthetic overdose. If it is given intravenously *(for status epilepticus)*, **use a large vein to decrease the risk of thrombophlebitis**. Diazepam affects the limbic system of the brain *(controls emotion)*. It has a high therapeutic index, produces some degree of amnesia, **and can be locally irritating to tissue and may produce local thrombophlebitis.** Among the more serious adverse reactions are withdrawal symptoms resulting from discontinuation of treatment. **Note: Propylene glycol,** which is in the IV mixture, is the **main cause of thrombophlebitis.**

PHARMACOLOGY

Cv

Which of the following drugs are considered to be **antiarrhythmic agents?**

- Sodium channel blockers
- Beta-adrenergic blockers
- Potassium channel blockers
- Calcium channel blockers
- All of the above

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• **All of the above**

The **Vaughan-Williams** classification system traditionally has been used to classify antiarrhythmic drugs. This scheme places the available agents into one of four groups, usually denoted by Roman numerals I – IV. This system is loosely based on the channel or receptor involved.

Group I: Sodium channel blockers

- **Group I drugs** are further classified on the basis of their *effects on action potential duration.*
- **Group IA agents** include: **Quinidine**, procainamide, amiodarone and disopyramide. These agents **prolong the action potential.**
- **Group IB agents** include: **Lidocaine**, mexiletine and tocainide. These agents **shorten the action potential.**
- **Group IC agents** include: **Flecainide**, moricizine and propafenone. These agents have **no effect on action potential duration.**

Group II: Beta-adrenergic blockers – **Propranolol** and **esmolol** are the prototype antiarrhythmic beta-blockers

Group III: Potassium channel blockers – Amiodarone is the prototype drug in this class.

Group IV: Calcium channel blockers – **Verapamil** is the prototype. Diltiazem is also included in this group.

Note: Miscellaneous antiarrhythmic drugs include: **adenosine** and **digitalis.**

Which **antiarrhythmic agent** is **effective only on the ventricle** and is often administered IV to treat life-threatening ventricular arrhythmias?

- Quinidine
- Lidocaine
- Disopyramide *(Norpace)*
- Propafenone *(Rythmol)*
- None of the above

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• **Lidocaine**

When lidocaine is **used IV** to treat ventricular arrhythmias, it acts on the fibrillating ventricles to decrease the cardiac excitability and spares the atria. It can effectively reverse a life-threatening situation.

Quinidine is considered as the prototype anti-arrhythmic agent and is used primarily to treat atrial fibrillation. It is not effective in treating life-threatening ventricular fibrillation.

Disopyramide *(Norpace)* is a more recent anti-arrhythmic agent used for the conversion of atrial arrhythmias to normal sinus rhythm.

Propafenone *(Rythmol)* is another more recent drug used to treat both ventricular arrhythmias and supraventricular tachycardias.

True or False

Procainamide is an anti-arrhythmic agent.

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• **True**

Procainamide is a potent Group IA antiarrhythmic agent that is used in the treatment of several cardiac arrhythmias including atrial fibrillation, atrial flutter, paroxysmal atrial tachycardia, and ventricular tachycardia. **It is a derivative of the ester local anesthetic procaine.** Procainamide has properties similar to those of other Group IA agents, **quinidine and disopyramide.** These drugs **decrease myocardial conduction velocity, excitability, and contractility** by inhibiting the influx of sodium through "fast" channels of the myocardial cell membrane, thereby increasing the recovery period after repolarization. **Note: Quinidine** is primarily used to treat **supraventricular tachyarrhythmias.** It is contraindicated in some arrhythmias, particularly those associated with **heart block**.

Remember: Generally speaking, the use of **beta-blockers** *(Group II agents)* as antiarrhythmics is reserved for patients who require only control of ventricular rate during atrial tachyarrhythmias or who have mildly symptomatic ventricular arrhythmias. Side effects include bradycardia and hypotension.

Important: Amiodarone *(Cordarone)* is often considered a Group III agent even though it also blocks sodium channels, a Group I action. It is unique in that it is the most potent and "broad-spectrum" antiarrhythmic compound currently available. **It blocks sodium, calcium, and potassium channels as well as beta receptors. It has impressive efficacy in suppressing both supraventricular and ventricular arrhythmias.**

Verapamil is useful for the treatment of:

- Angina
- Hypertension
- Supraventricular tachyarrhythmias
- All of the above

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• **All of the above**

Verapamil is the prototypical **Group IV** *(calcium channel blockers)* antiarrhythmic agent. It inhibits the intracellular entry of calcium through the slow channels of the calcium-dependent tissues of the myocardium, which are concentrated in the SA and AV nodes. It is the drug of choice for the suppression of **paroxysmal supraventricular tachycardias stemming from the AV node** *(which is characterized by a rapid cardiac rate, usually 160-190 per minute).* Of the calcium channel antagonists available, only **verapamil** and **diltiazem** possess significant antiarrhythmic activity.

Note: Calcium channel blockers *(e.g., verapamil, diltazem and nifedipine)* are useful as **antianginal agents** as well. They block calcium entry through the membranous calcium ion channels of cardiac and vascular smooth muscle. This has **two effects:**

1. Peripheral arterioles dilate *(vasodilators)* and total peripheral resistance decreases, reducing after-load and reducing myocardial oxygen requirements.
2. Increases oxygen delivery to the myocardium.

***They are used to treat chronic stable angina pectoris.

Important: Nitrites *(amyl nitrite)* and nitrates *(nitroglycerin)* are fast-acting antianginal agents that are used to **relieve acute anginal attacks**.

Note: For angina, nitroglycerin and nifedipine are usually used **before verapamil.**

PHARMACOLOGY

Anti-Convul

All the following are classified as **what type of drug?**

- Gabapentin *(Neurontin)*
- Carbamazepine *(Tegretol)*
- Phenytoin *(Dilantin)*
- Diazepam *(Valium)*
- Valproic acid *(Depakene)*

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• **Antiepileptic drugs or anticonvulsants**

Epilepsy is a **neurological disorder** characterized by sudden, recurring attacks of motor, sensory, or psychic malfunction with or without the loss of consciousness or convulsive seizures. The goal of anticonvulsant therapy is to reduce or eliminate these seizures.

Phenytoin *(Dilantin)* is used in the treatment of tonic-clonic *(grand mal)* seizures. **Phenytoin-induced gingival hyperplasia is common** and may partially or totally obscure the crowns of teeth. The rate of development of this condition can be diminished by proper oral hygiene.

Gabapentin is used as an adjunct to treat partial seizures.

Carbamazepine *(Tegretol)* is used as prophylaxis for partial seizures with complex symptomatology including psychomotor and temporal lobe seizures. It is also used to treat tonic-clonic seizures *(grand mal)* and **trigeminal neuralgia**. It rarely causes aplastic anemia.

Diazepam *(Valium)* is used for **status epilepticus** and in emergency treatment of seizures. May cause drowsiness, dizziness, and ataxia.

Valproic acid *(Depakene)* is used to treat patients with complex partial seizures; adjunctively in patients with multiple seizure types including absence seizures. May cause liver failure and blood dyscrasias.

Ethosuximide *(Zarontin)* is effective in treating absence seizures by causing:

- Sodium channel blockade
- Neuronal membrane hyperpolarization
- Calcium channel blockade
- GABA-chloride channel blockade

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- **Calcium channel blockade**

Ethosuximide *(Zarontin)* and **valproic acid** *(Depokote)* are the preferred drugs for treating absence seizures since they cause minimal sedation. **Valproic acid** functions by causing neuronal membrane hyperpolarization. The side effects of these drugs include: GI distress, lethargy, headache, and hepatotoxicity *(specifically with valproic acid).*

Important points to remember:

1. Phenytoin and carbamazepine *(Tegretol)* produce **sodium channel blockade.** Phenytoin is the **most extensively used** of all antiepileptics and is used IV for status epilepticus. **Note**: Carbamazepine is also used in the treatment of pain associated with **trigeminal neuralgia.** Side effects from carbamazepine include diplopia, ataxia, enzyme induction and blood dyscrasias.
2. **Barbiturates** *(Phenobarbital)* prolong the inhibitory effects of GABA.
3. Most of the commonly used anticonvulsants are **CNS depressants. Respiratory depression** may occur with overdosage.

The most widely used **tricyclic antidepressant** drug is:

- Imipramine *(Tofranil)*
- Amitriptyline *(Elavil)*
- Desipramine *(Norpramin)*
- Nortriptyline *(Aventyl)*
- Clomipramine *(Anafranil)*
- Doxepin *(Sinequan)*
- Protriptyline *(Vivactil)*

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• **Amitriptyline** ***(Elavil)***

The **tricyclic antidepressants** are generally considered to be the drugs of first choice for the **treatment of unipolar disorder** *(depression)*. These drugs **inhibit the neuronal re-uptake of norepinephrine and serotonin** *(5-HT)* in the brain. This results in the potentiation of their neurotransmitter actions at postsynaptic receptors.

Important: Amitriptyline displays the **greatest anticholinergics effects**, desipramine the least.

Drowsiness is the most frequent CNS adverse reaction. **Anticholinergic** adverse side effects include: dry mouth, constipation, blurred vision and tachycardia.

The selective serotonin reuptake inhibitors have revolutionized the treatment of depression. The most important clinical distinction of the SSRIs from all other antidepressants is their very high specificity for **blocking the reuptake of serotonin. Fluoxetine** *(Prozac)* is the prototype and has the longest half-life. **Paroxetine** *(Paxil)*, **Sertraline** *(Zoloft)* and **Fluvoxamine** *(Luvox)* have shorter half-lives and can be given once daily. These drugs are also effective for treating **panic attacks**. Side effects include nausea, headaches, anxiety, agitation, insomnia, and sexual dysfunction.

Which drug is the current drug of choice for the treatment of the **manic phase** of bipolar disorder *(or manic-depressive syndrome)*?

- Phenobarbital
- Imipramine
- Lithium
- Haloperidol

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• **Lithium**

Bipolar disorder *(or manic-depressive syndrome)* is characterized by cyclical changes in affective state between the **manic** and **depressive phases** of behavior. Bipolar patients cycle between the two affected states.

Lithium is **used primarily** to suppress recurrences of the **manic phase** of bipolar disorder. It is not useful for the acute manic episodes. Lithium can prevent the occurrence of both the depressive as well as the manic episodes in some but not all the patients. Antidepressants are often administered with lithium to manage the depressive phase of the illness if lithium alone is not sufficient.

Neuroleptic agents *(also referred to as anti-psychotic agents or major tranquilizers)* are used in the **acute manic** episodes. Chlorpromazine, which is a phenothiazine, and Haloperidol, which is not a phenothiazine but acts in a similar fashion, are **effective** in quelling the extreme mania and psychotic behavior.

Which drugs are **Monoamine Oxidase** *(MAO)* **inhibitors?**

- Isocarboxazid *(Marplan)*
- Tranylcypromine *(Parnate)*
- Phenelzine *(Nardil)*
- All of the above

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• **All of the above**

MAO inhibitors are used in the treatment of depression. MAO inhibitors antagonize the action of **monoamine oxidase** *(MAO)* responsible for the degradation of the naturally occurring monoamines – epinephrine, norepinephrine, dopamine, and serotonin. It is theorized that the increased level of monoamines in the brain is responsible for the anti-depressant effect of the MAO inhibitors.

Note: Local anesthetics containing epinephrine are generally contraindicated in patients who are taking MAO inhibitors.

The major limitation for the widespread use of the MAO inhibitors for the treatment of depression has been the potential for serious side effects. MAO inhibitors interact with many drugs and with foods containing large amounts of the amino acid tyramine. Among the drugs that interact with MAO inhibitors are meperidine *(Demerol)*, epinephrine and ephedrine.

PHARMACOLOGY

Anti-Hist

All of the following are antihistamine **H_1 blockers except:**

- Diphenhydramine *(Benadryl)*
- Chlorpheniramine *(Chlor-Trimeton)*
- Cimetidine *(Tagamet)*
- Loratidine *(Claritin)*
- Desloratidine *(Clarinex)*

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• **Cimetidine *(Tagamet)***

***Cimetidine is an antihistamine **H_2-blocker.**

Antihistamines are antagonizing agents that compete for receptor sites with natural histamine, which is stored preformed in cytoplasmic granules of tissue mast cells and blood basophils. It is **released in response to IgE-mediated** *(immediate)* **allergic reactions** and plays an important role in hay fever, urticaria and angioneurotic edema. **Note:** Histamine also plays an important role in the control of acid secretion *(HCL)* **in the stomach.**

There are **two types** of histamine receptors, **H_1-receptors**, which play an extremely important role in allergic reactions and **H_2-receptors**, which are important in gastric acid secretion. The antihistamines are divided into H_1 and H_2-blockers depending on the histamine receptor they compete for.

H_1 blockers include:

1. **First-generation** *(classical)* **agents:**
   • Diphenhydramine, chlorpheniramine, tripelennamine *(PBZ)*.

***These agents have a **broad spectrum of action** which includes antihistaminic, anticholinergic, antiserotonergic, antibradykinin and **sedative** properties.

2. **Second-generation agents:**
   • Cetirizine *(Zyrtec)*, fexofenadine *(Allegra)*, loratadine *(Claritin)* and desloratidine *(Clarinex)*.

*** Most of these agents, because of their poor CNS penetration, **cause less sedation and drowsiness** than the first-generation agents.

Important points: All of the H_1-blockers **do not prevent** the release of histamine but rather compete with free histamine for binding at H_1-receptor sites. In general, the binding is competitive, however, some second-generation agents bind non-competitively at higher doses. **Common side effects** include drowsiness, dizziness, anticholinergic effects *(dry mouth, nose and throat)*, and nausea. They can **both stimulate and depress the CNS.**

For which of the following conditions can **Ranitidine** be used?

- Prostatitis
- GERD *(heartburn)*
- Toxic-shock syndrome
- Renal failure

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• **GERD** ***(heartburn)***

H_2-receptor antagonists compete with histamine at the H_2-receptor. Histamine produces a wide variety of physiologic actions in many tissues. While H_2-receptors are located in the GI tract and in vascular and bronchial smooth muscle, H_2-receptor antagonists **compete with histamine only in the GI tract.** Inhibition of histamine at the parietal cell interferes with one of several mediators for signaling the parietal cell to secrete acid.

H_2-blockers include: cimetidine *(Tagamet)*, ranitidine *(Zantac)*, famotidine *(Pepcid)* and nizatidine *(Axid)*. These agents are all **reversible competitive antagonists** of the actions of histamine on H_2-receptors. They are orally active and because they are relatively non-toxic, they can be given in large doses, **so that the duration of action of a single dose may be 12-24 hours.** These agents are used to treat acid-peptic disease, especially **duodenal ulcers** and occasionally gastric peptic ulcers. These drugs are also used to treat **Zollinger-Ellison syndrome** *(a hypersecretory disease)* and **gastroesophageal reflux disease** *(GERD)* however, for both of these disorders **omeprazole** *(Prilosec)* which is a "proton-pump" inhibitor is more effective.

Note: Cimetidine is more likely than the other agents to provoke interactions with hepatically metabolized drugs *(it is a potent inhibitor of hepatic drug-metabolizing enzymes)* and also may cause gynecomastia. The other agents *(ranitidine, famotidine and nizatidine)* do not produce these side effects.

Diphenhydramine *(Benadryl)*, chlorpheniramine *(Chlor-Trimeton)*, loratidine *(Claritin)*, and desloratidine *(Clarinex)* are antihistamines at which histamine receptor site listed below?

- H_1-receptor site
- H_2-receptor site

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• **H_1-receptor site**

Antihistamines are **antagonizing agents** that compete for receptor sites with natural histamine. The two types of histamine antagonists are:

1. **H_1-receptor antagonists** → competitively block H_1-receptors blocking the effects of histamine at these receptors. They block the vasodilation, they block the constriction of the bronchi and they block capillary permeability which histamine ordinarily causes. The blockade of these effects of histamine overcomes the symptoms of seasonal allergies.

2. **H_2-receptor antagonists** → competitively block H_2-receptors, blocking the effects of histamine at these receptors. Examples of these drugs include: cimetidine *(Tagamet)*, ranitidine *(Zantac)*, and famotidine *(Pepcid)*. They block secretion of stomach acid and are used in the treatment of **duodenal ulcers**.

Note: Histamine is found in **all tissues**, particularly in mast cells and blood basophils. It is released in allergic and inflammatory reactions.

Antibiotic regimens for patients with **prosthetic implants** include which of the following drugs as standard therapy of first choice in patients not allergic to penicillin?

- Cephalexin
- Cephradine
- Amoxicillin
- All of the above

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- **All of the above**

From the "Advisory Statement, Antibiotic Prophylaxis for Dental Patients with Total Joint Replacement" published by the American Dental Association and the American Academy of Orthopedic Surgeons, in the J Amer Dent Assoc 1997; 128(7):1004-8, either cephalexin, or cephradine or amoxicillin can be used as the drugs of choice for standard prophylaxis medication in the patient with a total joint replacement.

- **Cephalexin** *(Keflex)* is a 1st generation cephalosporin; prophylaxis dose is 2 grams orally 1 hour prior to the dental procedure.

- **Cephradine** *(Velosef)* is a 1st generation cephalosporin; prophylaxis dose is 2 grams orally 1 hour prior to the dental procedure.

- **Amoxicillin** is a member of the penicillin family; prophylaxis dose is 2 grams orally 1 hour prior to the dental procedure.

PHARMACOLOGY

Clindamycin has which mode of action?

- Affects cell membrane
- Interferes with protein synthesis
- Affects cell wall
- Interferes with normal biosynthetic pathways

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- **Interferes with protein synthesis**

Mechanisms of action of antibiotics:

- **Agents affecting cell wall:**
  - Penicillins
  - Cephalosporins
  - Bacitracin
  - Vancomycin
  - Cycloserine
  - Aztreonam
  - Imipenem
- **Agents interfering with protein synthesis:**
  - Tetracycline
  - Aminoglycosides
  - Chloramphenicol
  - Erythromycin
  - Lincomycin
  - Clindamycin
  - Azithromycin
  - Clarithromycin
- **Agents interfering with normal biosynthetic pathways:**
  - Sulfonamides
  - Trimethoprim
  - Fluoroquinolones

Note: The most common clinical cause of bacterial resistance is the use of antibiotics when they are not indicated.

Which of the following antibiotics is considered a **broad-spectrum** antibiotic?

- Penicillin VK
- Cefaclor *(Ceclor)*
- Penicillin G
- Erythromycin

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• **Cefaclor *(Ceclor)***

Cefaclor is a member of the **cephalosporin** family of antibiotics. The cephalosporins are penicillin-like in action against bacteria. They are **bactericidal** antibiotics and act like the penicillins in that they **affect the bacterial cell wall** during cell division such that closure does not occur. Bacteria eventually lyse, resulting in death of the cell. Cephalosporins act against a wide range of gram-positive and gram-negative bacteria.

Currently there are **four generations** of cephalosporins. Progression from first through the fourth generation is associated with a broadening of action against more gram-negative bacteria and a decreased activity against gram-positive bacteria.

Important cephalosporins within each generation:
- **First** → cephalexin *(Keflex)*, cephradine *(Velosef)*, cefadroxil *(Duricef)*, cefazolin *(Ancef)*
- **Second** → cefaclor *(Ceclor)*, cefuroxime *(Ceftin)*, cefoxitin *(Mefoxin)*
- **Third** → cefixime *(Suprax)*, cefoperazone *(Cefobid)*
- **Fourth** → cefepime *(Maxipime)*

Note: Approximately 10% of individuals expressing allergy to the penicillin family of antibiotics will have **cross allergenicity** to the cephalosporins.

Tetracyclines are the drugs of first choice in the treatment of:

- Mycoplasma pneumonia
- Chlamydia infections
- Rickettsial infections
- Vibrio infections
- All of the above

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- **All of the above**

Popular Tetracyclines include:

- **Tetracycline** → used to treat acne, gonorrhea and syphilis in patients allergic to penicillin, exacerbations of chronic bronchitis, **Mycoplasma** infections and **Chlamydia** infections, and **Rickettsia** infections.
- **Minocycline** *(Minocin)* → used to treat acne, anthrax and meningococcal prophylaxis.
- **Doxycycline** *(Vibramycin)* → used to treat infections caused by Rickettsia, Chlamydia and Mycoplasma; alternative to mefloquine for malaria prophylaxis and treatment of syphilis.

Remember: The usefulness of the tetracyclines in the treatment of oral infections is limited. They are, however, **good alternatives to penicillin** for patients with ANUG *(acute necrotizing ulcerative gingivitis)* who require antibiotics.

True or False

Azithromycin *(Z-Pak, Zithromax)* is an antibiotic of the erythromycin *(macrolide)* class of antibiotics.

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• **True**

Azithromycin *(Z-Pak, Zithromax)* and **clarithromycin** *(Biaxin)* are members of the macrolide class of antibiotics in which erythromycin is the prototype agent.

The bacterial spectrums of activity of azithromycin and clarithromycin are similar to that of erythromycin but possess greater intrinsic activity against **H. influenzae.** These two macrolides concentrate within macrophages, making them useful against organisms that are taken up by macrophages such as **Mycobacterium avium intracellulare.**

The significant tissue penetration of both agents and the prolonged elimination half-life of azithromycin *(11-14 hours)* allows for once-daily dosing for azithromycin and twice-daily dosing for clarithromycin.

Note: Both agents appear to cause fewer GI tract adverse reactions and fewer drug interactions than erythromycin.

Erythromycin is well known to cause adverse:

- CNS effects
- GI effects
- Hematologic effects
- Renal effects

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• **GI effects**

Adverse GI effects are reported for approximately 21% of patients receiving erythromycin, about 10% of patients receiving clarithromycin, and less than 5% for azithromycin. A less well-known but nonetheless significant adverse reaction to erythromycin, especially after IV administration, is ototoxicity, manifested as tinnitus and/or deafness.

In general, oral bioavailability of erythromycin is poor. It is readily inactivated by stomach acid, and several salts have been developed to overcome this drawback.

- Erythromycin stearate *(Erythrocin)*
- Erythromycin estolate *(Ilosone)* has the best oral absorption but has been implicated in liver toxicity

***Since they are destroyed by stomach acid, erythromycins are usually **enteric coated.** This is a term designating a special coating applied to tablets or capsules which prevents their release and absorption of their contents until they reach the intestines.

Note: Erythromycin is metabolized in the liver, excretion is mainly via the bile.

Which of the following is a contraindication or precaution to the use of **Tobramycin?**

- Sunlight *(UV)* exposure
- Scleroderma
- Myasthenia gravis
- Syphilis

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• **Myasthenia gravis**

***Aminoglycosides** may cause severe neuromuscular weakness lasting hours to days because of their potential curare-like effect. Aminoglycosides may aggravate muscle weakness in patients with muscular disorders such as myasthenia gravis, infant botulism, or parkinsonism.

Aminoglycosides are bactericidal and have a broad antibacterial spectrum. Their use is primarily in the treatment of **aerobic gram-negative infections**. They have little action against gram-positive anaerobic or facultative bacteria. They bind irreversibly to the 30 S ribosomal subunit of bacteria, subsequently **inhibiting protein synthesis.** Aminoglycosides are usually reserved for the treatment of more **serious bacterial infections** because they have two well-known adverse effects, **ototoxicity and nephrotoxicity.** These agents are poorly absorbed after oral administration and so must be administered by IM or IV injection for a systemic effect. They are rapidly excreted by the normal kidney.

Aminoglycosides:

- **Gentamicin** *(Garamycin)*, **Amikacin** *(Amiken)*, **Tobramycin** *(Nebcin)*, and **Netilmicin** *(Netromycin)*: These are effective against serious infections caused by aerobic gram-negative bacteria, including E. Coli, Enterobacter, Klebsiella, Proteus, **Pseuodomonas aeruginosa,** and Serratia.
- **Streptomycin**: The first aminoglycoside and was shown to be effective in the treatment of tuberculosis. Is seldom used today.
- **Neomycin** *(Mycifradin)* and **Kanamycin** *(Kantrex):* Due to its toxic potential neomycin is used only **topically or locally,** e.g., in the GI tract. Kanamycin is rarely used because of its marked tendency to cause ototoxicity.
- **Spectinomycin**: Is sometimes used to treat gonorrhea.

Sulfonamides *(also known as sulfa drugs)* are structurally similar to:

- Penicillins
- Erythromycins
- Acetylsalicyclic acid *(Aspirin)*
- Para-aminobenzoic acid *(PABA)*

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• **Para-aminobenzoic acid *(PABA)***

Sulfonamides are often referred to as "sulfa drugs" because their molecules contain **sulfur** atoms. They have a different antibacterial mechanism from that of the antibiotics. The sulfonamides are structurally similar to PABA and this similarity is the basis for their antibacterial actions. PABA is needed by bacteria for the synthesis of folic acid. In turn, folic acid is needed for the synthesis of cellular components within the bacteria to allow for cell growth. Because of structural similarities between sulfonamides and PABA, the sulfonamides compete with PABA and are able to **inhibit the actions of PABA**. With PABA inhibited, folic acid is not synthesized within the bacteria, and bacterial cellular growth is inhibited. Sulfonamides are predominantly **bacteriostatic** agents.

Notes:

1. Sulfonamides are **not used** for treatment of dental infections because of a low degree of effectiveness against oral pathogens.
2. Sulfonamides are **used** in medicine primarily for the treatment of urinary tract infections.
3. **Bactrim** is the brand name for the combination of **trimethoprim and sulfamethoxazole.**It is considered the drug of choice for many urinary tract infections.
4. The trimethoprim component is an antimicrobial and the sulfamethoxazole is one of the sulfonamides.

Which antibiotic is not only effective against most staphylococci, aerobic and anaerobic streptococci, but is **most effective** in treating infections due to bacteroides species?

- Penicillin VK
- Erythromycin
- Tetracycline
- Cephalexin *(Keflex)*
- Clindamycin

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- **Clindamycin**

Clindamycin binds to the 50S ribosomal subunit, blocking bacterial protein synthesis. Its use is restricted by its side effects such as severe diarrhea and pseudomembranous colitis. These side effects are caused by the overgrowth of the bacterium known as **Clostridium difficile.**

Clindamycin is **bacteriostatic** and is active against most gram-positive and many anaerobic organisms, including the anaerobic gram-negative bacteria **Bacteroides fragilis.**

In dentistry, clindamycin is an **alternate antibiotic** in the following situations:

- When amoxicillin cannot be used for the standard regimen for prevention of bacterial endocarditis in patients undergoing dental procedures.
- For treatment of common oral-facial infections caused by aerobic gram-positive cocci and susceptible anaerobes.
- For prophylaxis for dental patients with total joint replacement.

Clindamycin can be given to patients allergic to penicillins since there is **no cross allergenicity** between penicillins and clindamycin.

Which antibiotic may cause **bone marrow disturbances** *(aplastic anemia)* and has limited use due to the side effects?

- Penicillin
- Tetracycline
- Chloramphenicol
- Doxycycline

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• **Chloramphenicol**

Chloramphenicol *(brand name Chloromycetin)* is a **broad-spectrum** antibiotic effective against gram-positive and gram-negative bacteria and against anaerobes. It is used as a second or third line drug in medicine to treat serious infections due to organisms resistant to other less toxic antibiotics.

Chloramphenicol is a **bacteriostatic** antibiotic. It reversibly binds to 50S ribosomal subunits of susceptible organisms preventing amino acids from being transferred to growing peptide chains thus **inhibiting** bacterial protein synthesis.

The three **major toxicities** associated with chloramphenicol include:

1. **Aplastic anemia**
2. **Bone marrow suppression** *(anemia and neutropenia may occur during the first week of therapy)*
3. **Gray syndrome** which is characterized by circulatory collapse, cyanosis, acidosis, coma and death.

Nitazoxanide *(Alinia)* is an oral antiprotozoal agent used to treat which of the following conditions?

- Leprosy
- Malaria
- AIDS
- Diarrhea caused by Clostridium difficile
- Diarrhea caused by Giardia lamblia

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- **Diarrhea caused by Giardia lamblia**

***This type of diarrhea is an intestinal infection also known as Giardiasis, and is the most common protozoan infection in the United States.

Antiprotozoal Agents:

- **Nitazoxanide** *(Alinia)* - is an antiprotozoal agent which is used in the treatment of Giardia, and those protozoal infections caused by Cryptosporidium parvum. Its mechanism is to interfere with the electron transfer reaction within the protozoa essential to anaerobic metabolism.
- **Atovaquone** *(Mepron)* - is an antiprotozoal agent used to treat Pneumocystitis carinii pneumonia *(PCP)* in patients who are intolerant to co-trimoxazole.
- **Eflornithine** *(Vaniqa)* - has orphan drug status for the treatment of meningoencephalitic stage of Trypanosoma brucei gambiense infection *(sleeping sickness)*. This drug is also indicated to be used as a cream to reduce unwanted hair from face and adjacent areas under the chin.
- **Furazolidone** *(Furoxone)* - is a antiprotozoal agent used to treat diarrhea caused by susceptible Giardias lamblia and Vibrio cholerae.
- **Metronidazole** *(Flagyl)* - is a synthetic antibacterial and antiprotozoal agent that is effective against Trichomonas vaginalis, which causes trichomoniasis. In addition, it is one of the most effective drugs available against anaerobic bacterial infections.

PHARMACOLOGY

Anti-Infect

All of the following drugs are useful for **treating** what disease?

- Isoniazid
- Streptomycin
- Rifampin
- Ethambutol
- Pyrazinamide

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- **Tuberculosis**

Tuberculosis is a bacterial infection caused by **Mycobacterium tuberculosis**. The anti-tubercular drugs either inhibit the growth of the bacteria or kill the bacteria. Because the Mycobacterium organism tends to develop resistance to any single antitubercular drug, combination drug therapy is standard in the treatment of tuberculosis.

Antitubercular drugs:

- **Isoniazid** → often given in a four drug regimen along with rifampin, pyrazinamide and ethambutol
- **Streptomycin** → often given in combination with isoniazid
- **Rifampin** → usually given in combination with other agents
- **Ethambutol** → usually given in combination with other agents
- **Pyrazinamide** → popular in combination with rifampin

The antiviral agent penciclovir *(Denavir)* is active against which virus?

- Herpes zoster
- Genital herpes
- Herpes simplex type 1 *(HSV-1)*
- Papilloma virus

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- **Herpes simplex type 1** ***(HSV-1)***

Penciclovir *(Denavir)* is a cream formulation indicated for the treatment of **recurrent herpes labialis** *(cold sores)* in adults. This condition is caused by the herpes simplex type 1 virus. Penciclovir is not available for systemic dosing.

Penciclovir *(Denavir)* inhibits viral action by selectively **inhibiting herpes viral DNA synthesis** and therefore resulting in the inhibition of viral replication.

Other agents **indicated for use** in treating the condition of herpes labialis are:

- Acyclovir tablets
- Acyclovir cream
- Docosanol cream *(Abreva)*
- Lysine tablets
- Valacyclovir *(Valtrex)*

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PHARMACOLOGY

Anti-Infect

The following drugs are used **to treat** what viral disease?

- Didanosine
- Zidovudine
- Ritonavir
- Indinavir
- Delavirdine

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• **AIDS** *(acquired immunodeficiency syndrome)*

AIDS has been recognized since 1981 as a unique clinical syndrome brought on by infection with the human immunodeficiency virus 1 *(HIV-1)* or virus 2 *(HIV-2)*. The major cellular defect caused by infection with HIV is a depletion of T-cells, primarily the sub-type T-helper cells known as CD4 cells. This results in a compromised immune system, which becomes susceptible to opportunistic infections.

HIV is a type of **retrovirus** that is responsible for the fatal illness from AIDS. A retrovirus has RNA as its nucleic acid and uses the enzyme **reverse transcriptase** to copy its genome into the DNA of the host's cells chromosomes. This DNA segment is then permanently incorporated into the host cell's DNA within the nucleus. The integrated DNA segment can produce new RNA in the cytoplasm of the host cell. The new RNA in turn synthesizes viral proteins, which are eventually passed on to other host cells such as the immune system macrophages. Ultimately enough of the human immune cells are compromised such that immune function is lost.

The **HIV drugs** such as didanosine *(brand name Videx)*, zidovudine *(brand name Retrovir)*, ritonavir *(brand name Norvir)*, indinavir *(brand name Crixivan)* work by inhibiting certain steps in the HIV infection process within the target cells to halt the destruction of the immune system by the HIV retrovirus.

Which of the following is classified as an **antifungal** agent?

- Bacitracin
- Amphotericin-B
- Polymyxin-B
- Neomycin

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• **Amphotericin-B**

This is an antifungal agent given intravenously or orally for the treatment of severe systemic fungal infections caused by fungi such as **Candida** species. Bacitracin, polymyxin-B and neomycin are not antifungal agents. These are antibiotics effective against susceptible bacteria. Antibiotics in general do not have antifungal properties.

Summary of Some Antifungal Agents			
Topical Agents (dissolve & swallow)	**Use**	**Mechanism on Fungal Cell**	**Form**
Clotrimazole *(Mycelex Troche)*	- Oropharyngeal Candidiasis	- Alters cell membrane	Troche
Nystatin *(Mycostatin)*	- Oral Cavity Candidiasis	- Alters cell membrane	Oral susp.
Topical Agents ***(cream / ointment)***	**Use**	**Mechanism on Fungal Cell**	**Form**
Amphotericin-B *(Fungizone)*	Cutaneous & Mucocutaneous Candidiasis	- Alters cell membrane	Cream
Ketoconazole *(Nizoral)*		- Alters cell membrane	Cream
Nystatin *(Mycostatin)*		- Alters cell membrane	Ointment
Systemic Agents	**Use**	**Mechanism on Fungal Cell**	**Form**
Amphotericin-B *(Fungizone)*	Systemic Candida	- Alters cell membrane	IV injection
Ketoconazole *(Nizoral)*	Oral, Esophageal, Oropharyngeal Candidiasis	- Alters cell membrane	Tablets
Fluconazole *(Diflucan)*		- Alters cell membrane	Tablets

Note: Systemic amphotericin-B is associated with high incidence of kidney toxicity.

Which is the **drug of choice** for treating Candidiasis?

- Penicillin
- Erythromycin
- Nystatin
- Chloramphenicol

Candidiasis is an infection, usually of the oral cavity or vagina, with a candida species, usually **C. albicans**, which causes an inflammatory, pruritic infection characterized by a thick, white discharge. It is common, especially in patients who have a deficiency in T-lymphocytes, or who are receiving chemotherapy, and in immunosupressed individuals *(AIDS patients)*. This yeast-like fungi is **a normal inhabitant** of the oral cavity and vaginal tract, however it is normally held in check by the indigenous bacteria of these areas. **Note:** Angular cheilitis *(bilateral ulcers at the corner of the mouth)* has been linked to C. albicans.

Remember: Nystatin and clotrimazole are the two antifungals that are used as **"swish and swallow"** to treat oral candida infections. Nystatin *(Mycostatin)* is taken as an oral suspension to be swished around the mouth and swallowed. **Clotrimazole** *(Mycelex)* is taken as a troche *(lozenge)* which is slowly dissolved in the mouth and swallowed. They work by binding to sterols in the fungal cell membrane, increasing permeability and permitting the leakage of intracellular components. This leads to the death of the affected fungal cell.

The prototype **penicillin** is:

- Penicillin V
- Penicillin G
- Ampicillin
- Amoxicillin

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• **Penicillin G**

All penicillins are **derivatives of 6-aminopenicillanic acid** and contain a **beta-lactam ring structure** joined to a thiazolidine ring. The beta-lactam ring is **essential for its antibacterial activity.** This basic structure is synthesized by the penicillium mold from two amino acids, L-cysteine and L-valine. Antibiotics containing this beta-lactam ring are referred to collectively as **beta-lactam antibiotics** and include the **penicillins, cephalosporins**, and the two newer groups of agents, the **carbapenems** and the **monobactams.**

Penicillin G *(benzylpenicillin)* is the prototype for comparison. By side chain substitutions *(specifically, this means substituting other groups at the R position of the penicillin molecule)* of the basic 6-aminopenicillanic acid molecule, the **semi-synthetic penicillins** are produced which are more **acid stable,** have a **broader spectrum**, or are **penicillinase resistant.**

Other Naturally occurring penicillins include:

- **Penicillin VK** *(Pen Vee K, V-cillin K)* – preferred for **treating oral infections** because it is more acid stable *(more reliable oral absorption).*
- **Penicillin G procaine** *(Cysticillin)* – always given by **IM route**
- **Penicillin G benzathine** *(Bicillin L-A)* – always given by **IM route.** It is used for the **treatment of syphilis and prevention of rheumatic fever.** It has a longer duration of action *(compared to procaine pen.).*

Remember:

1. Penicillins are **bactericidal**; they inhibit cell wall synthesis.
2. **Probenecid increases** blood levels of **natural penicillins** and may be given concurrently for this purpose.

Which penicillins are **degraded in stomach acid** and therefore must be used parenterally *(by injection)*?

- Penicillin VK
- Methicillin
- Amoxicillin
- Penicillin G
- Carbenicillin

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- **Methicillin** }
- **Penicillin G** } **degraded by stomach acid**
- **Carbenicillin** }

Acid stable penicillins *(may be used orally)* include Penicillin VK, Amoxicillin, Ampicillin, Nafcillin, Oxacillin, Cloxacillin, and Dicloxacillin.

Extended spectrum penicillins include the aminopenicillins *(Ampicillin and Amoxicillin)*.

Broad spectrum penicillins include Carbenicillin, Piperacillin and Ticarcillin. These three penicillins have the widest spectrum of all the penicillins.

Penicillinase-resistant penicillins include Methicillin, Nafcillin, Oxacillin, Cloxacillin, and Dicloxacillin.

Note: These antibiotics are **highly effective** against penicillinase-producing Staphylococcus aureus, but are much less active against other gram-positive bacteria, and they have almost no activity against gram-negative bacteria.

The antibiotic of choice for standard **prophylactic** regimen of **antibiotic coverage** for the prevention of bacterial endocarditis is:

- Penicillin VK
- Tetracycline
- Erythromycin
- Amoxicillin

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• **Amoxicillin**

Current American Heart Association Guidelines

Published June 11, 1997, JAMA Vol 277, pp 1794-1801

Premedication requirements for patients with valvular heart disease or congenital cardiac defects. **If in doubt,** have patient consult their physician as to need.

Standard Regimen

- **Amoxicillin**
  - Adults: 2.0 g orally 1 hour prior to appointment
  - Children: 50 mg/kg *(not to exceed adult dose)* orally 1 hour prior to appointment

Allergy to Amoxicillin:

Note: Only if allergy is not of anaphylactic type. Any one of the following can be used.

- **Clindamycin**
  - Adults: 600 mg orally 1 hour prior to appointment
  - Children: 20 mg/kg orally 1 hour prior to appointment
- **Cephalexin**
  - Adults: 2.0 g orally 1 hour prior to appointment
  - Children: 50 mg/kg orally 1 hour prior to appointment
- **Cefadroxil**
  - Adults: 2.0 g orally 1 hour prior to appointment
  - Children: 50 mg/kg orally 1 hour prior to appointment
- **Azithromycin**
  - Adults: 500 mg orally 1 hour prior to appointment
  - Children: 15 mg/kg orally 1 hour prior to appointment
- **Clarithromycin**
  - Adults: 500 mg orally 1 hour prior to appointment
  - Children: 15 mg/kg 1 hour prior to appointment

Which is the **most preferable antibiotic** for the treatment of non-penicillinase-producing gram-positive staphylococcal infections?

- Tetracycline
- Clindamycin
- Ampicillin
- Cefaclor *(Celcor)*
- Penicillin VK

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• **Penicillin VK**

It is prudent to use an antibiotic with narrow spectrum of action and one that is bactericidal in order to minimize the development of bacterial resistance. Penicillin VK has these properties. Ampicillin has a broader spectrum of action than penicillin VK and cefaclor is a broad spectrum cephalosporin. Tetracycline and clindamycin are bacteriostatic antibiotics and not bacteriocidal.

Note: The major **disadvantage** of the penicillins is their rather high incidence of allergic reactions. Approximately 10% of the general population is allergic to penicillins. This incidence probably holds for any of the specific penicillins since there is cross allergy from one to the other. Skin rash *(a delayed reaction)* is the most prevalent allergic manifestation. Life threatening anaphylaxis can occur, but is very rare, particularly with oral dosing. In **non-allergic** individuals, penicillins at normal therapeutic doses have virtually no side effects what so ever. Penicillins are **bactericidal**, that is, they actually cause death of the invading bacteria.

Which penicillin has the **widest spectrum** of antibacterial activity?

- Penicillin VK
- Dicloxacillin
- Amoxicillin
- Carbenicillin

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• **Carbenicillin**

Members of the penicillin family having the **broadest** spectrums of action are carbenicllin, ticarcillin, and piperacillin. In addition to being active against gram-positive cocci *(streptococci, staphylococci, pneumococci),* and gram-positive rods *(bacillus and others)*, these agents are active against Pseudomonas, Proteus, Klebsiella, and Bacteroides.

These antibiotics are recommended for the treatment of **urinary tract infections** and other infections caused by susceptible gram-negative Pseudomonas species and Proteus species.

Penicillin VK has a relative **limited spectrum** of action against aerobic gram-positive cocci and anaerobes.

Dicloxacillin has a similar spectrum as penicillin VK **but is active** against penicillinase-producing staphylococcus.

Amoxicillin is an aminopenicillin which has an **extended spectrum of action** which includes not only aerobic gram-positive cocci and anaerobes, but some gram-negative bacilli such as Hemophilus, Proteus and Salmonella. Ampicillin also falls into the category of an aminopenicillin. **None of the penicillins are active** against viruses, fungi, rickettsiae or other nonbacterial organisms.

Which of the following has a clinically significant drug interaction with **Amoxicillin?**

- Triazolam *(Halcion)*
- Methotrexate
- Calcitrol
- Candesartan

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• **Methotrexate**

***Amoxicillin in large doses inhibits the renal tubular secretion of methotrexate, thereby causing higher, prolonged serum levels of methotrexate.

Aminopenicillins *(ampicillin, amoxicillin and bacampicillin)* are characterized by the amino substitution of penicillin G. They are able to penetrate **gram-negative bacteria** more readily than are the natural penicillins or the penicillinase-resistant penicillins. **However**, the aminopenicillins are **not stable to beta-lactamases** *(penicillinase)* of either gram-positive or gram-negative bacteria.

Note: Gram-negative bacteria that are susceptible to aminopenicillins include: H.influenzae, some E.Coli and Proteus mirabilis.

Ampicillin *(Polycillin, Onipen)* and **Amoxicillin** *(Amoxil, Larotid)* are used primarily to treat infections such as otitis media, bronchitis, sinusitis, and acute bacterial cystitis caused by susceptible organisms. *Compared with ampicillin*, **amoxicillin has** a higher oral absorption, higher serum levels, a longer half-life, and is less likely to cause adverse GI effects *(diarrhea)*. Amoxicillin is given orally; ampicillin can be given orally or IV. **Note:** Ampicillin and amoxicillin are preferred agents in the treatment of urinary tract infections caused by susceptible **enterococci** *(streptococci that inhabit the intestine)*.

Bacampicillin *(Spectrobid)* is used to treat upper and lower respiratory tract infections, urinary tract infections, skin and skin structure infections. It is hydrolyzed to ampicillin during absorption from the GI tract; it has a better absorption than ampicillin and decreased GI side effects. **Remember:** Like other penicillins, aminopenicillins **inhibit bacterial cell wall synthesis.**

What do the following penicillin antibiotics have in common?

- Methicillin
- Amoxicillin and clavulanate potassium *(Augmentin)*
- Ampicillin and sulbactam *(Unasyn)*
- Cloxacillin
- Dicloxacillin

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• **They are all <u>penicillinase resistant</u> penicillins**

A functional part of the chemical molecule of all the penicillins is the so-called **beta-lactam ring**, which is a four-membered imbedded ring structure consisting of three carbons and one nitrogen atom which is responsible for the antibacterial activity of penicillins. Any alteration to the beta-lactam ring will also alter the antibacterial activity. **Penicillinase** is an enzyme secreted by bacteria which splits open the beta-lactam ring. This renders the penicillin molecule ineffective against those penicillinase secretors.

Methicillin, cloxacillin and dicloxacillin resist the actions of penicillinase because they have a **protected beta-lactam ring** which prevents the actions of the enzyme.

Augmentin and Unasyn contain the agents clavulanate potassium and sulbactam respectively which **block the actions** of penicillinase from reaching the beta-lactam ring.

The majority of penicillins are directly excreted into the urine through renal tubular cell secretion. **Probenecid** *(Benemid)*, an inhibitor of renal tubular cell secretion raises the blood levels of the penicillins by diminishing their tubular secretion. Probenecid is sometimes given simultaneously with penicillin to raise the blood levels for increased activity. Probenecid is a drug used to treat gout.

Which of the following **bacterial enzymes** belong to the family of beta-lactamases?

- Cephalosporinase
- Penicillinase
- ATPase
- Protein kinase

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- **Cephalosporinase**
- **Penicillinase**

Beta-lactamases are **enzymes** produced and secreted by a wide range of gram-positive and gram-negative bacteria as a defense weapon against cephalosporin and penicillin antibiotics. These enzymes destroy the beta-lactam nucleus within these antibiotics by splitting open the beta-lactam ring structure. This action renders the antibiotic ineffective. Those beta-lactamases that work against cephalosporins are called cephalosporinases, and those that work against penicillins are called penicillinases.

Structural modification of penicillins such as occurs in **methicillin** can render the molecule stable in the presence of the beta-lactamase enzyme, but may also narrow the spectrum of action, limiting the use of such antibiotics to gram-positive cocci such as the Staphylococcus species.

By combining **clavulanic acid** with a penicillin, the beta-lactamase enzyme is permanently inhibited by the acid, and the antibacterial activity of the penicillin is maintained. One popular commercial preparation is **Augmentin,** which contains amoxicillin and clavulanate potassium. Augmentin is used orally as pill or liquid form. **Sulbactam** is another beta-lactamase inhibitor. It is available for intravenous and intramuscular use combined with ampicillin under the brand name **Unasyn.**

Which of the following is often **co-administered with antibiotics** to delay the renal clearance of the antibiotic?

- Imipenem
- Probenecid
- Hydrochlorothiazide
- Aztreonam

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• **Probenecid**

***This elevates and prolongs the serum concentrations of the antibiotic when high tissue concentrations are necessary.

The **majority of penicillins** are handled by the kidneys as organic acids and excreted by tubular excretion. **Probenecid interferes** with tubular handling of organic acids within the nephron. Drugs affected by probenecid include most **cephalosporins and natural penicillins,** and other beta-lactam-related antibiotics such as aztreonam and imipenem. **Note**: In some cases, probenecid administration can more than double the serum concentration of the affected drug. The half-life is prolonged as well.

Important: Nafcillin, oxacillin, cloxacillin and dicloxacillin are lipophilic and are **excreted by biliary means.** No combination with probenecid or dosage adjustment for renal dysfunction is necessary for these penicillins.

Notes:

1. **Imipenem** is a beta-lactam antibiotic derived from thienamycin and is the first drug to be classified as a **carbapenem** antibiotic. It is currently the drug of choice for infections due to Enterobacter. It is usually combined with cilastatin and is used to treat severe or resistant infections, especially those that are nosocomial in origin.
2. **Aztreonam** is a parenteral synthetic beta-lactam antibiotic *(classified as a monobactum)*. The spectrum is limited to aerobic gram-negative rods *(i.e., Klebsiella, Pseudomonas, and Serratia)*. It has no gram-positive or anaerobic activity. It is synergistic with aminoglycosides.

Which **antibiotic** is used cautiously due to its side effects *(pseudomembranous colitis, severe GI upset)*?

- Azithromycin *(Zithromax; Z-Pak)*
- Clindamycin
- Penicillin VK
- Cephalexin *(Keflex)*

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- **Clindamycin**

Summary of Important Antibiotics			
Drug	**Antibacterial Action**	**Antibacterial Mechanism**	**Important Adverse Effects**
Penicillins:			
Penicillin VK	Inhibits cell wall synthesis	Bactericidal	Hypersensitivity
Amoxicillin	Same	Same	Same
Amoxicillin/Clav. Acid *(Augmentin)*	Same	Same	Same
Ampicillin	Same	Same	Same
Cephalosporins:			
Cephalexin *(Keflex)*	Inhibits cell wall synthesis	Bactericidal	Hypersensitivity
Cefaclor *(Ceclor)*	Same	Same	Same
Other:			
Clindamycin	Inhibits 50S ribosomes	Bacteriostatic	Pseudomembranous colitis
Macrolides:			
Azithromycin *(Z-Pak)*	Inhibits 50S ribosomes	Bacteriostatic	None significant
Clarithromycin *(Biaxin)*	Same	Same	GI hyperactivity
Erythromycin	Same	Same	Same
Tetracyclines:			
Tetracycline	Inhibits 30S ribosomes	Bacteriostatic	Superinfections
Doxycycline *(Vibramycin)*	Same	Same	None significant
Minocycline *(Minocin; Arestin)*	Same	Same	Same
Aminoglycosides:			
Streptomycin	Inhibits 30S ribosomes	Bactericidal	Ototoxicity, Nephrotoxicity
Gentamycin	Same	Same	Same
Miscellaneous:			
Ciprofloxacin *(Cipro)*	Inhibits DNA	Bactericidal	Nausea, Headache
Bacitracin	Inhibits cell wall synthesis	Same	Nephrotoxicity
Chloramphenicol	Inhibits 50S ribosomes	Bacteriostatic	Blood disorders

Which antibiotic / antimicrobial is associated with the highest incidence of drug allergy?

- Ciprofloxacin *(Cipro)*
- Penicillin VK
- Clindamycin
- Metronidazole *(Flagyl)*
- Chlorhexidine *(Periogard)*

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• **Penicillin VK**

Three groups of **allergic reactions** to the penicillins:

1. **Acute** *(anaphylactic shock)* → occurs within 30 minutes. Characterized by urticaria, angioedema, bronchoconstriction, GI disturbances, and shock. Death can result in a short time if treatment is not instituted immediately *(parenteral administration of epinephrine).*
2. **Accelerated** → occurs 30 to 48 hours after. Manifestations include urticaria, pruritis, wheezing, mild laryngeal edema, and local inflammatory reactions. **Not life threatening.**
3. **Delayed** → occurs after 2 to 3 days. Approximately 80-90% of all allergic reactions occurring with penicillin are of this type. Manifested by skin rashes.

Hypersensitivity reactions occur in up to 10% of patients receiving penicillin. Manifestations range from a mild rash to anaphylaxis. The rash may be urticarial, vesicular, bullous, or maculopapular. Rarely, thrombopenic purpura develops.

Note: A rash is the most common sign of an allergy to penicillin.

PHARMACOLOGY

Anti-Psych

Antipsychotic drugs include:

- The phenothiazines
- The thioxanthenes
- The butyrophenones
- All of the above

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• **All of the above**

By far the most widely used group of antipsychotic agents used in medicine are the **phenothiazines**, followed by the butyrophenones and the thioxanthenes. The **antipsychotic** *(improvement of mood and behavior)* and **neuroleptic effects** *(emotional quieting and development of extrapyramidal symptoms)* of the phenothiazines appear to result from their ability to **block dopaminergic sites** in the brain. Antipsychotics are primarily indicated for the treatment of **psychosis associated with schizophrenia,** paranoia and manic symptoms of manic-depressive illness.

Chlorpromazine *(Thorazine)* and **thioridazine** *(Mellaril)* are the two prototype phenothiazines. The pharmacologic effects of phenothiazines include sedation, **antiemetic activity**, alpha adrenergic receptor blockage and the potentiation of the action of narcotics. **Important:** The phenothiazines are the **most effective antiemetic agents**. They depress the chemoreceptor trigger zone *(CTZ)* to reduce nausea and/or vomiting.

All phenothiazines are withheld from patients with severe **CNS depression** or **epilepsy** and are given with caution to those with liver disease. Adverse effects include hypotension, liver toxicity *(jaundice)*, dry mouth, and **extrapyramidal reactions** *(tardive dyskinesia, etc.)*. **These extrapyramidal reactions are due to the effect on the basal ganglia.**

PHARMACOLOGY

Anti-Psych

All of the following are **phenothiazines, except:**

- Chlorpromazine *(Thorazine)*
- Haloperidol *(Haldol)*
- Prochlorperazine *(Compazine)*
- Trifluprομazine *(Vesprin)*
- Promazine *(Sparine)*
- Trifluoperazine *(Stelazine)*

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- **Haloperidol** ***(Haldol)***

Antipsychotic agents include:

1. **Phenothiazines:**
   - **Chlorpromazine** *(Thorazine)*
   - **Promazine** *(Sparine)*
   - **Thioridazine** *(Mellaril)*
   - **Fluphenazine HCL** *(Prolixin)*
   - **Mesoridazine** *(Serentil)*
   - **Trifluprомazine** *(Vesprin)*
   - **Acetophenazine** *(Tindal)*
   - **Trifluoperazine** (Stelazine)

***Tardive dyskinesia** *(an extrapyramidal disease)* is an irreversible side effect resulting from long-term phenothiazine therapy. It is characterized by involuntary, repetitious movements of the face, the limbs and the trunk.

2. **Butyrophenones:** Haloperidol and droperidol are highly effective antipsychotic drugs used to treat schizophrenia. In addition they have been found to be effective in the treatment of Tourette's syndrome.
3. **Thioxanthenes:** Chlorprothixene and thiothixene are a less potent group of antipsychotics. They are used for the treatment of schizophrenia.
4. **Diverse heterocyclic antipsychotics:** This group includes molindone *(Moban),* clozapine *(Clozaril),* loxapine *(Loxitane)* olanzapine *(Zyprexa),* risperidone (Risperdal) and quetiapine *(Seroquel).* These drugs are effective in treating schizophrenia and have proven to be more effective and less toxic than the older drugs *(i.e, phenothiazines, butyrophenones and thioxanthenes).*

PHARMACOLOGY Asp / Acet / NSAID's

Which of the following are symptoms of **salicylism** and are associated with overdoses of aspirin?

- Tinnitus *(ringing in the ears)*
- Vertigo *(dizziness)*
- Nausea
- Sweating
- Vomiting
- All of the above

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- **All of the above**

Salicylism is the term used to characterize all of the symptoms resulting from the ingestion of extremely large doses of aspirin. Headache and mental confusion can also occur with these large doses.

Aspirin inactivates the enzyme known as cyclooxygenase. Since cyclooxygenase synthesizes the prostaglandins, the inhibition of this enzyme results in the inhibition of prostaglandin synthesis. As a result, aspirin is analgesic, antipyretic *(fever reducing)*, and anti-inflammatory. Aspirin is an irreversible platelet inhibitor and can reduce blood clotting to prolong bleeding.

Low doses of aspirin taken regularly can have a cardioprotective effect. These doses reduce thromboxane production in platelets to result in the inhibition of platelet aggregation. In this way, aspirin has the ability to inhibit the formation of life-threatening thrombi *(blood clots)*.

Contraindications to the use of aspirin:

- Bleeding disorders *(aspirin will increase bleeding time)*
- Do not use in children with viral infections with or without fever due to a potential association with Reye's syndrome *(this syndrome is a serious neurological defect)*
- Pregnancy *(especially during the third trimester)*
- Peptic ulcers *(aspirin may cause bleeding of the GI tract)*

PHARMACOLOGY

Asp / Acet / NSAID's

The main ingredient in **Advil** is also the main ingredient in which of the following **prescription products?**

- Percodan
- Nuprin
- Motrin
- Naprosyn

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- **Motrin**

Advil, Nuprin, and **Medipren** contain 200 mg of **ibuprofen** and may be sold over-the-counter; whereas, **Motrin** contains 400 mg of **ibuprofen** or higher and can only be sold with a prescription.

Nonsteroidal anti-inflammatory drugs *(NSAID's)* have **anti-inflammatory** effects resulting from their ability to inactivate the enzyme prostaglandin endoperoxide synthase *(cyclooxygenase)*. By doing this they inhibit the cyclooxygenase step of the arachidonic acid cascade and thus reduce local prostaglandin synthesis. NSAID's also have **analgesic**, and **antipyretic** actions.

Nonsteroidal anti-inflammatory agents include:

- **Propionic acid derivatives**
  Ibuprofen *(Motrin, Rufen, Advil, Nuprin)*
  Fenoprofen *(Nalfon)*
  Ketoprofen *(Orudis)*
  Naproxen *(Naprosyn)*
  Naproxen sodium *(Anaprox)*
- **Acetic acid derivatives**
  Indomethacin *(Indocin)*
  Sulindac *(Clinoril)*
  Tolmetin *(Tolectin)*
- **Fenamic acid derivatives**
  Meclofenamate *(Meclomen)*
  Mefenamic acid *(Ponstel)*

PHARMACOLOGY — Asp / Acet / NSAID's

Which **non-narcotic** analgesics would you most likely use in a patient taking anti-coagulant medication?

- Ibuprofen *(Motrin, Advil)*
- Acetaminophen *(Tylenol)*
- Aspirin
- Naproxen sodium *(Aleve)*

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• **Acetaminophen** ***(Tylenol)***

Acetaminophen has **no effect** on platelets nor the coagulation pathways and does not affect bleeding times or prothrombin times even with high doses.

Acetaminophen has **two major** pharmacological actions: an **analgesic effect** and an **antipyretic** *(fever reducer)* **effect**. Acetaminophen is **not effective** enough to reduce severe pain, but it is effective in reducing mild to moderate pain. Acetaminophen is a weak inhibitor of prostaglandin formation.

Large doses of acetaminophen can cause liver toxicity. Alcohol can seriously increase the hepatotoxic potential of acetaminophen. There are approximately 100 deaths annually due to liver toxicity produced by ingesting large continuous doses of acetaminophen. Acetaminophen very rarely causes drug sensitivities and can be given to patients having an allergy to aspirin.

Differences **between** Acetaminophen and Aspirin are:

	Acetaminophen	**Aspirin**
• Affect blood clotting	no	yes
• Gastrointestinal ulcers	no	yes
• Cause Reye's syndrome	no	yes
• Anti-inflammatory effects	no	yes
• Analgesic effects	yes	yes

PHARMACOLOGY

Asp / Acet / NSAID's

Which agent has **anti-inflammatory** properties?

- Codeine
- Acetaminophen
- Hydrocodone
- Ibuprofen

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• **Ibuprofen**

Ibuprofen inhibits the production of prostaglandins in peripheral tissues at sites where pain and inflammation are present. Inhibition of prostaglandin production reduces the inflammatory response at sites of surgery, injury or infection. Reduction of inflammation results in reduction of perceived pain.

Acetaminophen is a weak inhibitor of prostaglandin production in peripheral tissues. Thus, the inflammatory response is not affected to any great degree. Acetaminophen reduces pain through mechanisms other than inflammatory reduction. It is unclear exactly how acetaminophen works to reduce pain.

Codeine and hydrocodone are narcotic analgesics that effectively reduce pain but do not reduce inflammation. Narcotics work within the brain to block ascending pain impulses traveling from the periphery into the brain.

PHARMACOLOGY

Barb

The barbiturates Phenobarbital *(Luminal)*, Mephobarbital *(Mebaral)*, and Primidone *(Mysoline)* are classified as what type of barbiturate?

- Ultrashort-acting
- Short-acting
- Intermediate-acting
- Long-acting

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• **Long-acting**

The length of action can be related to the **lipid solubility** with the ultrashort being the most lipid soluble and the long acting having the least lipid solubility. They are metabolized in the liver. These drugs possess serious drug dependence potential. They **do not** possess significant **analgesic** properties. The length of hypnotic action of the four different classes after a single dose are as follows:

- **Ultrashort-acting** → 5 to 20 minutes
- **Short-acting** → 1 to 3 hours
- **Intermediate-acting** → 3 to 6 hours
- **Long-acting** → 6 to 10 hours

Agents:

- **Ultrashort** for induction of general anesthesia. Examples include thiopental *(Pentothal)* and methohexital *(Brevital)*.
- **Short-acting hypnotics** for treating insomnia. Examples include secobarbital *(Seconal)* and pentobarbital *(Nembutal)*.
- **Intermediate-acting hypnotics** for treating insomnia. Examples include amobarbital *(Amytal)* and butabarbital *(Butisol)*.
- **Long-acting sedatives** for providing daytime sedation and as an anticonvulsant. Examples include phenobarbital *(Luminal)*, mephobarbital *(Mebaral)* and primidone *(Mysoline)*.

Remember: Barbiturates **do not** possess analgesic properties.

Note: The cause of death from acute barbiturate poisoning or overdosage is **respiratory failure.** Other adverse reactions include CNS depression, euphoria, and habituation. The most important therapeutic measure to be taken in a case of barbiturate poisoning is to **assure** adequate respiration.

PHARMACOLOGY

Barb

The **brief duration of general anesthetic action** of an ultra-short-acting barbiturate is due to what factor?

- Rapid rate of metabolism in the liver
- Low lipid solubility, resulting in a minimal concentration in the brain
- High degree of binding to plasma proteins
- Rapid rate of redistribution from the brain to peripheral tissues
- Slow rate of excretion by the kidneys

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- **Rapid rate of redistribution from the brain to peripheral tissues**

Remember: These agents will maintain anesthesia only while in the brain. Because of their high lipid solubility, these agents will rapidly leave the brain for other tissues; thus the patient wakes up within a few minutes of administration.

Examples of ultra-short-acting barbiturates include thiopental *(Pentothal)* and methohexital *(Brevital)*. These agents are administered by intravenous injection.

Contraindications to the use of ultra-short-acting barbiturates for general anesthesia:

- Porphyria
- Liver dysfunction *(they are metabolized in the liver)*
- Emphysema
- Previous addiction to sedative hypnotic drugs

Remember: These drugs have the **ability to produce dependence.**

PHARMACOLOGY

Barb

All of the following statements concerning **barbiturates** are true **except:**

- Barbiturates may increase the half-lives of drugs metabolized by the liver
- Barbiturates depress neuronal activity by increasing membrane ion conductance *(primarily chloride),* reducing glutamate-induced depolarizations and potentiating the inhibitory effects of GABA
- Compared with benzodiazepines, the barbiturates exhibit a steeper dose-response relationship
- Barbiturates may precipitate acute porphyria in susceptible patients

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• **Barbiturates may increase the half-lives of drugs metabolized by the liver**

***This is **false;** barbiturates may **decrease** the half-lives of drugs metabolized by the liver. **Barbiturates induce the formation of the liver microsomal enzymes that metabolize drugs**. This leads to an increased clearance of the affected drugs and possibly leads to a decrease in the drugs effectiveness.

The uses of the barbiturates are determined by their **duration of action:**

1. The **ultrashort-acting** agents are used intravenously for the induction of general anesthesia. For extensive procedures, they are used to induce stage III surgical anesthesia. For very brief procedures, they may be used alone.
2. The **short-acting** agents can be used orally for their hypnotic, calming effect. These agents can be given preoperatively, before a dental appointment, to allay anxiety.
3. The **intermediate-acting** agents can also be prescribed to relieve anxiety before a dental appointment, although their effects will last longer than those of the short-acting agents. These agents are used for daytime sedation and for the treatment of insomnia *(they suppress REM sleep).*
4. The **long-acting** barbiturates are used primarily for daytime sedation and the treatment of epilepsy.

Generally the primary pharmacological effects of the barbiturates are on the nervous tissue *(depression of).* **Barbiturates are metabolized in the liver. These drugs possess serious drug dependence potential.** They **do not possess** significant analgesic properties.

Alkylating agents are most effective in treating:

- Chronic leukemias
- Lymphomas
- Myelomas
- Carcinomas of the breast and ovary
- All of the above

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• **All of the above**

The **alkylating agents** contain a diverse group of compounds which all form **alkyl bonds to nucleic acids**. All of these agents share a similar mechanism of action and mechanism of resistance. The alkylating agents form **covalent bonds** with nucleic acids, and proteins. The N-7 position of guanine is a **common binding site.**

Major Alkylating Agents	
Drug	**Use**
Nitrogen Mustards:	
Mechlorethamine *(Mustargen)*	Hodgkin's disease and other lymphomas
Cyclophosphamide *(Cytoxan)*	Lymphomas, leukemias, multiple myeloma, neuroblastoma, retinoblastoma, and cancers of the breast & ovary
Chlorambucil *(Leukeran)*	Chronic lymphocytic leukemia
Melphalan *(Alkeran)*	Hodgkin's disease and other lymphomas
Nitrosoureas:	
Carmustine *(BCNU, BiCNU)*	Several types of cancer, including brain cancer
Lomustine *(CCNU, CeeNU)*	Several types of cancer, including brain cancer and Hodgkin's disease
Semustine *(Methyl-CCNU)*	Several types of cancer, including brain cancer
Busulfan: *(Myleran)*	Chronic myelogenous leukemia

Antimetabolites are cell cycle-specific drugs acting primarily in the:

- G_0 phase or resting phase of the cell cycle
- S phase of the cell cycle
- G_1 phase of the cell cycle
- G_2 phase of the cell cycle

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• **S phase of the cell cycle**

***The S phase of the cell cycle = **DNA synthesis**

Antimetabolites are one of the oldest and most important classes of antineoplastic agents. They attack the cells in the S phase of the reproduction cycle by **interfering with the biosynthesis of the purine and pyrimidine bases.**

Antimetabolites used as Antineoplastic Agents	
Drug	**Use**
Folic acid analog:	
Methotrexate *(Amethopterin)*	Choriocarcinoma, leukemia in the spinal fluid, osteogenic sarcoma, breast cancer, and head and neck cancer
Pyrimidine analogs:	
Fluorouracil *(5-FU)*	Several types of cancers including colon and head and neck cancers
Floxuridine *(FUDR)*	Some cancers involving the liver
Cytosine arabinoside *(Cytosar-U)*	Treating acute leukemias
Purine analogs:	
Mercaptopurine *(Purinethol)*	Several types of leukemia
Thioguanine *(6-TG, Tabloid)*	Acute nonlymphocytic leukemia

Important: Alopecia *(hair loss)* occurs with the administration of most chemotherapeutic agents **one to two weeks after treatment.** Other common side effects include GI upset, increased incidence of infection *(especially Candidiasis)*, and degeneration of lymphatic tissue.

Remember: Most chemotherapy drugs have been shown to be **teratogenic in humans** and should be avoided by pregnant women.

Name the **eight classes** of drugs used in cancer chemotherapy.

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• **Listed below are the eight classes of drugs used in chemotherapy:**

1. **Alkylating agents** – these agents alkylate DNA such that it cannot replicate. Popular agents include **Cisplatin**, and **Cyclophosphamide**.
2. **Anthracyclines** – these agents destroy DNA such that the cell cannot replicate. Popular agents include **Daunorubicin** and **Doxorubicin**.
3. **Antibiotics** – these antibiotics are not used for antibacterial therapy but were specifically designed for cancer chemotherapy. The agent in this class is **Dactinomycin**.
4. **Antimetabolites** – these agents interfere which selected biochemical reactions necessary for cell growth. Popular agents are **5-Fluorouracil** *(5-FU)*, **6-Mercaptopurine** and **Methotrexate**.
5. **Antimicrotubular** – affects the microtubular assembly with cells to inhibit cell mitosis. The popular agent is **Paclitaxel** *(Taxol)*.
6. **Antiestrogen** – these agents block the tumors on which estrogen has a stimulatory effect. The popular agent is **Tamoxifen** *(Nolvadex)*.
7. **Vinca Alkaloids** – these are mitotic spindle poisons. Examples include **Vinblastine** and **Vincristine**.
8. **Gonadotropin hormone-releasing antigen** – these inhibit gonadotropin secretion, an action which is effective in reducing certain carcinomas. The popular agent is **Leuprolide.**

Note: The anticancer drugs **Asparaginase** and **Interferons** do not fall within any category but are used in the treatment of certain cancers. Asparaginase deprives tumor cells of certain amino acids such that protein production is blocked. Interferons boost the immune system.

PHARMACOLOGY

Chol

The autonomic nervous system has **cholinergic neurons** that secrete:

- Epinephrine
- Norepinephrine
- Acetylcholine
- Dopamine

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• **Acetylcholine *(ACh)***

Cholinergic is a term for a nerve ending that **releases acetylcholine** *(ACh)* as the primary neurotransmitter; also, a synapse in which acetylcholine is the primary neurotransmitter.

Acetylcholine receptors are called **cholinergic receptors.** They are subdivided as follows:

- **Muscarinic receptors**: These receptors are located primarily **in autonomic effector cells** *(including the heart, vascular endothelium, smooth muscle, presynaptic nerve terminals, and exocrine glands)* **and in the CNS.** These receptors respond to muscarine as well as ACh.
- **Nicotinic receptors:** These receptors are located in **ganglia, in skeletal muscle end plates and in the CNS.** These receptors respond to nicotine as well as ACh **but not** to muscarine.

****Drugs that resemble ACh in chemical structure and bind to these receptors imitate the effects of **parasympathetic postganglionic activity.**

Important:

1. **Preganglionic** neurons of both divisions *(parasympathetic and sympathetic)* are **cholinergic**, as are **postganglionic** neurons of the **parasympathetic division.**
2. **Postganglionic** neurons of the **sympathetic** division are usually **adrenergic.**

Notes:

1. The action of acetylcholine at postganglionic parasympathetic sites is often referred to as a **muscarinic response.**
2. The term **nicotinic response** is used to describe the stimulating action of ACh on the ganglia, as well as its action at the neuromuscular junction of skeletal muscle.

PHARMACOLOGY

Chol

All of the following are **cholinergic actions, except:**

- Slowing of the heart
- Dilation of the pupils
- The stimulation of the smooth muscles of the bronchi, GI tract, gallbladder, bile duct, bladder and ureters
- The stimulation of sweat, salivary, tear and bronchial glands

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- **Dilation of the pupils**

Cholinergic actions:

- **The stimulation** of sweat, salivary, tear and bronchial glands
- **The stimulation** of the smooth muscles of the bronchi, GI tract, gallbladder, bile duct, bladder and ureters *(i.e., urination)*
- Slowing of the heart *(bradycardia)*
- Constriction of the pupils *(miosis)*

Important: Acetylcholine is the chemical mediator at all **autonomic ganglia** and **parasympathetic postganglionic** synapses. It is also the transmitter substance of the **neuromuscular junction** in skeletal muscle *(local anesthetics prevent or reduce the liberation of ACh at the NMJ)* and sweat glands. Acetylcholine causes an alteration in **cell membrane permeability** to produce the above actions.

Anticholinergic actions:

- The secretions of all glands in the nose, mouth, pharynx and respiratory tract **is inhibited** *(unpleasant "dry mouth")*
- An **inhibitory effect** on motility throughout the GI tract *(may cause constipation and urinary retention)*
- The heart rate increases *(tachycardia)*
- A rise in body temperature
- Dilation of the pupils *(mydriasis)*

PHARMACOLOGY

Chol

All of the following are **antimuscarinic** agents **except:**

- Atropine
- Scopolamine
- Glycopyrrolate *(Robinul)*
- Mecamylamine *(Inversine)*
- Propantheline *(Pro-Banthine)*

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• **Mecamylamine *(Inversine)***

***Mecamylamine is a **nicotinic ganglion-blocking drug.**

The typical effects of **anticholinergic** drugs include mydriasis, antispasmodic actions and reduction in gastric and salivary secretions *(dry mouth)*. **Important:** These drugs are **contraindicated** in patients with **glaucoma.**

These drugs have **no intrinsic activity** of their own; they simply occupy the receptor site and **prevent acetylcholine** from occupying the same receptor. Accepted therapeutic indications include treating Parkinson's disease, motion sickness, postoperative bladder syndrome and traveler's diarrhea.

Synthetic Anticholinergic Drugs	
Drug Name	**Major Use**
Glycopyrrolate *(Robinul)*	Inhibit salivation and excessive secretions preoperatively; control of upper airway secretions
Propantheline bromide *(Pro-Banthine)*	Traveler's diarrhea and antisecretory
Benzotropine mesylate *(Cogentin)*	Antiparkinsonism
Trihexyphenidyl HCl *(Artane)*	Antiparkinsonism
Atropine	To produce mydriasis and cycloplegia
Scopolamine	To prevent or reduce motion sickness

The currently available **ganglionic blockers** for clinical use include:

- Mecamylamine
- Hexamethonium
- Tetraethylammonium
- Trimethaphan

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- **Mecamylamine** ***(Inversine)***
- **Trimethaphan** ***(Arfonad)***

***Hexamethonium** and **tetraethylammonium** are no longer available in the U.S. for clinical use.

Nicotinic receptor antagonists *(nicotinic blocking agents)* are divided into **ganglion-blocking** drugs and **neuromuscular blocking** drugs.

Remember:
Two major types of nicotinic receptors:
1. Those at the neuromuscular junction of the somatic system
***neuromuscular blockers act here.**
2. Those at the autonomic ganglia *(both sympathetic and parasympathetic)*
***ganglionic blockers act here.**

Although they are among the most potent agents available, ganglionic-blocking drugs are seldom used because of the annoying and sometimes disabling **parasympathetic blockade**. The **side effects** *(caused by parasympathetic blockade)* include a very pronounced xerostomia, constipation, blurred vision, and postural hypotension. These drugs have only **very limited** clinical uses.

Mecamylamine *(Inversine)* and **Trimethaphan** *(Arfonad)* are used clinically for:
- The treatment of **severe or malignant hypertension**
- An **emergency hypertensive crisis**

***These agents cause a rapid and reversible **fall in blood pressure** that enables them to immediately reverse an emergency hypertensive crisis
- A "bloodless field" surgery

PHARMACOLOGY

Chol

The prototype **depolarizing** neuromuscular blocking agent is:

- Tubocurarine *(Curare)*
- Mivacurium *(Mivacron)*
- Succinylcholine *(Anectine)*
- Vecuronium *(Norcuron)*
- Doxacurium *(Nuromax)*
- Pancuronium *(Pavulon)*
- Atracurium *(Tracrium)*
- Cisatracurium *(Nimbex)*
- Rocuronium *(Zemuron)*

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• **Succinylcholine *(Anectine)***

Neuromuscular blocking drugs are important for producing complete skeletal muscle relaxation and facilitate endotracheal intubation, **as an adjunct to surgical anesthesia.** These agents interact with nicotinic receptors at the **skeletal neuromuscular junction.**

There are **two classes** of neuromuscular blockers:

1. **Nondepolarizing**: These agents **competitively** compete with acetylcholine at the nicotinic receptor. These agents bind to nicotinic cholinergic receptors and prevent acetylcholine from stimulating motor nerves, resulting in muscle paralysis. The **prototype** nondepolarizing agent is **tubocurare** *(Curare).* Other agents include mivacurium, vecuronium, doxacurium, pancuronium, atracurium, cisatracurium and rocuronium. **Note: Neostigmine or pyridostigmine** *(which are cholinesterase inhibitors)* can reverse the blockade of these agents.
2. **Depolarizing** *(non-competitive):* Succinylcholine *(Anectine)* is the only member of this group used in the United States. It acts like a nicotinic agonist and **depolarizes** *(desensitizes)* the neuromuscular end plate. It binds to the ACh receptor and stimulates depolarization causing initial excitation followed by block of neurotransmission and muscle paralysis. **Important**: Succinylcholine should be used with caution in patients with **low levels of pseudocholinesterase,** which breaks down succinylcholine. **Respiratory failure may result.**

***Succinylcholine may cause **muscarinic responses** such as bradycardia and increased glandular secretions.

Very important: The major danger of all of these neuromuscular blocking drugs is **too much paralysis** *(the patient cannot breathe).*

True or False

All of the following drugs have an action which results in increased effects of acetylcholine within the autonomic nervous system and at the neuromuscular junctions.

- Neostigmine
- Physostigmine
- Edrophonium
- Pyridostigmine

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• **True**

These agents are known as **cholinesterase inhibitors**. By inhibiting cholinesterase, acetylcholine produced within the body is not broken down into by-products, but is allowed to build up into substantial levels to stimulate the cholinergic portions of the autonomic nervous system and to stimulate skeletal muscle contractions at the neuromuscular junctions. Thus the actions of the cholinesterase inhibitors results in a **cholinergic effect**. This is also known as **an indirect cholinergic effect** since these drugs indirectly cause a cholinergic response by allowing for the build-up of acetylcholine.

The other way that a cholinergic effect can be produced by drugs is by direct stimulation of the cholinergic receptors by the drugs. Two groups of drugs which directly stimulate cholinergic receptors are:

1. **Choline esters** *(Methacholine, Carbachol, and Bethanecol)*
2. **Cholinergic alkaloids** *(Pilocarpine)*

***The actions of these drugs is known as a **direct cholinergic effect.**

Pilocarpine *(Salagen)* is used to stimulate salivary flow in patients suffering from xerostomia due to radiation therapy in the treatment of head and neck cancer.

Note: The stimulation of the skeletal muscle by excess acetylcholine eventually results in muscle paralysis.

PHARMACOLOGY

Chol

All of the following are **choline esters, except:**

- Pilocarpine *(Pilocar)*
- Methacholine *(Provocholine)*
- Bethanechol *(Urecholine)*
- Carbachol *(Isopto-Carbachol)*

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• **Pilocarpine** ***(Pilocar)***

***Pilocarpine** is a cholinergic alkaloid.

There are three classes of **cholinergic agonists**. These drugs stimulate muscarinic sites by **mimicking the actions of acetylcholine;** accordingly these drugs are referred to as muscarinics, cholinergics, cholinomimetics, or parasympathomimetics.

1. **Choline esters:** The most noticeable effects of these drugs are a fall in blood pressure attributable to generalized vasodilation, flushing of the skin, a slowing of the heart rate, and an increased tone and activity of both the GI and urinary tracts. Topical application of these drugs to the eye causes miosis and a decrease in intraocular pressure. These drugs include:
   - **Acetylcholine chloride:** used in ophthalmology to produce miosis
   - **Methacholine:** not used much
   - **Bethanechol:** used for postoperative abdominal distension and urinary retention
   - **Carbachol:** used in ophthalmology to produce miosis
2. **Cholinergic alkaloids:** These drugs include muscarine, pilocarpine, nicotine and lobeline. **Pilocarpine** is the most useful alkaloid being employed as a **miotic and to treat open angle glaucoma.**

***For the most part, these drugs *(esters and alkaloids)* are used to stimulate smooth muscle activity in several clinical situations. The choline esters and alkaloids are **direct-acting cholinomimetic agents** *(effects resemble those of ACh)*

3. **Cholinesterase inhibitors:** These drugs include physostigmine, neostigmine, edrophonium, pyridostigmine, malathion and parathion. They inhibit acetylcholinesterase at **both mucarinic and nicotinic sites.** They are **indirect-acting cholinomimetic agents.**

Note: If any of the cholinergic agents are administered before acetylcholine, the action of acetycholine will be enhanced and prolonged.

In dentistry, a **cholinergic drug** is used to:

- Produce a dry field for taking impressions
- Calm an anxious patient
- Treat dry mouth by inducing salivation
- Reduce nausea

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- **Treat dry mouth by inducing salivation**

Cholinergic drugs used in dentistry are: Pilocarpine *(Salagen)*, and Cevimeline *(Evoxac)*.

- **Pilocarpine** *(Salagen)* – is indicated for treatment of xerostomia caused by salivary gland hypofunction resulting from radiotherapy for cancer of the head and neck. It's pharmacologic category is **cholinergic agonist**. Prevalent side effects are excess sweating and nausea / heartburn / diarrhea due to the cholinergic nature of the drug.

- **Cevimeline** *(Evoxac)* – is indicated for the treatment of symptoms of xerostomia in patients with Sjögren's syndrome. It's pharmacologic category is **cholinergic agonist.** Prevalent side effects are increased sweating and nausea / heartburn / diarrhea due to the cholinergic nature of the drug.

PHARMACOLOGY

Chol

All of the following statements concerning **edrophonium** are true **except:**

- It is a direct-acting cholinergic agonist *(cholinomimetic)*
- It is a rapid-acting, short-duration, injectable cholinesterase inhibitor
- It is the drug of choice for diagnosing myasthenia gravis because of its rapid onset of action and reversibility
- It is also useful in differentiating a myasthenic crisis from a cholinergic crisis

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• **It is a direct-acting cholinergic agonist** *(cholinomimetic)*

***This is **false;** edrophonium is an **indirect-acting** cholinergic agonist *(cholinomimetic)* as are neostigmine, pyridostigmine and physostigmine *(they are all cholinesterase inhibitors).*

Neostigmine and pyridostigmine are prescribed in the **treatment** of myasthenia gravis, however **edrophonium** is used to **diagnose myasthenia gravis** and not in the treatment of it due to its **very short duration of action.**

Symptoms of a cholinergic crisis include: bradycardia *(decreased heart rate),* lacrimation, extreme salivation, vasodilation and muscle weakness. Because a cholinergic crisis can result in muscle weakness like that of a myasthenic crisis, distinguishing the two conditions may be difficult. **Administration of a short-acting cholinomimetic** such as edrophonium will improve a myasthenic crisis but worsen a cholinergic crisis.

Remember: Typical **cholinergic effects** caused by stimulation of acetylcholine receptors *(cholinergic receptors)* include salivation, miosis, excessive sweating, flushing, increased GI motility and bradycardia.

Poisoning with an **organophosphate** cholinesterase inhibitor can be treated with:

- Edrophonium
- Carbachol
- Pralidoxime
- Nicotine

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- **Pralidoxime**

Pralidoxime *(Protopam)* is a cholinesterase reactivator which is used as an antidote to reverse muscle paralysis resulting from **organophoshate anticholinesterase pesticide poisoning.** It is also used to reverse the effects of an **overdosage of anti-cholinesterase** agents used in the treatment of myasthenia gravis *(i.e., neostigmine, pyridostigmine and ambenomium).*

Symptoms of organophosphate poisoning include: excessive salivation, bron-choconstriction, diarrhea and skeletal muscle fasciculations *(twitching).*

Organophosphates are esters of phosphoric acid and an organic alcohol that inhibit the enzyme cholinesterase.

Examples include:
- **Isoflurophate:** used in the treatment of glaucoma
- **Malathion:** a widely used insecticide
- **Parathion:** an insecticide
- **Echothiophate:** used in the treatment of glaucoma
- **Tabun:** one of the most potent and toxic nerve gases
- **Metrifonate:** an anthelmintic agent *(destroys intestinal worms)*

PHARMACOLOGY

Corticosteroids

The major **natural** glucocorticoid is:

- Triamcinolone
- Cortisol *(Hydrocortisone)*
- Dexamethasone *(Decadron)*
- Prednisone
- Prednisolone

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• **Cortisol *(Hydrocortisone)***

The corticosteroids are steroid hormones produced by the adrenal cortex. They consist of two major groups:

1. **Glucocorticoids**: have important effects on metabolism, catabolism, immune responses and inflammation. The majority of the anti-inflammatory and immunosuppressive actions of the glucocorticoids are probably the result of their action on **arachidonic** acid metabolism. They induce the synthesis of a protein that inhibits phospholipase A2, thus **decreasing the production of both prostaglandins and leukotrienes.** The major natural glucocorticoid is cortisol. The synthetic glucocorticoids include prednisone, prednisolone, dexamethasone and triamcinolone.
2. **Mineralocorticoids**: regulate sodium and potassium reabsorption in the collecting tubules of the kidney. The major natural mineralocorticoid in humans is **aldosterone**. Other mineralocorticoids include deoxycorticosterone and fludrocortisone.

Corticosteroids are useful in treating many conditions, these include: **asthma**, arthritis *(if patient also has peptic ulcer disease, do not use corticosteroids)*, allergic reactions, **Addison's disease** *(for adrenal crisis, treat with 2 mL hydrocortisone)*, lupus erythematosus and aphthous stomatitis. **Note**: Corticosteroids **do not cure** any disease. They represent replacement only in Addison's disease.

Contraindications to corticosteroid use include: latent TB or fungal infection, AIDS, herpes infections and patients with peptic ulcer disease *(specifically, gastric ulcers)*. These drugs themselves may **cause peptic ulcers.**

Toxic effects of the corticosteroids include growth inhibition, hyperglycemia, osteoporosis, psychosis and salt retention.

PHARMACOLOGY

Corticosteroids

All of the following are pharmacologic effects of **glucocorticoids except:**

- A decrease in gluconeogenesis
- A decrease in the utilization of glucose
- The inhibition of protein synthesis
- An increase in protein catabolism
- Impaired wound healing
- A decreased resistance to infection

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• **A decrease in gluconeogenesis**

***This is **false;** glucocorticoids **enhance gluconeogenesis** through the breakdown of endogenous proteins to amino acids, which are then converted to glucose for storage in the liver or for use by the body.

Other effects of glucocorticoids include an **anti-inflammatory action,** immunosuppression, and an anti-allergenic action. **Note**: These effects occur in target cells following the interaction of the steroid with a specific glucocorticoid receptor.

The pharmacologic effects **of mineralocorticoids** include an increase in sodium retention and an increase in potassium depletion which can lead to edema and hypertension **if excessive** and may lead to dehydration and hypotension **if insufficient.**

Note: Beclomethasone, budesonide and flunisolide are special glucocorticoids *(used as inhalers)* that have been developed for use in **chronic asthma and bronchial disease.** These agents readily penetrate the airway mucosa but have very short half-lives after they enter the blood, so that systemic effects and toxicity are greatly reduced.

PHARMACOLOGY

Corticosteroids

The major natural **mineralocorticoid** in humans is:

- Dexamethasone
- Aldosterone
- Cortisol
- Prednisone

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• **Aldosterone**

***Cortisol, prednisone and dexamethasone are **glucocorticoids.**

Aldosterone is secreted by cells located in the **zona glomerulosa of the adrenal cortex**. The secretion of aldosterone is regulated by ACTH and by the renin-angiotensin system and is very important in the regulation of blood volume and pressure. Aldosterone promotes reabsorption of sodium into the blood from the glomerular filtrate. Potassium is lost in the urine because of the electronegativity that is created by the reabsorption of sodium in the kidney tubules.

Note: Increased blood aldosterone levels will result in high sodium and low potassium levels in the plasma.

Remember: Decreased sodium concentration causes the juxtaglomerular cells of the kidneys to **secrete renin,** which converts angiotensinogen to angiotensin I. Angiotensin I is converted to angiotensin II, **which, in turn, stimulates the adrenal cortex to release aldosterone.**

Notes:

1. Addison's disease is caused by the hyposecretion of aldosterone and cortisol.
2. ADH *(Vasopressin)* decreases the production of urine by increasing the reabsorption of water by the renal tubules *(it increases the permeability of the collecting ducts and distal tubules)*. Without ADH, there would be extreme loss of water into the urine. **Note: At high concentrations, ADH** causes arterioles to constrict *(increases blood pressure)*.

All of the following drugs are **direct vasodilators except:**

- Hydralazine *(Apresoline)*
- Minoxidil *(Loniten)*
- Diazoxide *(Hyperstat)*
- Captopril *(Capoten)*
- Sodium nitroprusside *(Nipride)*
- Nitroglycerin *(Nitrostat)*

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• ***Captopril (Capoten)***

***Captopril is an **angiotensin-converting enzyme inhibitor.**

Direct vasodilators exert their antihypertensive effect by a **direct vasodilator action on the smooth muscle of arterioles**, resulting in a decrease in peripheral resistance and blood pressure. Compensatory responses may be marked and include salt retention and tachycardia. Adverse side effects include GI upset, headache, dizziness and tachycardia.

Note: Minoxidil is extremely efficacious and is thus reserved for severe hypertension. **Nitroprusside** and **Diazoxide** are parenteral vasodilators which are used in hypertensive emergencies.

Calcium channel blockers are also effective vasodilators *(indirect)* and have been applied to the management of hypertension. **Verapamil** *(Calan SR)*, **Nifedipine** *(Procardia)*, and **Diltiazem** *(Cardizem)* have been given orally for the treatment of mild to moderate hypertension.

PHARMACOLOGY

Cv

Which drugs are used to **prevent** or to **provide relief** of angina pectoris?

- Nitroglycerin
- Nifedipine *(Procardia)*
- Diltiazem *(Cardizem)*
- Propranolol *(Inderal)*
- All of the above

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• **All of the above**

Angina pectoris is the pain in the heart and chest which occurs during the occlusion of coronary arteries. Triggers that can cause occlusion are physical exertion, increased blood pressure, and vasoconstriction.

Nitroglycerin is a coronary artery **vasodilator**. It relaxes blood vessels to provide increased blood flow and oxygenation to the heart muscle. It is sublingually effective within 2-4 minutes. The nitroglycerin skin patch releases the drug over a 12 hour period to provide sustained blood levels for prevention of angina. The two most common adverse effects caused by nitroglycerin are orthostatic hypotension and headache.

Nifedipine *(Procardia)* and **diltiazem** *(Cardizem)* are **calcium channel blockers** used to prevent angina attacks. These drugs are used to dilate coronary blood vessels for improved blood flow to heart muscle. Calcium channel blockers as a class have been associated with causing gingival hyperplasia.

Propranolol *(Inderal)* is representative of the beta blockers used to prevent angina attacks. **Atenolol** *(Tenormin)* is another popular beta blocker used for this purpose. Beta blockers are used to decrease the work load of the heart such that less oxygen is required.

PHARMACOLOGY

Cv

Amyl nitrite is used in the emergency treatment of cyanide poisoning because it:

- Oxidizes hemoglobin
- Irreversibly binds cyanide
- Competes with cyanide for binding of cytochromes
- Inhibits tubular reabsorption of cyanide

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- **Oxidizes hemoglobin**

***Amyl nitrite oxidizes hemoglobin to methemoglobin which binds cyanide tightly, keeping it in the peripheral circulation and preventing its access to tissues.

Amyl nitrite is a vasodilator and a highly volatile substance administered by inhalation only. It is the most rapidly acting of the antianginal drugs, producing effects within 10 seconds. Its duration of action is only 3 to 5 minutes. Because of its **extreme potency**, there are uncomfortable side effects that invariably occur with its use *(fainting and a pounding headache)*. **Important**: This drug is rarely prescribed and is not the first drug of choice in treating angina. **It is abused to produce euphoria and as a sexual stimulant.**

Other **anti-anginal** drugs include:

1. **Nitrate:** Nitroglycerin ***This drug is the **single most effective agent** available for the management of acute angina episodes.
2. **Nonnitrate vasodilator:**
   Dipyridamole *(Persantine)*
3. **Beta adrenergic blocking drugs:**
   Propranolol *(Inderal)*
   Nadolol *(Corgard)*
   Atenolol *(Tenormin)*
4. **Calcium channel blocking drugs:**
   Verapamil *(Isoptin, Calan)*
   Diltiazem *(Cardizem)*
   Nifedipine *(Procardia)*

PHARMACOLOGY

Cv

The positive inotropic effect of **Digoxin** *(Lanoxin)*:

- Is dependent upon a normal cardiac rhythm
- Directly increases the force of myocardial contractions
- Is antagonized by beta-blockers
- All of the above

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• **Directly increases the force of myocardial contractions**

***This **positive inotropic effect** is independent of a normal sinus rhythm and adrenergic stimulation.

The **cardiac glycosides** are often called **"digitalis"** because several come from the digitalis *(foxglove)* plant. **Digoxin** *(Lanoxin)* is the most versatile and widely used.

Note: They are used to treat most supraventricular arrhythmias, cardiogenic shock and congestive heart failure.

These drugs help the heart **beat more strongly** *(positive inotropic effect)*, **more slowly** *(bradycardia)* and **more efficiently.**

Cardiac glycosides inhibit the Na-K-ATPase membrane pump by inhibiting the adenosine triphosphate enzymes *(transport ATPase or Na-K-ATPase)*. Na-K-ATPase splits adenosine triphosphate in the nerve and muscle cell and thus provides the energy necessary for transporting sodium across the cell membrane. **Key point:** This inhibiton of the Na-K-ATPase enzyme leads to an increased calcium ion influx which **augments the positive inotropic effect** of cardiac glycosides. **Adverse side effects** include appetite loss and diarrhea. **Contraindications** to their use include ventricular fibrillation and ventricular tachycardia.
Remember: Most drugs useful in treating **cardiac arrhythmias act primarily by increasing the refractory period of cardiac muscle.**

The maximum dose of **articaine** *(Zorcaine)* that is recommended in one appointment is expressed as mg per kilogram body weight rather than total milligrams. Which dose is the maximum recommended dose for articaine in **children and adults:**

- 7 mg/kg
- 100 mg/kg
- 1 mg/kg
- 300 mg/kg

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• **7 mg/kg**

In a typical kg adult male, the dose of 7 mg/kg would equate to a total of 490 mgs. Thus, the maximum recommended amount of articaine that could be given to a 70 kg adult in one appointment is 490 mgs.

The table below shows the following number of dental cartridges containing 1.7 ml volume of solution to provide the indicated amounts of articaine hydrochloride 4% and epinephrine 1:100,000. **Note:** The carpule fluid volume of 1.7 ml is unique for articaine and is not the standard carpule volume of 1.8 ml of the other dental anesthetics.

Number of Cartridges *(1.7 ml)*	**Articaine HCl (4%)** *(mg)*	**Epinephrine 1:100,00** *(mg)*
1	68	0.017
2	136	0.034
3	204	0.051
4	272	0.068
5	340	0.085
6	408	0.102
7	476	0.119
8	544	0.136

All of the following are **angiotensin-converting enzyme** *(ACE)* **inhibitors except:**

- Captopril *(Capoten)*
- Hydralazine *(Apresoline)*
- Enalapril *(Vasotec)*
- Lisinopril *(Zestril)*

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• **Hydralazine *(Apresoline)***

***Hydralazine is a direct vasodilator.

ACE inhibitors interfere with the conversion of **angiotensin I** *(a weak vasoconstrictor)* to **angiotensin II** *(a highly effective constrictor)*. They do this by **being inhibitors of angiotensin-converting enzyme** *(ACE)*. These drugs are used to treat hypertension and congestive heart failure. **Adverse effects include** cough, hypotension, neutropenia, anorexia and polyuria.

Other ACE inhibitors include:

• Benazepril *(Lotensin)* • Ramipril *(Altace)* • Fosinopril *(Monopril)* • Quinapril *(Accupril)* • Perindopril *(Aceon)*

Angiotensin II receptor blockers include:

• Losartan *(Cozaar)* • Valsartan *(Diovan)* • Candesartan *(Atacand)* • Irbesartan *(Avapro)*

***These drugs are a new class of blood pressure medications that **inhibit angiotensin II** at its receptor site. This **prevents** angiotensin II from **constricting the blood vessels, which tends to raise the blood pressure.**

Note: ACE inhibitors and Angiotensin II receptor blockers indirectly **inhibit fluid volume increases** when interfering with angiotensin II **because** angiotensin II stimulates the release of aldosterone, which promotes sodium and water retention.

True or False

Diuretics are used to treat congestive heart failure by relieving edema and symptoms of dyspnea arising from pulmonary congestion.

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- **True**

Diuretics are also used to treat hypertension, and in the management of edema associated with hepatic or renal disease.

Three categories of widely used diuretics:

- **Thiazides** → inhibit sodium reabsorption in the distal portion of the renal tubule within the kidney causing increased excretion of sodium and water. Prototype agent is hydrochlorothiazide *(HCTZ)*.
- **Loop diuretics** → inhibit reabsorption of sodium and chloride in the ascending Loop of Henle thus causing increased secretion of water, sodium and chloride. Prototype agent is furosemide *(Lasix)*.
- **Potassium-sparing diuretics** → these agents conserve potassium while causing diuresis. Thus, no potassium is lost from the body as is the case with other diuretics such as the thiazides and loops. Examples include **Spironolactone** *(Aldactone)* which competes with aldosterone receptor sites in renal tubules causing increased secretion of sodium, chloride and water while conserving potassium. **Triamterene** *(Dyrenium)* is the other potassium sparing diuretic that promotes sodium and water excretion while retaining potassium.

***Dyazide** is the brand name for the combination of triamterene and hydrochlorothiazide *(HCTZ)*. This product combines the potassium-sparing diuretic with HCTZ for greater efficacy than either one individually.

Which of the following is a pharmacologic **antagonist of aldosterone** in the collecting tubule?

- Mannitol *(Osmitrol)*
- Glycerin *(Glyrol)*
- Spironolactone *(Aldactone)*
- Isosorbide *(Ismotic)*
- Urea *(Ureaphil)*

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• **Spironolactone** *(Aldactone)*

Potassium-sparing diuretics result in increased sodium and decreased potassium concentrations at the end of the distal convoluted tubules. There are two categories of potassium-sparing diuretics:

1. **Antagonist of aldosterone in the collecting tubules:** Spironolactone *(Aldactone)* **Note:** Spironolactone has been found to be effective in the treatment of **primary aldosteronism.** It may be useful in the treatment of **heart failure** because hyperaldosteronism is commonly seen in this condition.
2. **Block the sodium channels in the collecting tubules:** Triamterene *(Dyrenium)* and Amiloride *(Midamor)*.

***The most important toxic effect of potassium-sparing diuretics is **hyperkalemia.**

Osmotic diuretics are highly filtered by the glomerulus and exert a solute-induced diuresis in the proximal tubule. They are used to reduce excess edema associated with neurosurgery or trauma to the CNS. Examples include: **mannitol, glycerin, isosorbide and urea.**

PHARMACOLOGY

Hypoglycemics

All the following drugs are considered to be in what **drug class?**

- Glyburide *(DiaBeta)*
- Metformin *(Glucophage)*
- Pioglitazone *(Actos)*
- Chloropropamide *(Diabinese)*
- Tolbutamide *(Orinase)*

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• **Antidiabetic agents or oral hypoglycemic agents**

These drugs are used as adjuncts to diet to treat **non-insulin** dependent diabetes mellitus *(type 2 diabetes)* that cannot be controlled by diet alone.

Glyburide and **chloropropamide** stimulate insulin release from the pancreas. They work by reducing glucose output from the liver and by increasing insulin sensitivity at peripheral target sites.

Metformin and **pioglitazone** increase insulin sensitivity at peripheral target sites.

Tolbutamide is a **sulfonylurea** which stimulates the synthesis and release of insulin from the pancreas. It also increases the sensitivity of insulin receptors and improves the peripheral utilization of insulin.

True or False

Insulin zinc suspension, also known as lente insulin, is considered a long-acting insulin with a duration of over 24 hours after a single injection.

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• **False**

Lente insulin has a duration of 18-24 hours and is classified as an **intermediate-acting** insulin.

Insulin preparations mimic the activity of endogenous insulin, which is required for the proper utilization of glucose in normal metabolism. They are used in **type 1** diabetes and in **type 2** diabetes which cannot be controlled completely by the oral antidiabetic drugs or by diet alone.

Insulins differ in onset of action and duration of action:

- **Short-acting** insulins *(8-12 hours)* → insulin, regular
- **Intermediate-acting** insulins *(18-24 hours)* → Isophane insulin suspension *(NPH)* and insulin zinc suspension *(Lente)*
- **Long-acting** insulins *(>36 hours)* → protamine zinc insulin *(PZI)* and Ultralente insulin

Important: Hypoglycemia is the most serious and common complication of insulin therapy. Symptoms include: sweating, weakness, confusion, slurred speech and blurred vision. Administration of a concentrated glucose source will relieve mild hypoglycemia.

Humulin → is the brand name for the human form of insulin.

PHARMACOLOGY

Hypoglycemics

All of the following are **effects of insulin except:**

- Decreased gluconeogenesis
- Increased triglyceride storage
- Decreased protein synthesis
- Increased glycogen synthesis

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• **Decreased protein synthesis**

***This is **false;** insulin **increases** protein synthesis.

Insulin is a pancreatic hormone. It is secreted by the **pancreatic beta-cells of the islets of Langerhans** and is essential for the **metabolism of glucose** and for the homeostasis of blood glucose. Insulin is usually administered by subcutaneous injection. The various preparations are prepared from beef or pork pancreas and **differ primarily in their onset and duration of action.**

Insulin: Types and Activity		
	Activity *(Hours)*	
Type	**Peak**	**Duration**
Ultra-rapid acting Insulin Lispro	0.25-0.5	3-4
Rapid acting Insulin injection *(Insulin)* Prompt insulin zinc suspension *(Semilente insulin)*	0.5-3	5-7
Immediate acting NPH insulin *(isophane insulin suspension)* Lente insulin *(insulin zinc suspension)*	8-12	18-24
Long acting Ultralente insulin *(insulin zinc suspension extended)*	8-16	36

Exemestane *(Aromasin)* and letrozole *(Femara)* are anticancer drugs classified under what category listed below?

- Antibiotics
- Aromatase inhibitors
- Antimetabolites
- Alkylating agents
- Antimicrotubular

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• **Aromatase inhibitors**

Exemestane is an irreversible, steroidal aromatase inactivator. It prevents conversion of androgens to estrogens by tying up the enzyme aromatase. In breast cancers where growth is estrogen dependent, this drug will lower circulating estrogens.

Exemestane is used in the treatment of advanced cancer in post menopausal women whose disease has progressed following tamoxifen therapy.

Letrozole *(Femara)* works by a similar mechanism as above. It is used as first line treatment of hormone receptor positive or metastatic breast cancer in postmenopausal women. It is also indicated as an extended adjuvant treatment of early breast cancer in post menopausal women who have received 5 years of adjuvant tamoxifen therapy.

Humulin 70/30 is the brand name for a mixture of insulins containing: 70% isophane insulin suspension and 30% regular insulin injection. The advantage of this insulin mixture is which of the following?

- Fast onset, short duration
- Slow onset, short duration
- Fast onset, long duration
- Slow onset, long duration

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• **Fast onset, long duration**

Humulin 70/30 mixture is a popular form of insulin that many diabetic patients take. Its advantage is that after a single injection, the regular insulin component *(30%)* provides a fast onset of blood sugar control beginning one-half hour after injection and the isophane insulin suspension component *(70%)* starts acting within a couple of hours to provide a long duration of blood sugar control for 24 hours.

Note: Using either agent alone would not provide both the fast onset of insulin action along with the long duration.

Which of the following is a high molecular weight **heteropolysaccharide** that is found especially in the lungs and inactivates thrombin and other coagulation factors and thus prevents blood clotting?

- Prothrombin
- Fibrin
- Heparin
- Plasmin

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• **Heparin**

Heparin is contained within mast cells and basophils. These cells occur in connective tissue and in extracellular spaces near blood vessels, particularly the lungs.

Remember: Heparin not only neutralizes tissue thromboplastin, but also blocks thromboplastin generation.

Notes:

1. The administration of heparin will result in an increase in bleeding time due to a potentiation of antithrombin III thereby inactivating thrombin. This prevents the conversion of fibrinogen to fibrin.
2. It is used for prophylaxis and treatment of thromboembolic disorders.

Which substance is an enzyme formed in the kidney and released into the bloodstream where it has an important role in the **formation of angiotensin?**

- Plasmin
- Lysozyme
- Renin
- Heparin

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• **Renin**

Renin is **proteolytic enzyme** produced by and stored in the juxtaglomerular apparatus that surrounds each arteriole as it enters a glomerulus. Renin acts on the precursor substance **angiotensinogen**, which is manufactured by the liver and is present in the blood. Renin converts angiotensinogen to angiotensin I. **In turn**, angiotensin I is converted to angiotensin II by a converting enzyme associated with the walls of capillaries, particularly in the lungs. "The converting enzyme that converts angiotensin I to angiotensin II is known as angiotensin converting enzyme or **ACE**."

Angiotensin II is a **potent vasopressor**. It not only increases total peripheral resistance but, by stimulating **aldosterone release**, leads to an increase in plasma volume, venous return, stroke volume, and **ultimately** an increase in cardiac output.

Disulfiram *(Antabuse)* is used in the management of:

- Nicotine abuse
- Ethanol abuse
- Opioid abuse
- NSAID abuse

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• **Ethanol abuse**

Disulfiram is **not a cure** for alcoholism but is a deterrent to ethanol consumption. Disulfiram is an antioxidant that **interferes** with the hepatic oxidation of the **acetaldehyde** metabolized from alcohol. Specifically it inhibits **aldehyde dehydrogenase**, a mitochondrial enzyme found in the liver. Even the ingestion of small amounts of ethanol results in high concentrations of acetaldehyde in the body. The unpleasant reaction that occurs *(called the Disulfiram-Ethanol Reaction or DER)* consists of a throbbing headache, dyspnea, throbbing in the neck, nausea, copious vomiting, thirst, tachycardia and hypotension. **Note:** Metronidazole, also inhibits aldehyde dehydrogenase.

Ethanol is a sedative – hypnotic drug and is the most important alcohol of pharmacologic interest. Its abuse is responsible for many socioeconomic problems. Other alcohols include methanol and ethylene glycol. Drugs that are **synergistic** with ethanol include diazepam, meperidine, pentobarbital and chlorpromazine. When combined with alcohol these drugs could cause **fatal oversedation. Remember:**Synergism refers to the combined action of two or more drugs that is greater than that achieved with a single drug.

Aluminum salts used as **antacids** include:

- Hydroxide
- Carbonate
- Phosphate
- Aminoacetate
- All of the above

• **All of the above**

Aluminum hydroxide is the **most potent** of these but has less neutralizing capacity than calcium carbonate or sodium bicarbonate *(the only systemic antacid).* Gastric antacids are drugs that directly neutralize the gastric acid *(HCL)* secreted in the stomach. Antacid therapy is directed at **decreasing the concentration and total load of gastric acid.**
Note: Dyspepsia means an impairment of the power or function of digestion.
Some common over-the-counter **antacid products:**

- **Sodium bicarbonate products**
  - Alka-Seltzer
- **Calcium carbonate products:**
  - Amitone
  - Tums
- **Aluminum hydroxide products:**
  - Alterna GEL
  - Amphojel
- **Magnesium hydroxide products:**
  - Milk of magnesia
- **Bismuth salt products:**
  - Pepto-Bismol
- **Magnesium and aluminum products**
  - Maalox
  - Mylanta

Inhaled ammonia is the drug of choice for acting against:

- Anaphylaxis
- Heart attack
- Syncope
- Urticaria

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• **Syncope**

Inhaled ammonia irritates **trigeminal nerve sensory endings,** with a resulting reflex stimulation of medullary respiratory and vasomotor centers. An aromatic ammonia vapo-role is crushed between the fingers and held near the patient's nose. **Note:** The **administration of oxygen** will aid in combating tissue anoxia.

The **symptoms of syncope** include beads of sweat on the upper lip, a weak thready pulse, cold clammy skin, pallor and a dizzy feeling. The loss of normal vasomotor tonus produces pooling of blood peripherally so that the normal blood volume becomes insufficient. Placing the patient in a **supine position and elevating the feet** gives the patient a transfusion of whole blood by utilizing the forces of gravity. **Note:** The head should not be more than about 10 degrees lower than the rest of the body.

Types of syncope:

- Vasovagal
- Neurogenic
- Orthostatic

Treat with high-flowing 100% oxygen

- Hyperventilation syndrome – **Oxygen is not indicated**

Note: 100% oxygen is **contraindicated** for a person who suffers from chronic obstructive pulmonary disease *(COPD).*

Ethyl alcohol causes a **well-marked diuresis** by inhibiting the production of which hormone?

- Growth Hormone
- Insulin
- Antidiuretic Hormone *(ADH)*
- Epinephrine

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• **Antidiuretic Hormone *(ADH)*** – also called **vasopressin**

ADH is a hormone that decreases the production of urine by increasing the reabsorption of water by the renal tubules. Without ADH, there would be extreme loss of water into the urine. Ethanol *(ethyl alcohol)* **inhibits** the production of ADH.

Remember: Ethyl alcohol dilates blood vessels of the skin, depresses the CNS, and in blood levels in excess of 400 mg % usually results in coma and death. **Alcohol euphoria results** from removal of inhibitory activity if the cortex.

Note: Recent evidence has shown that frequent ingestion of moderate amounts of alcohol in any form *(beer, wine, distilled spirits)* will reduce the risk of heart disease, particularly in men.

PHARMACOLOGY

Misc.

Growth hormone is also called:

- Vasopressin
- Aldosterone
- Somatotropin
- Dopamine

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• **Somatotropin**

Growth hormone *(GH)* is produced by, and secreted from the anterior pituitary gland.

Basic Metabolic effects of growth hormone:

- Increased rate of protein synthesis in all cells of the body.
- Decreased rate of carbohydrate utilization throughout the body.
- Increased mobilization of fats and use of fat for energy.

Human growth hormone is prepared commercially and used as replacement therapy in patients with growth hormone deficiency. The commercial preparation is prepared as the purified polypeptide hormone of recombinant DNA origin with the same amino acid sequence as that produced by the pituitary gland.

Human growth hormone is indicated in children for the treatment of growth failure due to lack of adequate endogenous growth hormone secretion. It has been used in adults who have a growth hormone deficiency as a result of pituitary disease.

Human growth hormone is administered as subcutaneous injection or intramuscular injection, usually three times per week.

PHARMACOLOGY

Misc.

All of the following are useful in the **treatment of** what medical condition?

- Colchicine
- Indomethacin
- Sulfinpyrazone
- Probenecid
- Allopurinol

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• **Gout**

The therapeutic management of gout involves three different aspects of the disease:

1. Reducing the inflammation during **acute attacks:** The drug of choice is colchicine. **Colchicine** impairs leukocytic migration to inflamed areas and disrupts urate deposition and the subsequent inflammatory response. It is most effective when initiated **12 to 36** hours after symptoms begin. **Notes:** 1) Colchicine should **never be given IM** or **subcutaneously** *(it causes tissue irritation)*, 2) Colchicine can severely damage the liver and kidney; long-term therapy may cause bone marrow depression.
   - **NSAID's** are also important for the treatment of acute gouty arthritis. **Indomethacin** *(may cause renal damage or bone marrow depression)* is most commonly used.
2. Decreasing uric acid production: **Allopurinol** *(Zyloprim)* is the drug of choice in the management of chronic gout. **Inhibits xanthine oxidase**, an enzyme that converts hypoxanthine to xanthine, and xanthine to uric acid. May cause GI disturbances.
3. Enhancing uric acid clearance: Uricosuric agents include **probenecid** *(Benemid)* **and sulfinpyrazone** *(Anturane)*. These agents act primarily in the kidney *(specifically, the proximal convoluted tubules)* and inhibit the secretion of other weak acids *(i.e., penicillin)*, in addition to inhibiting the reabsorption of uric acid. **Remember:** Normally penicillins and cephalosporins have to be given in high and frequent doses due to their high rate of elimination by the kidneys. **Their excretion is slowed by giving probenecid.**

PHARMACOLOGY

Misc.

All of the following are **catecholamines except:**

- Epinephrine
- Norepinephrine
- Chlorpromazine
- Isoproterenol
- Dopamine
- Dobutamine

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• **Chlopromazine** *(Thorazine)*

Catecholamines are any one of a group of **sympathomimetic compounds** composed of a catechol molecule and the aliphatic portion of an amine. Some catecholamines are produced naturally by the body *(called endogenous)* and function as key neurological chemicals *(i.e., epinephrine, norepinephrine and dopamine)*. **Note: Epinephrine, Norepinephrine and Isoproterenol are considered to be direct-acting catecholamines.**

Sympathomimetics		
Drug (Catecholamines)	**Clinical Applications**	**Comments**
Epinephrine	Anaphylaxis, glaucoma, asthma, to cause vasoconstriction	Stimulates the myocardium
Norepinephrine	To cause vasoconstriction in hypotension	Stimulates the myocardium
Isopoterenol	Asthma	
Dopamine	Shock, heart failure	Immediate precursor of NE
Dobutamine	Shock, heart failure	
Drug (NOT Catecholamines)	**Clinical Applications**	
Amphetamine, phenmetrazine	Narcolepsy, obesity, attention deficit disorder	
Ephedrine	Urinary incontinence, to cause vasoconstriction in hypotension	
Phenylephrine	To cause mydriasis, vasoconstriction, decongestion	
Albuterol, metaproterenol, terbutaline	Asthma, premature labor	
Oxymetazoline, xylometazoilne	To cause nasal decongestion *(long acting)*	

All of the following are **skeletal muscle spasmolytic** drugs **except:**

- Methocarbamol *(Robaxin)*
- Cyclobenzaprine *(Flexeril)*
- Baclofen *(Lioresal)*
- Succinylcholine *(Anectine)*
- Carisoprodol *(Soma)*

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- **Succinylcholine *(Anectine)***

***Succinylcholine is the prototype **depolarizing neuromuscular blocking drug.**

Spasmolytic drugs *(skeletal muscle relaxants)* are agents that relieve muscle spasms without paralysis. They act in the **CNS or in the skeletal muscle cell** rather than at the neuromuscular end plate. These drugs are used in certain chronic diseases of the CNS *(i.e., multiple sclerosis, cerebral palsy, cerebrovascular accidents)* that are associated with painful muscle spasms. By reducing the spasms there is a reduction in pain and improved mobility for the patient.

Drugs used for **chronic** muscle spasm:

- **Baclofen:** is a derivative of GABA; its site of action in reducing muscle spasms is the spinal cord. Used is the treatment of multiple sclerosis and other spinal cord diseases. **Note: Diazepam** *(Valium)* and **tizanidine** *(Zanaflex)* also act in the spinal cord and are effective muscle relaxants.
- **Carisoprodol** *(Soma)* is used in the treatment of muscle spasms and pain associated with acute temporomandibular joint pain. Its precise mechanism of action is not clear but many effects have been ascribed to its central depressive action.

Drugs used for **acute** muscle spasm:

- **Cyclobenzaprine:** relieves muscle spasm through a central action, possibly at the brain stem level. It is used to relieve acute, painful musculoskeletal conditions. It is **not effective** for muscle spasm secondary to cerebral or spinal cord disease.
- **Methocarbamol:** is a centrally acting muscle relaxant that is used to relieve acute, painful musculoskeletal conditions and in the management of tetanus.

Note: Quinine is widely used for the effective relief of **nocturnal leg cramps. Carisoprodol** *(Soma)* is used in the treatment of muscle spasms and pain associated with acute temporomandibular joint pain. Its precise mechanism of action is not clear but many effects have been ascribed to its central depressive action.

All of the following drugs are used in the treatment of **Parkinson's disease except:**

- Levodopa *(Dopar)*
- Bromocriptine *(Parlodel)*
- Pergolide *(Permax)*
- Haloperidol *(Haldol)*
- Selegiline *(Eldepryl)*
- Amantadine *(Symmetrel)*

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- **Haloperidol *(Haldol)***

Parkinson's disease is a slowly progressing, degenerative disorder of the nervous system. It has several distinguishing characteristics: tremor *(shaking)* when at rest, sluggish initiation of movements and muscle rigidity. In Parkinson's disease, nerve cells in the basal ganglia degenerate, resulting in lower production of **dopamine**. It may be treated *(but not cured)* with a wide variety of drugs:

- **Levodopa** *(in combination with carbidopa)* is the precursor of dopamine. It is the **main treatment** for Parkinson's disease. It is given with carbidopa to increase effectiveness and reduce side effects.
- **Bromocriptine or pergolide** are dopamine agonists which are often given in addition to levodopa early in the treatment to enhance levodopa's action, or may be given later when levodopa's side effects become more of a problem.
- **Selegiline** is a selective inhibitor of MAO Type B, the enzyme that is responsible for the oxidative deamination of dopamine in the brain. It is used as an adjunct to levodopa.
- **Amantadine** appears to potentiate dopaminergic responses. Antiparkinsonian actions are unrelated to the antiviral effects. It is used in the early stages for mild disease.
- **Anticholinergic drugs** *(benztropine and trihexyphenidyl, certain antidepressants and antihistamines such as diphenhydramine)* may be given without levodopa in the early stages of disease, with levodopa in later stages.

All of the following are **CNS stimulants except:**

- Pentylenetetrazol *(Metrazol)*
- Doxapram *(Dopram)*
- Phenobarbital
- Nikethamide *(Coramine)*
- Picrotoxin
- Strychnine

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• **Phenobarbital**

***Phenobarbital is a **barbiturate** *(sedative-hypnotic).*

CNS stimulants are a heterogenous group of compounds that produce various degrees of stimulation. **Analeptic** is a term that refers to a CNS stimulant which has the ability to overcome drug-induced respiratory depression and hypnosis. In the past the CNS stimulants were widely used therapeutically, but today they have only limited clinical application.
Note: Their use for respiratory depression caused by **an overdose of CNS** depressants is generally **not safe or recommended.**

Analeptics and respiratory stimulants: Pentylenetetrazol, nikethamide, doxapram, picrotoxin and strychnine. These agents have limited use, but a few are occasionally indicated to stimulate respiration when a patient has pulmonary disease or to hasten recovery from a general anesthetic.

Xanthines include **caffeine, theophylline and theobromine**. These stimulants improve mental alertness, reduce the urge to sleep and elevate the mood. **Caffeine is the only approved OTC stimulant.** Theophylline and theobromine **are weaker** CNS stimulants than caffeine. Theophylline is the only xanthine important in the **treatment of asthma**. It stimulates the respiratory centers of the medulla and is able to cause bronchial dilation in patients with asthma.

Sympathomimetic amines include the amphetamines and other related agents *(i.e., methylphenidate, phenmetrazine, etc.)*. They are **potent CNS stimulants**. They are used to treat narcolepsy, obesity and attention deficit disorder.

The symptoms of chronic **caffeine consumption** can include all of the following **except:**

- Feelings of anxiety and nervousness
- Sleep disruption
- Coughing
- Irritability
- Diuresis
- Stomach complaints
- Palpitations and arrhythmias

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• **Coughing**

Caffeinism is a term used for people who are dependent upon caffeine *(i.e., suffer side effects from having too much caffeine, take larger amounts and need to keep drinking caffeine to function properly).* It is thought to occur if you have an intake of above 600 to 750 mg of caffeine per day *(more than 10 cups of coffee).* Drinking more than 1000 mg per day is well into the toxic range.

Important: Caffeine stimulates the CNS **unequally**, with the **cortex** being the most and the spinal cord being the least excited.

Caffeine Content of Commonly used Agents	
Agent **Beverages *(1 cup or glass)***	**Caffeine Content *(mg)***
Brewed Coffee	100-150
Instant Coffee	50-100
Decaffeinated Coffee	2-35
Tea	40-110
Cola Drinks	35-60
Agent ***(OTC Drugs)***	**Caffeine Content *(mg)***
Anacin	32
Excedrin	65
Nodoz	100
Vivarin	200

PHARMACOLOGY

Misc.

All of the following drugs are used to treat what **condition?**

Dexmethylphenidate *(Focalin)*
Extended-release methylphenidate *(Concerta)*
Mixed amphetamine salts *(Adderal)*
Atemoxetine *(Strattera)*
Controlled-delivery methylphenidate *(Metadate CR)*

- Insomnia in adults
- Tourette's syndrome in children
- Attention deficit hyperactivity disorder *(ADHD)*
- Insomnia in children

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• **Attention deficit hyperactivity disorder *(ADHD)***

Drugs used to treat ADHD:

Methylphenidate *(Ritalin)* → a mild central nervous system stimulant. In children with ADHD, this drug results in an increase in attention span, reduction in hyperactivity, and an improvement in behavior.

Focalin → new form of methylphenidate called dexmethylphenidate.

Concerta → a long-acting form of methylphenidate.

Adderal → the brand name for mixed amphetamine salts which act the same as methylphenidate in treating ADHD.

Strattera → the brand name for atemoxetine, the first non-stimulant approved for treating ADHD. It is approved for use in children and adults.

Metadate CR → the brand name for a controlled-delivery methylphenidate, another long-acting form of the drug.

Which of the following drugs **reduce GI motility** and are used to treat diarrhea?

- Loperamide *(Imodium)*
- Lorazepam *(Ativan)*
- Diphenoxylate and atropine *(Lomotil)*
- Propranolol *(Inderal)*

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- **Loperamide *(Imodium)***
- **Diphenoxylate and atropine *(Lomotil)***

Loperamide:

1. Is an anti-diarrheal which acts on intestinal muscles to inhibit peristalsis.
2. Is a member of the opioid family. It does not penetrate the central nervous system like the opioids such as codeine; thus it can be sold over the counter.
3. Has no evidence of drug abuse or dependence *(unlike other opioids such as codeine, morphine and meperidine)*.

Diphenoxylate is an anti-diarrheal and inhibits excessive GI tract motility and GI propulsion. Commercial preparations contain a sub-therapeutic amount of atropine to discourage abuse. **Diphenoxylate and atropine** *(Lomotil)*, unlike loperamide *(Imodium)*, requires a prescription.

Remember: Laxatives act in the reverse manner of the anti-diarrheals and increase the motility of the GI tract. They are used to treat constipation. **Examples include:** magnesium hydroxide *(Milk of Magnesia)*, castor oil, Metamucil and methylcellulose.

Which of the following are **prominent toxic effects** of mercury?

- Irritability
- Excessive saliva
- Loosened teeth
- Gum disorders
- Slurred speech
- Tremors
- All of the above

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• **All of the above**

These symptoms are all of the **chronic** form, which results from the inhalation of the vapors of dust of mercurial compounds or from repeated ingestion of very small amounts. The presence of mercury in the body is determined by a urine test. Treatment may include gastric lavage with milk and egg white or sodium bicarbonate, chelation with British anti-lewisite *(BAL)*, and fluid therapy. **Note: British Anti-Lewisite** *(BAL)* **or Dimercaprol** forms a stable complex with mercury and allows it to be excreted as an inactive compound.

Mercury that is absorbed into the circulatory system may be deposited in any tissue. Higher-than-average accumulations occur in the **brain, liver, and kidney**. Mercury does not collect irreversibly in human tissues. There is an **average half-life of 55 days** for transport through the body to the point of excretion. Thus, mercury that came into the body years ago is no longer present in the body.

All of the following are **central neurotransmitters.** Which of these elicit major excitatory effects within the CNS?

- Acetylcholine
- Dopamine
- Norepinephrine
- Serotonin
- GABA *(gamma-aminobutyric acid)*
- Opioid peptides *(beta-endorphin, enkephalins and dynorphin)*
- Glutamate and aspartate

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• **Glutamate and aspartate**

***Both of these amino acids have powerful excitatory effects on neurons in virtually every region of the CNS.

CNS Transmitters

Acetylcholine → Effects in CNS generated by interaction with a mixture of nicotinic and muscarinic receptors.

Dopamine → A catecholamine which acts through at least two subtypes D1 *(activates adenyl cyclase)*, D2 *(inhibits adenyl cyclase)*.

Norepinephrine → A catecholamine which works through alpha 1, alpha 2 and beta adrenergic receptors in CNS.

Epinephrine → a catecholamine; CNS neurons containing epinephrine have been identified but their physiological properties are unknown.

Serotonin → Is 5-hydroxytryptamine which works through at least 14 subreceptor "tryptominergic" type neurons.

GABA → The major inhibitory transmitter within the CNS.

Oral contraceptives block ovulation by **inhibiting** which anterior pituitary hormones below?

- Follicle stimulating hormone *(FSH)*
- Growth hormone *(GH)*
- Thyroid stimulating hormone *(TSH)*
- Luteinizing hormone *(LH)*
- Adrenocorticotrophic hormone *(ACTH)*

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- **Follicle stimulating hormone *(FSH)***
- **Luteinizing hormone *(LH)***

In addition to the above effects, oral contraceptives produce alterations in the genital tract, including changes in cervical mucus, rendering it unfavorable for sperm penetration even if ovulation occurs. Changes in the endometrium may also occur rendering it unfavorable for nidation *(implantation of the fertilized ovum)*.

Oral contraceptive agents usually contain **both** an estrogenic agent and a progestational agent. Estrogenic agents include ethinyl estradiol and mestranol. Some of the popular progestational agents include levonorgestrel, norethindrone, norgestimate and norgestrel.

Warnings / Precautions with Oral Contraceptives:

- The risk of cardiovascular side effects increases in women who smoke cigarettes, especially those who are **>35** years of age.
- May increase the risk of thromboembolism. Women with hypertension should be encouraged to use a non-hormonal form of contraception.

Note: Antibiotics have the potential to diminish the effectiveness of oral contraceptives. Advise patients to use additional method of birth control when taking antibiotics and oral contraceptives concurrently.

Drugs travel through the blood stream by binding to albumin plasma protein. However, only a small number of drugs travel through the blood stream by this mechanism.

- The first statement is true; the second statement is false
- The first statement is false; the second statement is true
- Both statements are true
- Both statements are false

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• **The first statement is true; the second statement is false**

***Most drugs travel through the blood stream by binding to albumin protein, which is abundant in plasma. In this way, drugs can be carried to all the tissues and organs.

A drug which is bound to plasma albumin always has some fraction which is not bound. The unbound portion is free to leave the blood compartment to be taken up by tissues where the drug will elicit its pharmacological effect. The remaining bound fraction of drug then continuously releases more free drug to be taken up by tissues. Eventually all drug in the blood compartment will be taken up by this process.

Interactions between two or more drugs can occur if they compete for binding on the plasma albumin. If drug A is bound to albumin prior to the patient taking drug B, and drug B has a greater binding affinity to albumin than drug A, then when drug B is taken, it will displace drug A from albumin to result in large amounts of unbound drug A which could lead to adverse reactions due to the sudden large amounts gaining access to the tissues.

What are **four criteria** to consider when selecting an analgesic agent for a patient?

- Type of pain
- Location of pain
- Age of patient
- Sex of patient
- Patient's weight
- Concurrent medication
- Pregnancy

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- **Type of pain**
- **Age of patient**
- **Concurrent medications**
- **Pregnancy**

Criteria considered when selecting an analgesic agent:

1. **Type of pain:**
   - Severe
   - Mild
   - Moderate
2. **Age of the patient:**
   - Infant } When calculating dosage, the height, weight, body surface area and renal
   - Child } hepatic function must be taken into account
   - Adult
   - Elderly: drug response is affected by age-related changes in physiology and pharmokinetics
3. **Concurrent medications:** Consider unwanted interactions, especially with the elderly
4. **Pregnancy:** Because virtually any drug a pregnant woman takes can cross the placenta and enter the fetal circulation, drug use in pregnant patients is a source of special concern *(check with patient's OB/GYN).*

Warfarin *(Coumadin)* and **Dicumarol** are classified as:

- Antiplatelet Agents
- Anticoagulants
- Antiarrhythmics
- Antianginal

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• **Anticoagulants**

These drugs **antagonize Vitamin K**, resulting in a decreased liver synthesis of vitamin K-dependent clotting factors *(II, VII, IX, and X).* **They prolong blood clotting times.**

They are used after a myocardial infarction to **prevent** coronary occlusion, in the treatment of pulmonary embolism, and in the treatment of venous thrombosis.

Note: Enhanced anticoagulant effects are seen when these drugs are combined with aspirin. For this reason, use acetaminophen *(Tylenol)* for pain control.

Patients on anticoagulant therapy may have excess bleeding after dental treatment. Always check medical history. If patient is on anticoagulants, his / her physician should have documentation of INR values to assess anticoagulant effects. INR stands for **International Normalized Ratio** and essentially is the ratio of the prothrombin time measured in the patient divided by a standard prothrombin time value, and multiplied by a constant. An INR value of **1** means **normal** prothrombin times of approximately 12 seconds; normal blood clotting would be present. INR values greater than 1 indicate that that there is an anticoagulant effect. The **higher the INR value, the greater the anticoagulant effect.** Many patients taking anticoagulants have INR values of 2, 3 and even up through 6.

PHARMACOLOGY

Narc Analg

Opioid drugs *(narcotics)* are used as which of the following?

- Analgesics
- Anti-inflammatories
- Antitussives
- Antidiarrheals
- Preanesthetic medications

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- **Analgesics**
- **Antitussives**
- **Antidiarrheals**
- **Preanesthetic medications**

Opiates are **very effective** analgesics. They suppress the cough reflex *(antitussive).* They cause constipation and thus are effective antidiarrheal agents. When used as pre-anesthetic medications, opiates permit a reduction in the amount of general anesthetic required for surgical anesthesia.

Some common side effects of opiates *(narcotics)* include: sedation / drowsiness, dizziness and nausea. Other less common adverse effects are vomiting, hypotension, irregular / labored breathing *(dyspnea)*, lightheadedness, nightmares and insomnia.

Respiratory depression is dose related and is the **cause of death** in narcotic drug overdose. It can happen with any of the narcotics.

Abuse **can and does occur** with all the narcotics. They are all controlled drugs falling under DEA Schedule II or DEA Schedule III of the schedules of controlled substances.

Note: A DEA number is required in order to prescribe narcotics.

Stavudine *(also known as d4T, or Zerit)* is an antiretroviral drug used in the treatment of adults with HIV infection in combination with other antiretroviral agents. Which of the following categories does this agent belong to?

- Nucleoside reverse transcriptase inhibitor
- Protease inhibitor
- Non-nucleoside reverse transcriptase inhibitor

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• **Nucleoside reverse transcriptase inhibitor**

These agents chemically are nucleosides and work by inhibiting the viral enzyme known as reverse transcriptase. This results in an inhibition of the HIV viral RNA from being made into a DNA segment; thus the genome of the HIV virus is not copied from RNA. Other agents in this class include **didanosine** *(Videx)*, **zalcitabine** *(Hivid; ddC)* and **zidovudine** *(Retrovir; AZT).*

Protease inhibitors suppress viral replication by inhibiting protease, the enzyme responsible for cleaving viral precursor peptides into infective virions. Some agents in this class include **indinavir** *(Crixivan)*, **nelfinavir** *(Viracept)*, **ritonavir** *(Norvir)*, and **saquinivir** *(Invirase).*

Nonnucleoside reverse transcriptase inhibitors inhibit the catalytic reaction of reverse transcriptase that is independent of nucleotide binding. Some agents in this class include **delavirdine** *(Rescriptor)*, **adefovir** *(Hepsera)* and **nevirapine** *(Viramune).*

Which drug **is the standard** to which all opiates *(narcotics)* are compared?

- Codeine
- Oxycodone
- Hydrocodone
- Meperidine
- Morphine
- Fentanyl

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- **Morphine**

Morphine has the following pharmacological effects:

- Analgesia
- Drowsiness
- Euphoria
- Mental clouding
- Miosis *(pupillary constriction)*
- Constipation
- Nausea and vomiting
- Respiratory depression

Remember:

1. Codeine is weaker than morphine and less addictive.
2. Oxycodone has a similar potency as morphine. In combination with acetaminophen it is known as Percocet or Tylox.
3. Hydrocodone has a similar potency as morphine. In combination with acetaminophen it is known as Vicodin and Lorcet.
4. Meperidine *(Demerol)* is weaker than morphine but just as addictive. It has a shorter duration of action. It is the only narcotic agent that does not cause miosis *(pupillary constriction)*.

PHARMACOLOGY

Narc Analg

The **major disadvantage** with the use of opioid analgesics is:

- Allergic response
- Nausea
- Vomiting
- Respiratory depression

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• **Respiratory depression**

The most significant and well-known adverse reaction to opioids is **respiratory depression**. Death secondary to opioid overdose is nearly always due to respiratory depression. When opioids are appropriately used, the risk of severe respiratory depression is generally small as tolerance rapidly develops to this effect.

Opioid Analgesics		
Drug	**Length of Effectiveness**	**Other Information**
Morphine	**Intravenous or intramuscular:** 2-3 hrs. **By mouth:** 3-4 hrs. **Sustained release:** 8-12 hrs.	Starts to work quickly. Oral form can be very effective for cancer pain.
Codeine	**By mouth:** 3-4 hrs. Taken with aspirin or acetaminophen	Less potent than morphine
Meperidine	**Iutravenous or intramuscular:** about 3 hrs. **By mouth:** not very effective	Can cause seizures, tremors, and muscle spasms
Methadone	**By mouth:** 4-6 hrs., sometimes longer	Also used for treating heroin withdrawal
Propoxyphene	**By mouth:** 3-4 hrs.	Generally taken with aspirin or acetaminophen to treat mild pain
Levorphanol	**Intravenous or intramuscular:** 4 hrs. **By mouth:** about 4 hrs.	Oral form is strong. Can be used instead of morphine.
Hydrocodone	**By mouth:** 4-6 hrs.	Usually combined with acetaminophen
Oxycodone	**By mouth:** 3-4 hrs.	Usually combined with aspirin or acetaminophen
Pentazocine	**By mouth:** up to 4 hrs.	Can block painkilling action of other opioids. About as strong as codeine. Can cause confusion and anxiety, especially in the elderly.

Which is the **most frequently** utilized route of administration for sedation in pediatric patients?

- Oral
- Inhalation
- IV
- IM

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• **Inhalation**

***The agent used most frequently is **nitrous oxide.**

Nitrous oxide is a slight sweet smelling, colorless, inert gas. It must always be coupled with **no less than 20% oxygen.** Nitrous oxide is quickly absorbed from the lungs and is physically dissolved in the blood. There is no biotransformation, and the gas is rapidly excreted by the lungs when the concentration gradient is reversed. It is recommended that the patient be maintained on oxygen for 5 to 10 minutes after the sedation period.

Dose response for nitrous oxide: *(always given in mixture with oxygen)*
- **10% - 20%** → tingling of hands, feet, body warmth
- **20% - 40%** → mild sleepiness, relaxation, some analgesia, mind dissociation, heightened auditory perception
- **Above 50%** → this is too much nitrous → nausea, sweating

***Rapid onset → 5 minutes
***Rapid recovery → 5 minutes

Note: Nitrous oxide **does not** provide enough analgesic effect to preclude the use of local anesthesia in dental surgery or restorative procedures. Local anesthetics **must be used** along with nitrous oxide.

All of the following are **opium alkaloids, except:**

- Meperidine
- Morphine
- Codeine

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- **Meperidine**

Chemical classification of opioid analgesics:

Opium alkaloids:
- Morphine
- Codeine

Synthetic derivatives:

Morphine group
- Hydromorphone *(Dilaudid)*
- Oxymorphone *(Numorphan)*
- Nalbuphine *(Nubain)*

Codeine group
- Hydrocodone *(in Vicodin)*
- Oxycodone *(in Percodan, Percocet and Tylox)*

Synthetic narcotics:

Meperidine group
- Meperidine *(Demerol)*
- Alphaprodine *(Nisentil)*
- Alfentanil *(Alfenta)*
- Fentanyl *(Sublimaze)*
- Sufentanil *(Sufenta)*
- Diphenoxylate *(in Lomotil)*
- Loperamide *(in Imodium)*

Methadone group
- Methadone *(Dolophine)*
- Propoxyphene *(Darvon)*

Which of the following are **advantages** of using nitrous oxide analgesia?

- Rapid onset of action
- Elevates pain threshold
- Produces euphoria
- Pleasant induction
- Titratable
- Rapid and complete recovery
- Virtually no adverse effects in absence of hypoxia
- Therapeutic sedative for many medically compromised patients
- Suitable for all ages
- All of the above

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- **All of the above**

Important points about nitrous oxide:

- The **main** therapeutic effect of nitrous oxide is relaxation / sedation. Mild analgesia is a secondary effect.
- The **first symptom** of nitrous oxide onset is **tingling of the hands**.
- Nitrous oxide has **no local** anesthetic properties. Therefore, the addition of local anesthesia is necessary in procedures in which pain is anticipated.
- Long term exposure to low doses of nitrous oxide has been shown to increase the incidence of spontaneous abortions. Environmental contamination by nitrous oxide can be kept to a minimum by employing a scavenger system.
- **Nitrous oxide** is stored under pressure in steel cylinders painted blue. **Oxygen** is stored in green tanks.
- Nitrous oxide delivery machines come pre-equipped with a failsafe mechanism that will not allow less than 20% oxygen to be delivered to the patient.

PHARMACOLOGY

Narc Analg

Which drug is the **prototype opioid antagonist?**

- Morphine
- Naloxone
- Propranolol
- Ibuprofen

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- **Naloxone** *(Narcan)*

Naloxone *(Narcan)* is a **narcotic antagonist** and is used in medical emergencies to reverse narcotic overdose. Overdose of narcotics results in respiratory depression and death due to respiratory shut down. Naloxone will reverse the respiratory depressant effects of the narcotics thus counteracting the lethal effects of these agents. Naloxone is given intravenously, intramuscularly or subcutaneously.

Nalmefene *(Revex)* and **naltrexone** *(ReVia)* are the other two **narcotic reversal** agents used to reverse the respiratory depressive effects of the narcotic analgesics.

Typical narcotic analgesic drugs which **are reversed by** naloxone *(Narcan)*, nalmefene *(Revex)*, and naltrexone *(ReVia)* include:

- Codeine
- Morphine
- Hydrocodone
- Oxycodone
- Meperidine *(Demerol)*
- Fentanyl

Note: Naltrexone *(ReVia)* is also used to treat alcohol dependence.

The central nervous system contains **three types** of endogenous opioids. Which one of the following **is not** one of them?

- Beta-endorphins
- Morphine
- Enkephalins
- Dynorphins

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• **Morphine**

Opioid receptors in the central nervous system are thought to be activated by endogenous chemicals under physiologic conditions. The body contains three types of these chemicals that produce **morphine-like effects** *(reduce pain):*

- **Beta-endorphins** bind to opioid receptors in the brain and have potent analgesic activity.
- **Enkephalins** bind to opioid receptors in the brain and are more widely distributed in the brain than the beta-endorphins. Seem to play a role in pain perception, movement, mood, and behavior.
- **Dynorphins** are the **most powerful** of these chemicals and are found throughout the central and peripheral nervous systems. Some research supports the theory that they regulate pain at the spinal cord level, influence feeding behavior at the hypothalamic level and function with other endogenous opioids to regulate the cardiovascular system.

Opioid receptors:

1. mu *(μ)* – the prototypical opioid agonist for this receptor is **morphine**, and its analgesic activity is considered to depend on its binding to this receptor.
2. delta *(δ)* – the **enkephalins** are considered to be the typical agonist for this receptor.
3. kappa (κ) – the **dynorphins** are thought to be the typical agonist for this receptor.

***Opioid analgesics** *(i.e., morphine, codeine, meperidine, propoxyphene, etc.)* mimic endogenous opioids at CNS opiate receptors, **raising the pain threshold and increasing pain tolerance.**

All of the following sites are generally accepted for **IM injections, except:**

- The buttocks
- The biceps muscle
- The deltoid muscle
- The anterior thigh

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• **The biceps muscle**

***For **young children**, the anterior thigh is usually the place to give IM injections.

Absorption from an intramuscular injection is often faster and there is a higher bioavailability than with oral administration.

Proper depth of needle in muscle: In big muscle *(adult)* go in **one inch;** in children go in **one-quarter of an inch. Never** go beyond two-thirds of the needle length.

PHARMACOLOGY

Trms / Def

Which term refers to a drug that **binds to physiological receptors** to result in specific cellular effects producing a predictable pharmacologic response?

- Pharmacologic agonist
- Pharmacologic antagonist

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• **Pharmacologic agonist**

Drugs that bind to physiologic receptors and mimic the regulatory effects of endogenous signaling compounds will produce a pharmacologic effect as a result of binding to the receptor. A drug that elicits a full response through this process is referred to as a pharmacologic agonist.

A **partial agonist** is a drug which acts on the physiologic receptor but elicits an effect which is **only partly as effective** as an agonist drug.

An **antagonist** is a drug which binds to the physiologic receptor **but does not** trigger an effect. When antagonist is present, the agonist drug cannot reach the receptor site to produce an effect.

1. **Competitive antagonism** occurs when a response **can be achieved** by increasing the dose of agonist in the presence of antagonist.
2. **Noncompetitive antagonism** occurs when a response **cannot be achieved** with increasing doses of agonist in the presence of antagonist.

PHARMACOLOGY

Anesth

All the following are considered to be what **type of anesthetic?**

- Ether *(diethyl ether)*
- Halothane
- Enflurane
- Isoflurane
- Sevoflurane
- Methoxyflurane
- Desflurane

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• **Inhalational anesthetics**

Inhalation anesthetics are drugs which are vaporized from the liquid form and inhaled to produce general anesthesia. These diverse drugs are relatively simple **lipophilic molecules** ranging from ethers such as diethyl ether, halogenated hydrocarbons such as halothane and halogenated ethers such as isoflurane, enflurane, sevoflurane, methoxyflurane, and desflurane.

Vaporization of these liquids occurs in a vaporizer and delivery to the patient occurs through an anesthesia machine via a nasal mask. Although it was the first general anesthetic discovered in 1846, diethyl ether is no longer used in anesthesia because of its explosive nature and slow onset time.

All of the following are **basic types of binding** that may be involved when a drug interacts with its receptor **except:**

- Ionic bonding
- Nitrogen bonding
- Hydrogen bonding
- Van der Waals forces
- Hydrophobic interactions

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• **Nitrogen bonding**

Implicit in the interaction of a drug with its receptor is the chemical bonding of that drug to one or more specific sites on the receptor molecule. There are four basic types of binding that may be involved:

1. **Ionic bonds** – these result from the **electrostatic** attraction between ions of opposite charge.
2. **Hydrogen bonds** – represent a special type of interaction between polar molecules.
3. **Van der Waals forces** – collectively describe the weak interactions that develop when two atoms are placed in close proximity.
4. **Hydrophobic interactions** – hydrophobic interactions between the drug, its receptor and the aqueous environment can play a major role in stabilizing drug-receptor binding.

Note: Covalent bonds arise from the sharing of electrons by a pair of atoms. Although covalent bonds are required for the structural integrity of molecules, **they are generally not involved in drug-receptor interactions.**

Which of the following routes of drug administration is generally used for **local drug effects?**

- Topical administration
- Oral ingestion
- Subcutaneous injection
- Intravenous injection

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• **Topical administration**

Common routes of **administration** of drugs:

1. **Parenteral administration** (**not** by way of intestine or GI tract)
   - **Intravenous** *(IV)* – directly into the bloodstream. Acts very rapidly
   - **Intra-arterial** – injected into a specific artery. Be careful, it burns
   - **Intramuscular** *(IM)* – injected into a muscle area, where it is promptly absorbed
   - **Subcutaneous** – injection beneath the skin. **Absorption may be less rapid**
2. **Enteral administration** *(by way of the intestine or GI tract)*
   - **Buccal or sublingual** – a tablet is placed under the tongue or in the cheek
   - **Oral** – *(most common route)*, the drug is swallowed. It is the most convenient for safe drug administration. **It is safe, painless and economical.**
   - **Rectal** – the drug in solution *(enema)* or suppository form is inserted into the rectum
3. **Inhalation** – the drug is given as an aerosol into the respiratory tract
4. **Topical administration** – the drug is placed on the skin for a **local effect**
5. **Transdermal** – the drug is placed on the skin for a **systemic effect**

Remember:

1. The **onset of action** of a drug is determined primarily by the **rate of absorption.**
2. The **major effect** of a drug is a factor of how much of the drug is **free in plasma.**

The most important enzyme systems for the biotransformation of drug molecules are found in:

- Lungs
- Liver
- Brain
- Kidneys
- Gastrointestinal tract

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• **Liver**

Hepatic metabolism of drugs occurs in Phase I reactions catalyzed by a microsomal mixed-function oxidase system *(also known as the P-450 system)* and in phase II reactions known as conjugation reactions.

Phase I reactions — occur in the liver microsomal enzyme system *(mixed-function oxidase system or P-450 system)*. In this system, drug metabolism occurs in three basic patterns. **First**, the active parent drug can be converted to the inactive metabolite. **Second**, an active parent drug may be converted to a second active compound which is subsequently converted to an inactive compound; and **thirdly**, an inactive parent drug may be transformed to an active compound.

The most common reaction in drug metabolism is an oxidation reaction in which oxygen in the form of hydroxyl group is attached to the drug molecule.

There are at least five distinct groups of microsomal drug metabolizing enzymes: These enzymes "families" are identified as a cytochrome *(CYP prefix)* followed by their numerical digestion *(e.g., 1A2)*. Thus the enzyme CYP 1A2 is a distinct drug metabolizing enzyme that converts a variety of drugs to the oxidized product.

Phase II reactions — Conjugation reactions involve coupling the drug with an acid present in cells *(usually glucuronic acid)*. When coupled to glucuronic acid, the process is known as glucuronide conjugation with the resulting metabolite referred to as the "glucuronide". Conjugations occur in the liver, kidney and to a lesser extent in other tissues.

Conjugation of drugs results in polar, water-soluble compounds that are rapidly excreted in urine. Thus, the parent drug is effectively rendered inactive and transported out of the body by this process.

True or false

Implicit in the interaction of a drug with its physiological receptor is that the drug does r create effects, **but rather**, modulates intrinsic physiological cell functions.

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• **True**

There are four major families of **physiological receptors** that drugs can bind to produce effects:

Receptors as enzymes *(i.e., cell surface protein kinases)* → These kinases exert their regulatory effects by phosphorylating proteins within the cell which alters the cellular biochemical activities. By binding to these kinases, drugs can also cause the alteration in biochemical activities resulting in a drug effect.

Ion channels → Drugs can bind to ion channels in cell membranes to cause opening or closing. This alters the cell's membrane potential to result in a drug effect.

G protein-coupled receptors → When drug binds to these receptors, second messengers are produced such as cyclic AMP to produce an effect within the cell. This results in a drug effect.

Receptors in cell nucleus → Receptors for steroid hormones are soluble DNA-transcription factors within the nucleus that regulate the transcription of specific genes. Modification of the transcriptions of these genes results in a drug effect.

The **safest and easiest** route for drug administration is:

- Oral
- IV
- Rectal
- Inhalation

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• **Oral** ***(abbreviated P.O.)*

However, it is also the **most unpredictable and least effective** route available. Drugs taken by mouth have to be absorbed *(usually from the small intestine)* before they can be transported to their site of action. Absorption may be slow, unpredictable and irregular due to the presence of variable amounts of food in different stages of digestion and to the varying degrees of acidity and alkalinity of the digestive juices. **Moreover**, blood from the intestinal tract passes first to the liver: some drugs are metabolized in the liver and others may be stored there to be released only slowly. These considerations make it clear that oral administration is usually **unsuitable in emergencies** or on other occasions when a rapid effect is needed.

Note: A major advantage of IV administration of a drug is it allows for titration of the drug.

Other advantages of IV administration include:

- Rapid onset
- Drugs that cause irritation when administered subcutaneously can be given IV with no irritation
- In case of emergency, there is an open line through which emergency drugs can be injected

One major **disadvantage** of IV injection is that since it has such a rapid onset of action, overdosage may have effects so immediate that it is impossible to reverse them.

What serious dental effect is associated with the following drugs: zolendronic acid *(Zometa)*; palmidronate *(Aredia)*; alendronate *(Fosamax)*?

- Mucositis
- Osteonecrosis of the jaw bone
- Angular cheilitis
- Oral yeast infection
- Xerostomia

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• **Osteonecrosis of the jaw bone**

Zoledronic acid *(Zometa)*, **palmidronate** *(Aredia)*, and **alendronate** *(Fosamax)* are members of the bisphosphonate class of drugs used to treat and manage osteoporosis and to prevent hypercalcemia of malignancy. Bisphoshonate therapy has been associated with osteonecrosis, primarily of the jaw; this has been observed mostly in cancer patients, but also in patients with postmenopausal osteoporosis and other diagnoses. Risk factors include a diagnosis of cancer, with concomitant chemotherapy, radiotherapy or corticosteroids; anemia, coagulopathy, infection or pre-existing dental disease. Symptoms included nonhealing extraction socket or an exposed jawbone. There is no data addressing whether discontinuation of therapy reduces the risk of developing osteonecrosis. However, as a precautionary measure, dental exams and preventative dentistry should be performed prior to placing patients with risk factors on chronic bisphosphonate therapy. Invasive dental procedures should be avoided during treatment.

The **physiochemical properties** of drugs that influence their passage across biologic membranes are:

- Lipid solubility
- Degree of ionization
- Molecular size and shape
- *All of the above*

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• **All of the above**

The **mechanism of drug transfer** across biological membranes is by:

1. **Passive transfer** *(is essential to various processes of metabolism)*
   - **Simple diffusion**: lipid-soluble substances move across the lipoprotein membrane by this process. The majority of drugs penetrate biomembranes by this process through membrane phospholipids. The amount of drug dissolving in the membrane at any time is directly proportional to the concentration gradient and its degree of lipid solubility *(***Note:** *Only nonionized drugs are soluble in lipid).*
   - **Filtration**: water-soluble molecules small enough to pass through membrane channels may be carried through the pores by the bulk flow of water. Drugs of molecular weights of 60,000 or less can "filter" through capillary membranes.
2. **Specialized transport**
   - **Active transport**: lipid-insoluble substances *(for example glucose)* are shuttled across plasma membranes by forming complexes with specific membrane constituents called **carriers**. These carrier molecules within a cell furnish **energy** for transportation of the drug to regions of higher concentration. **Facilitated diffusion** is the term given to carrier-based transfer when the driving force is simply the concentration difference of the drug across the membrane.

Note: Osmosis is the movement of a pure solvent, as water, through a semipermeable membrane from a solution that has a lower solute concentration to one that has a higher concentration. **The membrane is impermeable to the solute but is permeable to the solvent.**

A **teaspoon** can hold what volume of liquid?

- 5 milliliters
- 10 milliliters
- 15 milliliters
- 20 milliliters

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- **5 milliliters**

Approximate Household Measures and Weights					
Teaspoons	**Tablespoons**	**Cup or Glasses**	**Fluid Ounces**	**Milliliters**	**Grams**
1			0.125	5	5
3	**1**		0.05	15	15
48	16	1	8	237	240

All of the following drugs are known to produce **orthostatic hypotension** as an adverse reaction **except:**

- Levodopa *(Larodopa, Dopar)*
- Prazosin *(Minipress)*
- Chlorpromazine *(Thorazine)*
- Indomethacin

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- **Indomethacin**

***Indomethacin *(Indocin)* is an **NSAID** which may cause GI bleeding, ulcers and possible stomach perforation.

Drugs that may produce **orthostatic hypotension:**

- **Antihypertensives**: for example guanethidine *(Ismelin)*
- **Phenothiazines**: for example chlorpromazine *(Thorazine)* and thioridazine *(Mellaril)*
- **Tricyclic antidepressants**: for example doxepin *(Sinequan)*, amitriptyline *(Elavil)* and imipramine *(Tofranil)*
- **Narcotics**: for example meperidine *(Demerol)* and morphine
- **Antiparkinson drugs**: for example levodopa *(Larodopa, Dopar)* and carbidopa + levodopa *(Sinemet)*

Orthostatic hypotension *(also called postural hypotension)* is abnormally low blood pressure occurring when an individual assumes the standing posture.
Important: Following vasovagal syncope, orthostatic hypotension is the most likely cause of transient unconsciousness in the dental office. Many factors have been identified that may be responsible for the development of orthostatic hypotension, including several which are important to the practice of dentistry. They include the administration and ingestion of drugs, prolonged recumbency and convalescence, an inadequate postural reflex, pregnancy, various defects in the legs, Addison's disease, physical exhaustion and starvation and chronic orthostatic hypotension *(Shy-Drager syndrome)*. The incidence increases with age.

Pain that has **no organic basis** and is fixed upon some portion of the anatomy is referred to as:

- Intractable pain
- Referred pain
- Psychogenic pain
- Phantom pain

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- **Phantom pain**

***For example**, the sensation of pain felt in a limb, although that limb has been amputated.

Other **terms** to know:

- **Intractable pain** is pain that is **resistant** or refractory to ordinary analgesic agents
- **Referred pain** is pain felt in an area **other than** the site of origin, such as pain near the shoulder associated with biliary disease
- **Psychogenic pain** is pain produced or caused by **psychic** or mental factors rather than organic factors

Remember: Pain threshold refers to the **lowest level of pain a patient will detect.**

Which schedule of drugs would include a drug that is considered to have a **strong potential for abuse or addiction**, but which has **legitimate medical use?**

- Schedule I
- Schedule II
- Schedule III
- Schedule IV
- Schedule V

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• **Schedule II**

Schedule I – a category of drugs **not considered legitimate for medicinal use.** Among the substances so classified by the DEA are mescaline, LSD, heroin, and marijuana. Special licensing procedures must be followed to use these or other Schedule I substances.

Schedule II – a category of drugs considered to have a strong potential for abuse or addiction, **but which have legitimate medical use**. Among the substances so classified by the DEA are Morphine, Cocaine, Pentobarbital, Oxycodone, Methadone, and straight Codeine.

Schedule III – a category of drugs considered to have less potential for abuse or addiction than Schedule I or II drugs. Among the substances so classified by the DEA are various analgesic combination compounds containing codeine *(i.e., acetaminophen and codeine →Tylenol #3)* and various analgesic combination compounds containing hydrocodone *(i.e., hydrocodone and acetaminophen →Vicodin; Lorcet).*

Schedule IV – a category of drugs that have less potential for abuse or addiction than those of Schedules I to III. Among the substances so classified by the DEA are diazepam *(Valium),* lorazepam *(Ativan),* triazolam *(Halcion),* alprazolam *(Xanax),* and chloral hydrate.

Schedule V – a category of drugs that have a small potential for abuse or addiction. Among the substances so classified by the DEA are many commonly prescribed medications that contain a small amount of Codeine.

Note: Schedule II and III must have a written prescription signed by the health professional *(laws vary from state to state).* The FDA determines which drugs are to be sold by prescription only. The prescription must have the address of the patient and dentist as well as the DEA number of the dentist.

A drug with a high LD_{50} and a low ED_{50} has a:

- High therapeutic index and is, therefore, very dangerous
- High therapeutic index and is, therefore, relatively safe
- Low therapeutic index and is, therefore, very dangerous
- Low therapeutic index and is, therefore, relatively safe

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• **High therapeutic index and is, therefore, relatively safe**

Explanation: The purpose of an acute toxicity test is to determine the nature and extent of the untoward reactions which might follow the administration of a single dose *(or an overdose)* of a drug. A quantitative aspect of acute toxicity testing is the determination of the drug's lethal dose. This is usually expressed as the LD50. Standing alone, it conveys less information than does the ratio of the lethal to the effective doses **(LD_{50} / ED_{50})**, a quantity which is often known as the therapeutic index. The greater a drug's **therapeutic index**, the less likely that fatalities will follow an accidental overdose.

Notes:

1. **ED_{50}** is the effective dose at which 50% of people will respond.
2. **LD_{50}** is the lethal dose at which, in theory, 50% of people will die ***Lethal doses are always determined in mice, not people.

In the ideal situation, the therapeutic index would be 100 *(this never happens)*.

Which route of administration of a drug listed below is most known for its significant hepatic **"first pass"** metabolism?

- Intramuscular
- Inhalation
- Sublingual
- Oral

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• **Oral**

The oral administration of a drug is the one most acceptable to the patient. It is convenient because drugs can be given in the form of tablets or capsules which contain an exact dose. It is easy and the patient can take the drug without help from anyone else.

One of the disadvantages of drugs taken by mouth is that they have to be absorbed *(usually from the small intestine)* before they can be transported to their site of action. Blood from the intestinal tract **passes first to the liver:** some drugs are metabolized in the liver *("first-pass effect")* and others may be stored there to be released slowly. **This consideration makes it clear that oral administration in usually unsuitable in emergencies or on other occasions when a rapid effect is needed.**

Intramuscular injection is an injection made into a large muscle. The advantages of IM injection are that it results in **uniform absorption** and that it can be used for solutions too irritant for subcutaneous injection. The speed of absorption of drugs given by IM injection depends on the vehicle in which they are dissolved: **absorption is rapid from aqueous solutions and slow from oily solutions.**

The term **bioavailability** of a drug refers to:

- The movement of a drug into the body tissues over time
- The dissolution of a drug in the GI tract
- The measurement of the rate and amount of therapeutically active drug that reaches the systemic circulation
- The relationship between the physical and chemical properties of a drug and the systemic absorption of the drug
- The amount of drug destroyed by the liver prior to systemic absorption from the GI tract

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- **The measurement of the rate and amount of therapeutically active drug that reaches the systemic circulation**

The bioavailability of a drug is affected by:

- The dissolution of a drug in the GI tract
- The destruction of a drug by the liver

Remember: IV injection provides complete *(100%)* bioavailability

Note: After oral administration, drugs will generally be **absorbed best from the duodenum.** The duodenum has a large surface area due to the presence of villi and microvilli.

Which term is a **compulsive, uncontrollable dependence** on a substance, habit or practice to such a degree that cessation causes severe emotional, mental or physiological reactions?

- Habituation
- Tolerance
- Addiction
- None of the above

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• **Addiction**

Habituation is an acquired tolerance from repeated exposure to a particular stimulus. Psychological and emotional dependence on a drug, tobacco or alcohol result from the repeated use of the substance **but without the addictive, physiological need to increase dosage.**

Tolerance is the phenomenon of **decreased responsiveness to a drug** following chronic administration.

Note: Physiological dependence is common to all forms of drug dependence and abuse. These drugs of abuse all have the ability to change one's mood and sensory perception.

PHARMACOLOGY

Trms / Def

Which route of drug administration will give the **most rapid onset** of pharmacological effect?

- Oral administration
- Subcutaneous injection
- Intravenous injection
- Intramuscular injection

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• **Intravenous injection**

When a drug is given intravenously, it is placed directly into the systemic circulation. **The drug is delivered rapidly to all tissues,** including the drug receptor sites. For all other routes of drug administration *(with the exception of intra-arterial injection)*, the drug must be systemically absorbed prior to distribution to the drug receptor sites, and therefore the onset of pharmacological effects is slower.

Also, when a drug is given by IV injection there is a complete *(100%)* **bioavailability**. The entire dose is placed into the systemic circulation. With other routes of administration, the drug may be lost prior to reaching the systemic circulation. **For example,** with first-pass effects, a portion of an orally administered drug is eliminated, usually through degradation by liver enzymes, before the drug reaches its receptor sites.

Remember: The initial distribution of a drug into the tissues is determined chiefly by the **rate of blood flow to the tissue,** whereas drug affinity for the tissue will determine whether the drug will concentrate at that site. **Note: Gastric emptying time and degree of plasma protein binding** also have an effect on drug distribution but are **less important than the rate of blood flow to the tissues.**

When a drug is administered **repeatedly**, a higher concentration of the drug than is desired may be achieved. The **effect of this excessive accumulation** is known as:

- Additive effect
- Synergistic response
- Cumulative action
- Idiosyncrasy

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• **Cumulative action**

An additive effect occurs when additive drugs are administered. The response is no greater than that which would be expected had the drugs been given one at a time. There is no enhancement of potential of the individual drugs as a result of being used in combination.

A synergistic response occurs when the combined action of two drugs is greater than the sum of their individual actions. **Examples of synergism:** Alcohol is synergistic with diazepam *(Valium)*, narcotics, barbiturates and phenothiazines. It should be avoided if taking these drugs.

Idiosyncrasy is a response to a drug that is unusual or abnormal or one that grossly deviates from the routine reaction.

List the **three** reasons why **epinephrine** is included in dental local anesthetics.

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Epinephrine, a **vasoconstrictor**, is included in dental local anesthetic preparations for the following three reasons:

1. It prolongs the duration of local anesthesia.

2. To provide a hemostasis such that local bleeding is controlled or reduced.

3. To delay the absorption of anesthetic into the systemic circulation thus reducing the chance of systemic toxicities.

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The Controlled Substance Act of 1970 uses which criteria for inclusion of a drug into one of the five schedules?

- Potential for **abuse**
- Medical **usefulness**
- Degree to which it produces **physiological dependence**
- Degree to which it produces **physical dependence**
- All of the above

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• **All of the above**

1. Potential for abuse *(most important)*
2. Medical usefulness
3. Degree to which it produces physiological dependence
4. Degree to which it produces physical dependence

Remember: Schedule I drugs *(LSD, heroin, etc.)* cannot be prescribed and are made available only for specific approved research projects. Schedule II drugs *(amphetamines, morphine, codeine, oxycodone etc.)* can be prescribed but can not be refilled. A new prescription must be written for refills. Prescriptions for Schedule II drugs cannot be called into the pharmacy over the telephone. Schedule III drugs *(hydrocodone with acetaminophen, codeine with acetaminophen, etc.)* may be called in to the pharmacy over the telephone. The prescriber can authorize refills without need of a new written prescription.

***The prescriber must have a Drug Enforcement Agency **authorization number** *(DEA #)* in order to prescribe scheduled drugs.

PHARMACOLOGY

Trms / Def

Which of the following refers to the **efficacy** of a drug?

- The relative concentrations of two or more drugs that produce the same drug effect
- The **ability of a drug** to produce a desired therapeutic effect **regardless of dosage**
- The dose of a drug that will **kill a patient**

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• **The ability of a drug to produce a desired therapeutic effect regardless of dosage**

***Efficacy** is also referred to as **"intrinsic ability", maximal, or "ceiling effect".** It is the maximum effect a drug can bring about, regardless of dose.

Potency is the relative concentrations of two or more drugs that produce the same drug effect. The effect usually chosen is 50% of the maximal effect and the dose causing this effect is called the EC50. Potency is determined mainly by the affinity of the receptor for the drug. **Note:** The smaller the EC50, the greater the potency of the drug. Potency is a **comparative term** *(one drug is more potent than another drug).*

Example: Drug #1 in a dose of 10 mg produces the same magnitude of response as Drug #2 in a dose of 50 mg. The following is true: **Drug #1 is five times as potent as Drug #2 but Drug #1 is not potent in and of itself.**

Also: if Drug #1 has a **greater efficacy** than Drug #2, then Drug #1 is **capable of producing a greater maximum effect** than Drug #2.

When comparing drugs with respect to intensity of response, the drug that produces the **greatest maximum effect** is the one with the **highest efficacy.**

Name the **four stages** of general anesthesia.

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1. **Amnesia / analgesia**
2. **Delerium**: begins with unconsciousness and ends with loss of eyelid reflex, purposeless movements and hyper-reaction, dilated pupils, reflex vomiting, tachycardia, and hypertension
3. **Surgical**: four planes
4. **Medullary paralysis**: cessation of respiration, ending with death without proper treatment

Note: These four stages of anesthesia apply to the **inhalants** and not to the intravenous general anesthetics.

Agents used for **general anesthesia:**

- **Inhalation agents**: volatile liquids used as inhalants → desflurane, sevoflurane, halothane, isoflurane and enflurane.

Note: Nitrous oxide is **not considered** a general anesthetic since hypoxic levels are required to produce anesthesia. It is considered a sedative. It is used alone to produce sedation or in combination with the above agents to supplement the anesthetic response. It is a gas at room temperature and pressure.

- **Intravenous agents**: **barbiturates** → thiopental *(Pentothal)* and methohexital *(Brevital)*; others include: etomidate, propofol *(Diprivan)*, ketamine *(Ketalar)*; **benzodiazepines** → diazepam *(Valium)*, midazolam *(Versed)*, lorazepam *(Ativan)*; neuroleptic-opioid combinations which are called **neuroroleptanalgesics** combine fentanyl and droperidol.

True or false

Nitrous oxide is used as a single agent to produce **general** anesthesia.

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• **False**

Nitrous oxide is **unable** to produce general anesthesia except if it is given at concentrations greater than 80%. At these concentrations, the lack of oxygen would cause hypoxia in the patient. Inhalant anesthetics such as halothane and isoflurane can produce general anesthesia at concentrations approximating 3-5%. As such they are very useful in anesthesia.

Nitrous is used to produce sedation and mild analgesia. It is usually used in concentrations of 30-50% along with pure oxygen. It is a colorless, nonirritating gas at room temperature and pressure, and is not flammable nor explosive. The onset of sedation is within 5 minutes and the recovery is just as rapid. It is excreted unchanged by the lungs. The most common complaint from patients taking nitrous oxide is mild nausea. Nitrous oxide is **contraindicated** in patients with upper respiratory infections, emphysema, bronchitis, and in the first trimester of pregnancy. It is **also contraindicated** in patients with whom communication is difficult *(i.e., autistic patients)*. It should never be used on patients having a contagious disease since it is difficult to sterilize entire tubes.

Note: Always give patient 100% oxygen at the end of the procedure to prevent diffusion hypoxia.

Which component of a lidocaine **local anesthetic** solution causes an allergy?

- Water
- Bisulfites
- Lidocaine
- Epinephrine *(vasoconstrictor)*

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• **Bisulfites**

Patients may exhibit hypersensitivity to sulfites contained in some anesthetic solutions. Sodium metabisulfite prevents the oxidation *(deterioration)* of the epinephrine vasoconstrictor in those commercial preparations containing epinephrine. Most of the patients reacting to bisulfites have a history of asthma and the airway is hyperactive to the sulfites. Allergic reaction usually results in an asthmatic syndrome of wheezing and bronchial constriction. Bisulfites are present in only those commercial preparations containing vasoconstrictor. Preparations **without** vasoconstrictor such as mepivacaine 3% *(Carbocaine 3%)* **do not contain** bisulfites.

Note: Hypersensitivity or allergic reactions to local anesthetics, particularly the amides, are much more rare than allergic reactions to the bisulfites.

Of the **amide-type** local anesthetics, which is the **only one** that is metabolized in the bloodstream rather than the liver?

- Articaine *(Septocaine, Zorcaine)*
- Mepivacaine *(Carbocaine)*
- Lidocaine *(Xylocaine)*
- Prilocaine *(Citanest)*
- Bupivacaine *(Marcaine)*

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• **Articaine** *(Septocaine, Zorcaine)*

Articaine is an **amide-type** local anesthetic. However, it is chemically unique in that it has an ester group attached to its molecule which can be acted upon by plasma cholinesterase to render it ineffective. Therefore, it is the only amide which is metabolized in the bloodstream and not the liver.

Articaine *(Septocaine, Zorcaine)* is supplied as articaine HCL 4% solution with epinephrine 1:100,000. It is indicated for local, infiltrative, or conductive anesthesia in both simple and complex dental and periodontal procedures. The onset of anesthesia following administration of articaine *(Septocaine, Zorcaine)* has been shown to be 1 to 6 minutes after injection. Complete anesthesia lasts approximately 1 hour. Articaine *(Septocaine, Zorcaine)* is **contraindicated** in patients with hypersensitivity to local anesthetics of the amide type or to sodium bisulfite.

Which of the following is a **common adverse effect** caused by the use of tetracyclines?

- Photosensitivity
- Nausea
- Diarrhea
- Discoloration of teeth and enamel hypoplasia in young children
- All of the above

• **All of the above**

Another common adverse effect is the occurrence of **opportunistic** *(superinfection)* infections caused by Candida albicans. This is due to the wide spectrum of antibacterial action which alters normal flora. Examples include both vaginal and oral candidiasis. These conditions are also known as vaginal and oral yeast infections.

Members of the tetracycline family of antibiotics include:

- Tetracycline *(generic)*
- Doxycycline *(Vibramycin)*
- Minocycline *(Minocin)*
- Demeclocycline *(Declomycin)*

Remember: The usefulness of the tetracyclines in the treatment of odontogenic infections is limited since they can cause "yeast" infections very easily. They have been used as alternatives to penicillin in patients with **ANUG** *(acute necrotizing ulcerative gingivitis)* who require antibiotics.

Which **two** penicillins are referred to as aminopenicillins?

- Penicillin VK
- Ampicillin *(Polycillin)*
- Cloxacillin *(Cloxapen)*
- Amoxicillin *(Amoxil)*

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- **Ampicillin *(Polycillin)***
- **Amoxicillin *(Amoxil)***

These two antibiotics are characterized by the amino substitution of penicillin G. They work against many gram-positive organisms and some gram-negative bacteria such as *Haemophilus influenzae*, some *Escherichia coli,* and *Proteus mirabilis*. They **are not** penicillinase resistant. They are used for upper respiratory infections.

The major difference in the drugs is the higher oral absorption, higher serum levels, and longer half-life for amoxicillin compared with ampicillin. Amoxicillin is given orally; ampicillin can be given orally and IV.

Oral amoxicillin is recommended as the drug of choice for standard general prophylaxis for bacterial endocarditis in patients undergoing invasive dental procedures.

Parenteral ampicillin is recommended as the drug of choice in patients unable to take oral medications and who are not allergic to penicillin for prophylaxis for bacterial endocarditis.

Cephalosporins have which of the following modes of action?

- Affect bacterial cell wall
- Affect bacterial DNA
- Affect bacterial protein synthesis
- Interfere with bacterial metabolic pathways

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- **Affect bacterial cell wall**

Basic **mechanisms of actions** of antibiotics:

Agents affecting **bacterial cell wall:**
- Penicillin family
- Cephalosporin family

Agents affecting **bacterial DNA:**
- Quinolone family *(i.e., ciprofloxacin)*
- Metronidazole *(Flagyl)*

Agents affecting **bacterial protein synthesis:**
- Tetracycline family
- Erythromycin family
- Clindamycin
- Chloramphenicol

Agents interfering with **bacterial metabolic pathways:**
- Sulfonamides *(sulfa drugs)*

Which penicillin is prescribed primarily in the treatment of **severe penicillinase-producing** staphylococcal infections?

- Methicillin
- Ampicillin
- Penicillin VK
- Carbenicillin

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• **Methicillin**

Methicillin is **not frequently** used because of the incidence of interstitial nephritis and the availability of equally efficacious alternatives *(nafcillin and oxacillin)*. It is given **IV** in severe penicillinase-producing staphylococcal infections.

Penicillinase-Resistant Penicillins	
Agent	**Administration**
Methicillin	IV
Nafcillin	IV
Oxacillin	IV
Cloxacillin	Oral
Dicloxacillin	Oral
Amoxicillin / Clavulanate potassium *(Augmentin)*	Oral
Ampicillin / Sulbactam *(Unasyn)*	IV; IM
Piperacillin / Tazobactam *(Zosyn)*	IV; IM
Ticarcillin / Clavulanate potassium *(Timentin)*	IV

Note: Methicillin-resistant Staph. aureus *(MRSA)* is a group of resistant Staph. bacteria that can be life-threatening. These bacteria are resistant to all the penicillinase-resistant penicillins and cephalosporins. Such strains are usually resistant as well to aminoglycosides, tetracyclines, erythromycins, and clindamycin. In the past, vancomycin has been used against MRSA. However, microrganisms resistant to vancomycin have been reported and its use has been curtailed.

Which of the following is the **mode of action** of the Tetracyclines?

- Bacterial cell wall destruction
- Prevent protein synthesis in the bacterial cell
- Interfere with nucleic acid synthesis
- Cause mutations within bacterial DNA

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• **Prevent protein synthesis in the bacterial cell**

The tetracyclines **inhibit protein synthesis** by binding to the 30 S subunit of the bacterial ribosome. The inhibition of this ribosomal function interferes with the attachment of the growing amino acid chain thus preventing complete formation of peptides from the ribosome. Since no peptides are formed, no proteins are formed. Since proteins are necessary for the bacterial cell to metabolically function, the lack thereof will cause a static state in which the bacterium becomes vulnerable to phagocytosis by the body's immune system.

Absorption of the tetracyclines from the GI tract **is inhibited** by divalent and trivalent cations such as **CA^{++}**, **Mg^{++}**, **Fe^{++}**, and **Al^{+++}**. These ions form chelation products with the tetracyclines and thus prevent their absorption. This is why tetracyclines should not be given with milk and dairy products *(contain Ca^{++})*, iron-containing vitamins *(contain Fe^{++})*, mineral supplements containing these irons, or antacids *(contain Mg^{++})*.

Which antibiotic exerts its antibacterial activity through **inhibition of protein synthesis** in the bacterial cell?

- Cephalosporins
- Ampicillin
- Tetracycline
- Penicillin G
- Penicillin VK

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• **Tetracycline**

The tetracyclines are a group of broad-spectrum, bacteriostatic antibiotics that inhibit protein synthesis by binding to the **30S subunit** of the bacterial ribosome. The inhibition of this ribosomal function interferes with the attachment of the growing amino acid chain thus preventing complete formation of peptides from the ribosome. Since no peptides are formed, no proteins are formed. Since proteins are necessary for the bacterial cell to metabolically function, the lack thereof will cause a static state in which the bacterium becomes vulnerable to phagocytosis by the body's immune system.

Tetracyclines are **useful in treating** the following infections:

- Medical infections caused by susceptible gram-positive and gram-negative bacteria
- Infections caused by Mycoplasma, Chlamydia or Rickettsia
- Exacerbations of chronic bronchitis
- Treatment of acne
- Treatment of gonorrhea and syphilis in patients allergic to penicillin
- Dental: treatment of periodontitis associated with the presence of Actinobacillius actinomycetemcomitans *(AA).*

True or False

Erythromycin is a **bacteriostatic** antibiotic which binds to the 50 S ribosomal subunit of susceptible bacteria. The result is the **inhibition** of protein synthesis.

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• **True**

Erythromycin-type antibiotics are members of the **Macrolide** family of antibiotics.

Members of the **Macrolide** family *(generic and brand names)* are:

- Azithromycin *(Zithromax; Z-Pak)*
- Clarithromycin *(Biaxin)*
- Dirithromycin*(Dynabac)*
- Erythromycin base *(E-mycin; Eryc)*
- Erythromycin ethylsuccinate *(E.E.S.)*
- Erythromycin stearate *(Erythrocin)*
- Erythromycin estolate *(Ilosone)*

All of the erythromycins are **very effective** against gram-positive bacteria but **not so effective** against gram-negative bacteria.

All of the erythromycins act through the **same mechanism** to bind to the 50 S ribosomal subunit of susceptible bacteria.

Note: GI Tract upset is the **most common** side effect of the erythromycins *(take with food).*

Which antimicrobial below is associated with **photosensitivity?**

- Tetracycline
- Penicillin VK
- Metronidazole *(Flagyl)*
- Clindamycin

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- **Tetracycline**

The photosensitivity reaction caused by the tetracycline family of antibiotics results in the appearance of red rashes or blotches over the skin in the presence of sunlight.

Common adverse effects caused by the other agents:

- **Penicillin VK** → hypersensitivity resulting in skin rash and rare anaphylaxis.
- **Metronidazole** *(Flagyl)* → dizziness, headache, nausea.
  Note: metronidazole is not a true antibiotic since it is not found in natural organisms; it is a synthetic substance produced in the chemical laboratory.
- **Clindamycin** → diarrhea, abdominal pain; known to cause pseudomembranous colitis.

Which statement describes the **extrapyramidal syndrome** *(EPS)* caused by the phenothiazine-type antipsychotics?

- Orthostatic hypotension
- Sedation
- Headache
- Dry mouth
- Muscle spasms of the oral-facial region

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• **Muscle spasms of the oral-facial region**

Extrapyramidal syndrome *(EPS)* refers to a variety of signs and symptoms that are a result of the blockade of dopamine receptors in specific brain regions. These symptoms include: Parkinson-like movements *(shuffled gait, pill-rolling effect of fingers),* muscle rigidity, spasms of neck and facial muscles, tremors, and loss of muscle movement.

Tardive Dyskinesia is a serious, irreversible neurological disorder that can appear at any age. Tardive Dyskinesia is a side effect of taking antipsychotic / neuroleptics drugs. Symptoms can be hardly noticeable or profound. Symptoms involve uncontrollable movement of various body parts, including the body trunk, legs, arms, fingers, mouth, lips, or tongue. Once any symptom of Tardive Dyskinesia appear, the antipsychotic / neuroleptic must be discontinued. About 20 percent of people taking antipsychotic / neuroleptic drugs for more then one year will be affected.

PHARMACOLOGY

Anti-Psych

All of the following drugs are used to treat psychosis **except**:

- Chlorpromazine *(Thorazine)*
- Thioridazine (*Mellaril)*
- Risperidone *(Risperdol)*
- Quetiapine *(Seraquel)*
- Clozapine *(Clozaril)*
- Olanzapine (*Zyprexa)*
- Haloperidol *(Haldol)*
- Nitrous oxide

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- **Nitrous oxide**

Antipsychotics are primarily indicated for **treatment of psychosis** associated with schizophrenia, paranoia, and manic symptoms of manic-depressive illness.

Chlorpromazine and thioridazine are **phenothiazines** which act by blocking dopaminergic sites in the brain. Two other phenothiazines are **trifluoperazine** *(Stelazine)* and **fluphenazine** *(Prolixin)*.

Risperidone, quetiapine, clozapine, and olanzapine are newer agents that not only antagonize dopamine, **but also** antagonize serotonin in the brain.

Note: Haloperidol is a butyrophenone and is a **potent dopamine antagonist.**

PHARMACOLOGY

Cv

All of the following drugs are classified as what type?

- Enoxaparin *(Lovenox)*
- Dalteparin *(Fragmin)*
- Tinzaparin *(Innohep)*

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- **Low molecular-weight heparin type anticoagulants**

These type of anticoagulants agents are used to treat acute symptomatic deep vein thrombosis. They are used to prevent deep vein thrombosis following knee or hip surgery.

They are administered subcutaneously since they are unable to be absorbed from the GI tract.

Standard heparin consists of components with molecular weights ranging from 4000-30,000 daltons with a mean of 16,000 daltons. Low molecular-weight heparins range in molecular weights from 2,000 to 8,000 daltons.

Heparin acts as an anticoagulant by enhancing the inhibition rate of clotting proteases by antithombin III impairing normal hemostasis and inhibition of factor Xa. Low molecular weight heparins have a small effect on partial thromboplastin time but strongly inhibit factor Xa.

PHARMACOLOGY

Cv

All of the following drugs are classified as what type?

- Abciximab *(Reopro)*
- Eptifibatide *(Integrilin)*
- Tirofiban *(Aggrastat)*

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- **Glycoprotein IIb / IIa inhibitor type of antiplatelet agent**

These agents are reversible anti-platelet agents used to prevent acute cardiac ischemic complications and used in patients with acute coronary syndrome. They are administered intravenously.

These agents block the platelet glycoprotein IIb / IIIa receptor, the binding site for fibrinogen, von Willebrand factor, and other ligands. Inhibition of binding at this final common receptor reversibly blocks platelet aggregation and prevents thrombosis. Platelet aggregation inhibition is reversible following cessation of the IV administration of the drug.

PHARMACOLOGY

Cv

All of the following drugs are classified as what type?

- Lepirudin *(Reflidan)*
- Argatroban
- Danaparoid *(Orgaran)*

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- **Thrombin-inhibitor type anticoagulant**

These agents are administered intravenously for prevention of post-operative deep vein thrombosis following elective hip replacement surgery; for prophylaxis or treatment of thrombosis in adults with heparin-induced thrombocytopenia.

Their mechanism of action is through the direct inhibition of thrombin within the coagulation pathway, thus inhibiting fibrin formation.

The following two drugs are classified as what type?

- Etanercept *(Enbrel)*
- Infliximab *(Remicade)*

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• **Anti-rheumatic agents**

Etanercept *(Enbrel)* is used to reduce the signs and symptoms of active rheumatoid arthritis in patients who have had inadequate response to one or more disease-modifying anti-rheumatic drugs (DMARDs). It is a recombinant DNA-derived protein which binds to tumor necrosis factor (TNF). TNF plays an important role in the inflammatory processes of rheumatoid arthritis (RA) and the resulting joint pathology.

Infliximab *(Remicade)* is used to treat Crohn's disease and rheumatoid arthritis. Infliximab is a chimeric monoclonal antibody that binds to tumor necrosis factor alpha *(TNF alpha)* thereby reducing the inflammatory actions of this endogenous compound.

Triazolam *(Halcion)*, a pre-operative sedative in dentistry, is metabolized in the liver by the P-450 isoform CYP 3A4 enzyme. Drugs which inhibit the actions of CYP 3A4 would affect triazolam in which way?

- Cause an increase in serum levels of triazolam
- Cause a decrease in serum levels of triazolam
- Cause no change in serum levels of triazolam

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• **Cause an increase in serum levels of triazolam**

Triazolam is known to interact with drugs that inhibit its metabolism via the CYP 3A4 enzyme. Drugs that inhibit the metabolic pathway may have a profound effect on the clearance of triazolam. The resultant effects would be an increase in serum concentrations with an associated unexpected increase in the actions of triazolam. Consequently, triazolam should be avoided in patients receiving very potent inhibitors of CYP 3A4.

Antifungal agents *(itraconazole, ketoconazole, fluconazole, miconazole, voriconazole)* can significantly elevate the serum levels of triazolam resulting in toxicity with therapeutic doses. These antifungal agents inhibit the CYP 3A4 isoform responsible for hepatic metabolism of triazolam. Thus the normal metabolism of triazolam is inhibited.

Do not administer triazolam to patients taking any of these antifungal agents.

PHARMACOLOGY

Cancer / Chemo

The following drugs belong to what **pharmaceutical class of agents:** darbepoetin alfa; pegfilgrastim; sargramostin.

- Immune modulators
- Monoclonal antibodies
- Colony stimulating factors
- Interferons
- Immunosuppressants

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• **Colony stimulating factors**

These drugs stimulate the production of neutrophils and erythroid progenitor cells in the hematopoietic process.

Darbepoetin alpha: induces erythropoiesis by stimulating the division and differentiation of erythroid progenitor cells. It is used to treat anemia associated with chronic renal failure.

Pegfilgrastim: stimulates the production, maturation and activation of neutrophils. It is used to decrease the incidence of infection by stimulation of granulocyte production in patients with nonmyeloid malignancies.

Sargramostin: is used for myeloid reconstitution after autologous bone marrow transplantation.

When writing a prescription, **q.i.d.** is the abbreviation for what?

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- **Four times a day**

Other popular and common abbreviations used when writing prescriptions:

- **b.i.d.** = twice a day
- **t.i.d.** = three times a day
- **stat.** = immediately
- **p.r.n.** = as needed
- **p.c.** = after meals
- **q.4.h.** = every 4 hours
- **q.12.h.** = every 12 hours
- **h.s.** = at bedtime
- **a.c.** = before meals

Note: Always **document** prescriptions that are given to a patient in the patient's chart, along with the date they were written and any specific instructions for patient use.

PHARMACOLOGY

Misc.

All of the following drugs are useful in treating what **medical condition?**

- Pilocarpine *(Isopto-Carpine)*
- Latanoprost *(Xalatan)*
- Betaxolol *(Betoptic)*
- Bimatoprost *(Lumigan)*

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- **Glaucoma**

Glaucoma is characterized by an increase in intraocular pressure. It is caused by poor drainage of the aqueous humor *(fluid in the eye)* and can cause blindness.

How these **drugs work** in treating glaucoma:

- **Pilocarpine** *(Isopto-Carpine)* → eye drops in the eyes causes papillary constriction thus allowing for drainage of the aqueous humor to reduce pressure.

- **Latanoprost** *(Xalatan)* → a prostaglandin analog; eye drops in the eye reduces intraocular pressure by increasing the outflow of the aqueous humor.

- **Betaxolol** *(Betoptic)* → a beta blocker; eye drops in the eye reduces intraocular pressure by reducing the production of aqueous humor.

- **Bimatoprost** *(Lumigan)* → same action as latanoprost *(Xalatan).*

The following drugs are noted for causing what **prominent** oral side effect?

- Amitriptyline *(Elavil)*
- Diphenhydramine *(Benadryl)*
- Atropine
- Diazepam *(Valium)*

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- **Xerostomia** *(dry mouth)*

Xerostomia can be caused by certain drug classes that **inhibit** the production and secretion of saliva.

- **Amitriptyline** *(Elavil)* is representative of the tricyclic antidepressants, a class of drugs that causes significant xerostomia. They probably work through an anticholinergic action.

- **Diphenhydramine** *(Benadryl)* is representative of the sedating-type antihistamines, a class of drugs that causes significant xerostomia. They probably work through an anticholinergic action.

- **Atropine** is a powerful anticholinergic which blocks the production of saliva in the salivary glands. Other anticholinergics will have a similar action.

- **Diazepam** *(Valium)* is representative of the benzodiazepine tranquilizers. These drugs have moderate anticholinergic actions to reduce the outflow of saliva.

The xerostomia actions produced by these classes of drugs are **reversible** with normal salivary flow regained after discontinuance of the drug.

PHARMACOLOGY

Misc.

Write up a sample prescription, illustrating all of the **pertinent information** that needs to be included when writing a prescription for a patient.

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Dr. John Doe, DDS
11 Any Street
Any City, Any State 00000
(000) 555-1212

License #
Federal Drug Registry #

Patient's Name: _____ Age: _____
Patient's Address: _____ Date: _____

Rx: Amoxicillin 500 mg. tablets
Disp. Four *(4)* tabs
Sig: Take 4 tabs *(2000 mg)* 1 hour prior to dental appointment

Signature: _____

Substitution permissible _____
Substitution not permissible _____
Number of refills _____

Notes:

- **Superscription** – Patient's name, address, age, date
- **Inscription** – Name of drug and the strength of the drug *(i.e., 500 mg tablets)*
- **Subscription** – Directions to the pharmacist *(dosage form and amount to be given → Disp.)*
- **Transcription** or signa – Directions to the patient *(Sig.)*
- **Signature** – Signature of person prescribing medication **must** appear

All of the following drugs are useful for treating what **common** medical condition?

- Prednisone
- Gold injections
- Methotrexate
- Nabumetone *(Relafen)*
- Piroxicam *(Feldene)*

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- **Rheumatoid arthritis**

Rheumatoid arthritis *(RA)* is a chronic inflammatory disease of joints that results in joint pain, swelling, and destruction. RA is characterized by chronic inflammation of the synovium, which lines the joint. With disease progression, there is the accumulation of prostaglandins, leukotrienes and other mediators in the inflammatory changes and tissue destruction in the synovial lining.

How these **drugs work** in treating rheumatoid arthritis:

- Prednisone→ decreases the inflammatory response
- Gold injections → may decrease prostaglandin production
- Methotrexate → unknown, but may affect immune function
- Nabumetone *(Relafen)* → an NSAID that inhibits prostaglandin synthesis
- Piroxicam *(Feldene)*→ an NSAID that inhibits prostaglandin synthesis

Note: All of the above drugs except gold injections are also useful in the treatment of osteoarthritis (OA). OA is characterized by progressive loss of articular cartilage. This may be the result of excessive loads on the joint or other factors. Agents useful in treating OA provide an analgesic and anti-inflammatory action to reduce pain within the joint.

PHARMACOLOGY

Anti-Infect

Which drug can be used in the **pregnant patient?**

- Tetracycline
- Doxycycline *(Vibramycin)*
- Minocycline *(Minocin)*
- Penicillin VK

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• **Penicillin VK**

Penicillin VK is **not harmful** to the fetus when taken by pregnant mothers. It is not incorporated into bony tissue or in the teeth of children like the tetracyclines.

Tetracycline and all members of the tetracycline family are **contraindicated** in children up to 8 years old and in pregnant women. Tetracyclines have the ability to chelate calcium ions and become incorporated in the bony tissues. The teeth of children who have been given the drug may develop a greenish-brown discoloration. This effect is sometimes seen in the newly erupted teeth of infants whose mothers have received tetracycline during pregnancy.

Which **gastrointestinal drugs** reduce the formation of stomach acid by **inhibiting the proton pump** of the stomach parietal cells?

- Ranitidine *(Zantac)*
- Cimetidine *(Tagamet)*
- Famotidine *(Pepcid)*
- Omeprazole *(Prilosec)*
- Lansoprazole *(Prevacid)*

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- **Omeprazole *(Prilosec)***
- **Lansoprazole *(Prevacid)***

Hydrochloric acid *(HCl)* is produced by the parietal cells of the stomach through a pump within each cell which pumps protons (H^+) into the stomach contents. The pump is called the H^+ / K^+ ATPase pump. HCl is used for food digestion but an abundancy can cause heart burn and acid indigestion. Omeprazole and lansoprazole inhibit the pump such that no protons are pumped into the stomach contents and thus no HCl is produced. These two drugs are classified as **proton-pump inhibitors.**

Stomach acid can also be reduced by inhibiting the effects of histamine in the stomach at the histamine type-2 receptors (H_2 receptors). Ordinarily, histamine stimulates the gastric parietal cells to produce hydrochloric acid. Ranitidine, cimetidine and famotidine block the effects of histamine by blocking at the H_2 receptors. These three drugs are classified as **H_2 receptor blockers.**

Both the proton-pump inhibitors and H_2 blockers are used to treat heartburn, indigestion, sour stomach, active duodenal ulcer disease and gastroesophageal reflux disease *(GERD).*

Remember: Antacids neutralize excess stomach acid by a chemical reaction. Antacids include aluminum hydroxide (*Amphogel),* bismuth subsalicylate *(Pepto-Bismol),* calcium carbonate *(Tums)* magnesium hydroxide *(Maalox)* and sodium bicarbonate.

Which drugs inhibit blood clotting by **affecting the coagulation pathway** to prevent fibrin formation?

- Heparin
- Warfarin *(Coumadin)*
- Vitamin K
- Aspirin
- Clopidogrel *(Plavix)*

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- **Heparin**
- **Warfarin *(Coumadin)***

Heparin inactivates thrombin and prevents the conversion of fibrinogen to fibrin *(blood clot)*; warfarin *(Coumadin)* interferes with the hepatic synthesis of vitamin-K dependent coagulation factors II, VII, IX and X resulting in the inability of the coagulation pathway to form fibrin *(blood clot)*.

Vitamin K is a group of fat soluble vitamins that are essential for the synthesis of coagulation factors II, VII, IX and X, and prothrombin in the liver. Vitamin K will enhance blood clotting rather than inhibit blood clotting.

Aspirin inhibits blood clotting by inhibiting platelet aggregation in an irreversible manner. Inhibition of platelet aggregation prevents activation of the coagulation pathway; thus no fibrin *(clot)* is formed. Aspirin **does not affect** the coagulation pathway. Discontinuation of aspirin for 5 to 7 days allows for normal clotting time to reappear due to the synthesis of new platelets.

Clopidogrel *(Plavix)* inhibits blood clotting by inhibiting platelet aggregation in an irreversible manner. Thus the effects on blood clotting are the same as aspirin. Clopidogrel *(Plavix)* does not cause gastric ulcers like aspirin does and is the antiplatelet agent of choice in patients with history of ulcers.

A patient was given penicillin 15 minutes ago and develops the following signs and symptoms. **What kind of reaction is this patient having?**

- Laryngeal edema
- Urticaria *(welts that itch)*
- Severe hypotension
- GI disturbances
- Bronchoconstriction *(airway constriction)*
- Shock

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• **Anaphylactic reaction** *(anaphylactic shock)*

*** This reaction most commonly occurs with parenteral administration of penicillin.

This reaction can be fatal if countermeasures such as the injection of epinephrine are not taken promptly. Epinephrine prevents the release of substances from mast cells and antagonizes the actions of histamine and leukotrienes of smooth muscle.

The most common adverse effect of penicillin therapy is an **allergic reaction.** These reactions occur in up to 10% of patients receiving penicillin. The most common manifestation is a **mild rash.**

Allergic reactions to penicillin are **classified** into three groups:

1. **Immediate – onset reactions** *(anaphylaxis)* occur within 30 minutes; Ig-E mediated.
2. **Accelerated – allergic reactions** arise 30 minutes to 48 hours after administration. Urticaria, pruritis, wheezing, and local inflammatory reactions. In general, not life-threatening.
3. **Delayed – allergic reactions** take longer than two days to develop. **80-90%** of penicillin reactions are of this type. Basic skin rashes, which are generally mild.

PHARMACOLOGY

Corticosteroids

Which **corticosteroid** is administered by inhalation to treat asthma?

- Hydrocortisone
- Prednisone
- Cortisone
- Fluticasone (*Flonase)*
- Methylprednisolone *(Medrol)*

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- **Fluticasone** *(Flonase)*

Corticosteroids in the inhaled form, decrease the inflammation in the airway in asthma. Reduction of inflammation enhances the bronchodilating effects of the beta 2 adrenergic agonists.

The following are some of the other popular **inhaled corticosteroids** used in the treatment of asthma:

- **Triamcinolone** *(Azmacort)*
- **Beclomethasone** *(Beconase)*
- **Budesonide** *(Pulmicort)*

Note: Inhaled steroids very often cause a fungal infection of the mouth and throat.

All the following are classified as **what type of drug?**

- Gabapentin *(Neurontin)*
- Carbamazepine *(Tegretol)*
- Phenytoin *(Dilantin)*
- Diazepam *(Valium)*
- Valproic acid *(Depakene)*

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• **Antiepileptic drugs or anticonvulsants**

Epilepsy is **neurological disorder** characterized by sudden, recurring attacks of motor, sensory, or psychic malfunction with or without the loss of consciousness or convulsive seizures. The goal of anticonvulsant therapy is to reduce or eliminate these seizures.

Phenytoin *(Dilantin)* is used in the treatment of tonic-clonic *(grand mal)* seizures. **Phenytoin-induced gingival hyperplasia is common** and may partially or totally obscure the crowns of teeth. The rate of development of this condition can be diminished by proper oral hygiene.

Gabapentin is used as an adjunct to treat partial seizures.

Carbamazepine *(Tegretol)* is used as prophylaxis for partial seizures with complex symptomatology including psychomotor and temporal lobe seizures. It is also used to treat tonic-clonic seizures *(grand mal)* and **trigeminal neuralgia**. It rarely causes aplastic anemia.

Diazepam *(Valium)* is used for **status epilepticus** and in emergency treatment of seizures. May cause drowsiness, dizziness, and ataxia.

Valproic acid *(Depakene)* is used to treat patients with complex partial seizures; adjunctively in patients with multiple seizure types including absence seizures. May cause liver failure and blood dyscrasias.

Pharmaceutical agents that bring about tissue responses resembling those produced by stimulation of the **sympathetic nervous system** are called?

- Cholinomimetic
- Antiadrenergic
- Parasympathomimetic
- Sympathomimetic

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• **Sympathomimetic**

The terms **sympathomimetics, sympathomimetic amines, adrenergic agonists** and **adrenergic agents** are almost always synonymous.

Epinephrine is the sympathomimetic agent used in dentistry. It is the vasoconstrictor for anesthetic solutions and the vasoconstrictor component in gingival retraction cords. As a component of local anesthetic preparations, it is used to prolong the duration of local anesthesia.

In medicine, sympathomimetics are used as pressor agents to maintain blood pressure in vascular shock. They are used as bronchodilators for asthma attacks and for allergic states including anaphylactic shock. Sympathomimetics used in medicine include dopamine, epinephrine, norepinephrine, isoproterenol, and phenylephrine.

Epinephrine is indicated in medicine to treat bronchospasm and hypersensitivity reactions. It is the agent of choice for **reversing anaphylactic reactions**. It is used to restore cardiac activity in cardiac arrest.

The medical uses of a drug having adrenergic agonist actions would include which of the following?

- Reverse an anaphylactic reaction
- Treat hypertension
- Prevent angina pectoris
- Reduce anxiety
- All of the above

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• **Reverse an anaphylactic reaction**

The prototypical **adrenergic agonist** agent is epinephrine. Epinephrine stimulates both beta adrenergic receptors and alpha adrenergic receptors within the sympathetic division of the autonomic nervous system. Anaphylaxis is characterized by rapid, extreme reduction in blood pressure and bronchospasms. Epinephrine, upon injectable administration, will rapidly reverse the hypotension by causing vasoconstriction via the $alpha_1$ receptor stimulation; it will dilate the bronchial tubes via $beta_2$ receptor stimulation; it will increase cardiac output via $beta_1$ receptor stimulation on the cardiac muscle.

Epinephrine is:

- **Ineffective** in treating hypertension because of its alpha receptor stimulatory actions on the vasculature which could cause an even further elevation of blood pressure.
- **Contraindicated** in angina conditions because its cardiostimulatory effects would aggravate this condition.
- **Will not reduce anxiety** but will likely increase anxiety since it has central nervous system stimulatory effects.

A **fear reaction** activates the sympathetic division of the autonomic nervous system to result in:

- Miosis
- Bradycardia
- Hypertension
- Increased salivation

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• **Hypertension**

Activation of the sympathetic portion of the autonomic nervous system will cause alpha$_1$ adrenergic receptor activation to result in arteriolar vasoconstriction with an associated elevation of blood pressure leading to hypertension.

Notes:

1. Sympathetic activation of the eye would result in mydriasis *(dilation)* **not** miosis.
2. Sympathetic activation of the heart would result in tachycardia, **not** bradycardia.
3. Sympathetic activation of the salivary glands would result in a thick, ropey-type salivary flow, **not** increased salivation.

Miosis *(pupillary constriction)*, bradycardia, and increased salivation are **physiological effects** all resulting from **activation of the parasympathetic division** of the autonomic nervous system.

When two drugs having similar effects are administered in combination, and the resulting response is the sum of the individual actions of each drug when given alone, then this result is referred to as:

- An additive effect
- A synergistic response
- An antagonistic effect
- Cumulative action

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• **An additive effect**

An **additive** effect occurs when additive drugs are administered. The response is no greater than that which would be expected had the drugs been given one at a time. There is no enhancement of potential of the individual drugs as a result of being used in combination.

A **synergistic** response occurs when the combined action of two drugs having similar pharmacological effects is greater than the sum of the individual actions. Alcohol is synergistic with the Valium-family drugs *(i.e., Valium, Xanax, Halcion, etc)*, narcotics and barbiturates. Alcohol should be avoided when taking these medications.

PHARMACOLOGY

Chol

Which drug action would be **most useful** to induce salivation?

- Cholinergic
- Anticholinergic

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• **Cholinergic**

Cholinergic drugs **stimulate acetylcholine cholinergic receptors** to cause:

- Salivation
- Excessive sweating
- Increased GI motility
- Miosis *(papillary constriction)*
- Flushing
- Bradycardia

Direct acting cholinergic drugs include: methacholine, carbachol, bethanecol and pilocarpine.

Indirect acting cholinergic drugs *(cholinesterase inhibitors)* include: neostigmine, physostigmine, edrophonium and pyridostigmine.

Anticholinergic drugs produce the **opposite effects** of cholinergic agents. These effects are:

- Dry mouth
- Decreased GI motility
- Dry skin
- Mydriasis *(pupillary dilation)*
- Tachycardia

Anticholinergic drugs include: atropine sulfate, glycopyrrolate *(Robinul)*, belladonna derivatives, propantheline bromide *(Pro-banthine)*, and scopolamine.

PHARMACOLOGY

Chol

Which of the following drugs may be useful in **controlling salivary secretions** to help in obtaining a dry field?

- Atropine sulfate
- Glycopyrrolate *(Robinul)*
- Belladonna derivatives
- Propantheline bromide *(Pro-banthine)*
- All of the above

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• **All of the above** *(they are antisialagogues)*

They are all classified as **anticholinergics**. They block postganglionic cholinergic fibers.

Contraindications to their use include:

- Glaucoma
- Cardiovascular problems
- Obstruction of the GI or GU tract
- Asthma

These drugs also **reduce spasms** of smooth muscle in the bladder, bronchi, and intestine; relax the iris sphincter; decrease gastric, bronchial, and salivary secretions; decrease perspiration; and accelerate impulse conduction through the myocardium by blocking vagal impulses.

For a patient who is taking anticoagulants, what is the **most valuable test** used in evaluating the patient as a surgical risk?

- PTT *(Partial Thromboplastin Time)*
- PT *(Prothrombin Time)*
- Platelet count

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• **PT** ***(Prothrombin Time)***

This test is a one-stage test for **detecting certain plasma coagulation defects** owing to a deficiency of factors V, VII, or X. Thromboplastin and calcium are added to a sample of the patient's plasma and simultaneously, to a sample from a normal control. The length of time required for clot formation in both samples is observed. Thrombin is formed from prothrombin in the presence of adequate calcium, thromboplastin, and the essential tissue coagulation factors. A prolonged PT therefore indicates deficiency in one of the factors, as in liver disease, vitamin K deficiency, or anticoagulation therapy with the drug coumarin.

International Normalized Ratio *(INR)*. Once prothrombin times are determined, they are expressed as an INR value. INR stands for International Normalized Ratio and essentially is the ratio of the prothrombin time measured in the patient divided by a standard prothrombin time value, and multiplied by a constant. An INR value of 1 means normal prothrombin times of approximately 12 seconds; normal blood clotting would be present. INR values greater than 1 indicate that that there is an anticoagulant effect. The **higher the INR value, the greater the anticoagulant effect**. Many patients taking anticoagulants have INR values of 2,3 and even up through 6. For surgical procedures, an INR of 1 to 1,5 would be indicative of a prothrombin time between normal *(approximately 12 seconds)* and approximately 18 seconds. This is generally agreed to be within the safe range.

Some of the major drug groups that can **increase a patient's bleeding times** are aspirin, the non-selective NSAIDs *(ibuprofen, naproxen, ketoprofen, flurbiprofen)*, the anti-platelet drugs clopidogrel *(Plavix)* and ticlopidine *(Ticlid)*, and the anti-coagulants warfarin *(Coumadin)* and heparin.

All of the following conditions are managed by using anticoagulants and anti-platelet agents **except:**

- Coronary Artery Disease *(CAD)*
- Angina Pectoris *(Unstable Angina)*
- Myocardial Infarction *(Heart Attack)*
- Stroke
- Hypertension

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• **Hypertension**

Anticoagulants such as warfarin *(Coumadin)* and anti-platelet agents such as aspirin and clopidogrel *(Plavix)* are used in the conditions listed for the following reasons:

- **Coronary artery disease** *(CAD)* → will help prevent threat of myocardial infarction in CAD patients.
- **Angina pectoris** *(unstable angina)* → will help prevent thrombus from forming within the coronary arteries.
- **Myocardial infarction** *(MI)* → drugs that prevent blood clotting have been shown to prevent the threat of future infarcts.
- **Stroke** → will help prevent thrombus from forming thus preventing threat of a cerebral embolism.

Hypertension → Unless there are other accompanying cardiovascular problems such as those listed above, anticoagulant drugs are not necessary in the treatment and management of hypertension. These drugs do nothing to lower blood pressure.

True or false

The class of drugs known as **HMG-CoA Reductase Inhibitors**, also known as the "statin" drugs, lower blood cholesterol levels by inhibiting a key enzyme in the cholesterol synthesis pathway in the liver.

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• **True**

HMG-CoA Reductase is an enzyme known as hydroxy methylglutaryl coenzyme A reductase and is necessary in the key step to synthesize cholesterol. When the **"statin"** drugs inhibit this enzyme, cholesterol is not produced in the liver and blood levels decrease.

The family of "statin drugs" include **atorvastatin** *(Lipitor),* **simvastatin** *(Zocor),* **fluvastatin** *(Lescol),* **lovastatin** *(Mevacor)* **pravastatin** *(Pravachol) and* **rosuvastatin** *(Crestor)*

Coronary artery disease *(CAD)* is a condition of narrowing of the blood vessels of the heart restricting oxygen flow to heart muscle. It has been correlated with the levels of blood cholesterol and triglycerides. If not treated, CAD can lead to myocardial infarction *(heart attack)*. Drugs such as the statins, which lower blood cholesterol are effective in minimizing the threat of CAD.

Important: The "statin" drugs have the capability to increase the breakdown of skeletal muscle thereby releasing muscle protein. If the protein overloads the kidneys, renal failure could result. The erythromycin drugs enhance the capabilities of the "statins" to cause this effect. It is advisable for patients medicated with a "statin" drug not to be given erythromycin products.

True or False

Antihypertensive agents lower blood pressure by **reducing** total peripheral resistance and by **reducing** cardiac output through a variety of mechanisms.

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• **True**

Antihypertensive agents are classified by **mechanisms** of action:

- **Diuretics** for example **hydrochlorothiazide** *(HCTZ)* and **furosemide** *(Lasix)* → inhibit sodium reabsorption in renal tubular cells within the kidney to cause excess sodium and urinary excretion resulting in reduced blood volume.
- **Beta adrenergic receptor blockers** *(beta blockers)* → reduce the volume of cardiac output into the circulation resulting in reduce peripheral pressure.

Two types:

1. **Cardioselective beta blockers** *(beta 1 receptor block in heart muscle)* – examples include **atenolol** *(Tenormin)* and **metoprolol** *(Lopressor; Toprol XL)*.
2. **Non-cardioselective beta blockers** – examples include **nadolol** *(Corgard)* and **propranolol** *(Inderal)*.

- **Angiotensin-converting enzyme inhibitors** *(ACE Inhibitors)*, examples include **lisinopril** *(Prinivil; Zestril)*, **ramipril** *(Altace)* and **enalapril** *(Vasotec)* → inhibit the conversion of inactive angiotensin I to the angiotensin II, a vasoconstrictor. This results in peripheral vasodilation and secondarily, an increase in urinary volume excretion. Both actions result in reduced blood pressure.
- **Calcium channel blockers** examples include **amlodipine** *(Norvasc)*, **diltiazem** *(Cardizem)*, **nifedipine** *(Procardia)* → inhibit calcium entry into vascular smooth muscle causing vasodilation of coronary and peripheral blood vessels thus lowering blood pressure.

PHARMACOLOGY

Asp / Acet / NSAID's

Your patient has a history of drug abuse. Which agent could be given *(if needed in the treatment plan)* with **no liability** to cause an addiction?

- Phenobarbital
- Ibuprofen
- Hydrocodone
- Meperidine
- Codeine

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• **Ibuprofen**

Ibuprofen is an **NSAID** and is classified as a **non-narcotic analgesic**. Non-narcotic analgesics have **no liability** for abuse or addiction. They **are not** a controlled substances.

Phenobarbital is a **barbiturate** used as a sedative. Barbiturates all have the **potential** to cause abuse and addiction, and are **controlled substances** requiring a DEA number from the prescriber.

Hydrocodone *(Vicodin),* **meperidine** *(Demerol)* and **codeine** are narcotic analgesics with the **potential** to cause abuse and addiction. They are all **controlled substances** requiring a DEA number from the prescriber.

PHARMACOLOGY — Asp / Acet / NSAID's

All of the following agents **reduce** the production of what substance associated with pain and inflammation?

- Flurbiprofen *(Ansaid)*
- Ibuprofen *(Motrin, Advil)*
- Naproxen *(Naprosyn)*
- Piroxicam *(Feldene)*
- Naproxen sodium *(Anaprox, Aleve)*
- Nabumetone *(Relafen)*

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• **Prostaglandins**

They are all **non-steroidal anti-inflammatory drugs** *(NSAID's).* The mechanism of action for these drugs is that they **inhibit** the cyclooxygenase step of the arachidonic acid cascade and thus **inhibit** the activity of prostaglandin synthetase. Prostaglandins are a group of hormone-like substances that mediate a range of physiological functions, such as metabolism and nerve transmission.

NSAID's have **analgesic, antipyretic,** and **anti-inflammatory properties** *(similar to aspirin).* They are used for pain control, arthritis, and painful menstruation. Adverse reactions include GI upset *(possible ulcers)*,and **prolongation of bleeding time** *(reduction in platelet aggregation).* Contraindications to the use of NSAID's are impaired renal function, pregnancy, and GI disease *(ulcers).*

PHARMACOLOGY Asp / Acet / NSAID's

Which of the following agents has **little value** in treating acute inflammation?

- Ibuprofen
- Acetaminophen
- Aspirin
- Naproxen

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• **Acetaminophen** *(Tylenol)*

Acetaminophen inhibits **central** prostoglandin synthesis – It is **analgesic** for low intensity pain and antipyretic. Because it is less effective than salicylates *(aspirin)* in blocking peripheral prostaglandin synthesis, it has **no anti-inflammatory activity** and **does not** affect platelet function and therefore **will not** affect clotting time. **Note:** In large doses, acetaminophen can cause hepatic necrosis.

Remember: Aspirin and Nonsteroidal Anti-inflammatory Agents *(Ibuprofen, Naproxen and others)* inactivate the enzyme known as cyclooxygenase. Since cyclooxygenase synthesizes the prostaglandins, the inhibition of this enzyme results in the inhibition of prostaglandin synthesis. Cyclooxygenase has an acronym of COX. Thus the Nonsteroidal Anti-inflammatory Agents *(NSAID's)* are also known as COX inhibitors. As a result, they have analgesic, antipyretic, and anti-inflammatory actions. NSAID's must be used cautiously in patients with peptic ulcer disease.

PHARMACOLOGY

Asp / Acet / NSAID's

Your patient is taking the following medications. Which of the medications are used to reduce the signs and symptoms of **rheumatoid arthritis?**

- Amlodipine *(Norvasc)*
- Enalapril *(Vasotec)*
- Piroxicam *(Feldene)*
- Prednisone

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- **Piroxicam** *(Feldene)*
- **Prednisone**

Piroxicam *(Feldene)* is a non-steroidal anti-inflammatory drug *(NSAID)* that inhibits prostaglandin synthesis. It is used to manage inflammatory disorders and used for the symptomatic treatment of acute and chronic rheumatoid arthritis and osteoarthritis.

Prednisone is a corticosteroid and has anti-inflammatory actions. It is used for the treatment of a wide variety of inflammatory diseases including rheumatoid arthritis and osteoarthritis.

Remember:
Common side effects of NSAID's such as piroxicam *(Feldene)* include:
- Gastric irritation
- Heart burn
- Nausea

Short-term side effects of corticosteroids such as prednisone include:
- Insomnia
- Indigestion
- Arthralgia

Long-term side effects of corticosteroids include:
- Edema *(abdominal distension)*
- Psychological disturbances
- Peptic ulcer
- Osteoporosis
- Muscle weakness

These drugs act to induce gene transcription, inhibit cellular growth and alter the state of cell differentiation.

- Immune globulins
- Immunosuppressants
- Keratinocyte growth factor
- Interferons

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- **Interferons**

Interferons are used for a variety of conditions including: **hairy cell leukemia** *(interferon alpha-2a)* **chronic hepatitis B** *(interferon alpha-2b),* **recurring genital warts** *(interferon alpha-n3)* and **treatment of multiple sclerosis** *(interferon beta-1a).*

PHARMACOLOGY — Asp / Acet / NSAID's

Celecoxib *(Celebrex)* is a member of which category of drugs?

- Salicyclates
- Opiates
- COX-2 selective inhibitors
- Non-selective COX inhibitors
- Steroidal anti-inflammatories

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• **COX-2 selective inhibitors**

Cyclooxygenase, or COX, is the enzyme which produces prostaglandins. Two forms of COX exist: COX-1, and COX-2. The **COX-1 enzyme** produces prostaglandins in the GI Tract. The prostaglandins formed act as a protective substance against the formation of gastrointestinal ulcers. The traditional NSAID's such as ibuprofen, naproxen, aspirin, and others inhibit the COX-1 enzyme thus diminishing the formation of the protective prostaglandins. Gastrointestinal ulcers are therefore a potential adverse effect with these drugs. The **COX-2 enzyme** produces prostaglandins at the sites of surgery, infection and inflammation. When this enzyme is inhibited, less prostaglandins are produced and there is less pain and inflammation. The traditional NSAID's such as ibuprofen, naproxen and aspirin inhibit COX-2 along with COX-1. Thus they are effective in reducing pain and inflammation, but are capable of inducing gastrointestinal ulcers. The COX-2 selective inhibitors will reduce pain and inflammation without any significant risk of causing gastrointestinal ulcers.

For the traditional NSAID's such as ibuprofen, naproxen, and aspirin, because they inhibit both COX-1 and COX-2 enzymes, they belong to the category of **non-selective** COX inhibitors. For **celecoxib** *(Celebrex),* because it inhibits COX-2 enzyme only, it belongs to the category of **COX-2 selective** inhibitors.

The **COX-2 selective** inhibitors:
– **Are not** salicylates because they are not aspirin drugs
– **Are not** opiates because they do not work like morphine
– **Are not** steroidal anti-inflammatories because they are not corticosteroids such as hydrocortisone

PHARMACOLOGY — Asp / Acet / NSAID's

Which category of drugs **does not** affect blood clotting and can be given to patients taking "blood thinners"?

- Salicylates
- NSAID's *(ibuprofen, naproxen, etc.)*
- Aspirin
- COX-2 selective inhibitors

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• **COX-2 selective inhibitors**

The COX-2 selective inhibitors include: **celecoxib** *(Celebrex)*. This COX-2 selective inhibitor **does not affect** platelet function. These drugs can be given to patients concomitantly taking "blood thinners" such as warfarin *(Coumadin)* or aspirin.

The salicylates, including aspirin, interfere with blood clotting mechanisms by irreversibly reducing platelet adhesiveness *(stickiness or aggregation)*. Bleeding time is prolonged and remains prolonged until new platelets are formed. The traditional NSAID's *(ibuprofen, naproxen, etc.)* reduce platelet aggregation in a reversible fashion. Normal platelet function will return as soon as these drugs leave the system.

The COX-2 selective inhibitors are used to treat signs and symptoms of rheumatoid and osteoarthritis, acute pain, and pain from dysmenorrhea.

PHARMACOLOGY

Narc Analg

Which **narcotic** is not used in dentistry?

- Morphine
- Hydrocodone *(component of Vicodin)*
- Oxycodone *(component of Percocet)*
- Meperidine *(Demerol)*
- Codeine
- Fentanyl

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• **Morphine** *(opiates)*

Therapeutic indication for opioids *(narcotic analgesics)* are the relief of moderate-to-severe pain, as preanesthetic medications, as analgesic adjuncts during anesthesia, as antitussives, and as antidiarrheals. **Note:** They are administered **with caution** to patients with head injury or those with a history of drug abuse and dependency.

Notes:

1. Morphine is not used in dentistry because of its high addictive liability.
2. Hydrocodone in combination with acetaminophen is known as Vicodin, Lorcet, Lortab, Maxidone, and Zydone.
3. Hydrocodone in combination with ibuprofen is known as Vicoprofen.
4. Oxycodone in combination with acetaminophen is known as Roxicet, Percocet, and Tylox.
5. Oxycodone in combination with ibuprofen is known as Combunox.
6. Oxycodone alone is known as OxyContin.
7. Meperidine *(Demerol)* in combination with promethazine is known as Mepergan Fortis.
8. Codeine in combination with acetaminophen is known as Tylenol #3.
9. Fentanyl is available as a transmucosal preparation known as Actiq, a transdermal patch formulation known as Duragesic and as an intravenous preparation known as Sublimaze.

Among the opiates available for use in dentistry, **hydrocodone** products are commonly the drugs of choice.

Which of the following analgesics may **interact** with warfarin *(Coumadin)* to cause increased bleeding?

- Acetaminophen
- Ibuprofen *(Motrin; Advil)*
- Hydrocodone with acetaminophen
- Aspirin

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- **Ibuprofen *(Motrin; Advil)***
- **Aspirin**

Ibuprofen and other non-selective NSAIDs *(inhibitors of both cyclo-oxygenase 1 and cyclo-oxygenase 2 enzymes)* such as **naproxen** *(Anaprox)* and **flurbiprofen** *(Ansaid)* inhibit platelet aggregation. This action would enhance the anti-coagulant effect of **warfarin** *(Coumadin)* to increase the risk of bleeding. Aspirin inhibits platelet aggregation to potentiate the anticoagulant effects of warfarin *(Coumadin)* and increase the risk of bleeding.

Acetaminophen is a non-narcotic analgesic that **does not affect** platelet aggregation nor does it affect the coagulation pathway. Thus, it will not affect the anticoagulant nature of warfarin *(Coumadin)*. Acetaminophen can be given safely to patients taking warfarin *(Coumadin)*.

Hyrocodone with acetaminophen is a combination of a narcotic analgesic with acetaminophen. Common brand names for this combination are **Vicodin, Lorcet and Lortab.** Hydrocodone and the entire drug class of narcotic analgesics **do not affect** blood clotting and will not enhance the anticoagulant effects of warfarin *(Coumadin)*. Narcotics with acetaminophen can be given safely to patients taking warfarin *(Coumadin)*.

PHARMACOLOGY

Narc Analg

Your patient has a history of neuropathic pain. They are currently taking **oxycodone** *(OxyContin)*, which is an opiate analgesic. Which of the following are **adverse effects** of oxycodone?

- Nausea
- Constipation
- Peptic ulcers
- Insomnia

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- **Nausea**
- **Constipation**

The **most common** side effect of the narcotic *(opiate)* analgesics is nausea. The **most serious** side effect of the narcotic analgesics is **respiratory depression.** The cause of death from overdose of narcotics is respiratory depression and shut down of the respiratory system.

Narcotic analgesics **do not** cause peptic ulcers. Nor do they cause insomnia, but rather would cause drowsiness and sedation since narcotics depress the conscious centers of the brain.

PHARMACOLOGY

Narc Analg

Which **analgesics** can produce drug dependence and addiction?

- Aspirin
- Ibuprofen
- Codeine
- Morphine
- Meperidine *(Demerol)*

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- **Codeine**
- **Morphine**
- **Meperidine** *(Demerol)*

These three analgesics belong to the **opiate class** of drugs. They are also known as narcotic analgesics since the actions of this family is to cause drowsiness and sleep as a side effect.

The opiates **produce** drug dependence leading to addiction. Psychic dependence, physical dependence, and tolerance can develop upon repeated administration. Psychic dependence is unlikely if an opiate is taken for a short period for pain relief. Physical dependence is a condition in which continued administration of the drug is required to prevent unpleasant withdrawal symptoms. Tolerance occurs when increasingly large doses of opiate are required in order to produce the same degree of analgesia.

Opiate drugs **used in dentistry** to provide pain relief after dental surgery include:
– Codeine
– Hydrocodone *(Vicodin)*
– Oxycodone *(Percocet; Tylox)*
– Meperidine *(Demerol)*

PHARMACOLOGY

Corticosteroids

All of the following drugs are classified as what type?

- Hydrocortisone
- Methylprednisolone
- Prednisone
- Triamcinolone
- Dexamethasone

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• **Corticosteroids** *(steroids)*

Two types:

1. **Glucocorticoids** affect carbohydrate, lipid, and protein metabolism. They are used as **anti-inflammatory** agents.
2. **Mineralocorticoids** regulate sodium and potassium metabolism.

These drugs **are used to treat** a variety of conditions which include asthma, arthritis, allergies, aphthous stomatitis, lupus erythematosus, and TMJ pain.

Contraindications to their use include latent infections *(fungal, viral, or bacterial)*, AIDS, herpes infections, gastric ulcers, and congestive heart failure.

Adverse reactions include Cushing's syndrome *(obesity and weakening of muscles)*, hyperglycemia, osteoporosis, peptic ulcers, and an increased risk of infection.

Corticosteroids do not cure any disease. They represent replacement only in Addison's disease.

Inhaled corticosteroids used for asthma **do not achieve** significant blood levels to cause the adverse effects listed above for systemic agents. Popular aerosol corticosteroids are **triamcinolone** *(Azmacort)*, **beclomethasone** *(Beconase)*, **fluticasone** *(Flovent)* and **budesonide** *(Pulmicort)*. Localized infections with Candida albicans occur frequently in the mouth and pharynx with repetitive use of inhalant corticosteroids.

Nasal spray corticosteroids used for seasonal allergies also **do not achieve** significant blood levels, and are use for their localized effects. Popular nasal spray corticosteroid products are **triamcinolone** *(Nasocort)*, **fluticasone** *(Flonase)* and **budesonide** *(Rhinocort)*.

PHARMACOLOGY

Cancer / Chemo

Which is the **most likely** complication seen with chemotherapy treatment?

- Renal failure
- Alopecia
- Peripheral neuropathy
- Glaucoma

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• **Alopecia**

Alopecia *(hair loss)* occurs with administration of most chemotherapeutic agents **one to two weeks after treatment.** Other common side effects include GI upset, increased incidence of infection *(especially Candidiasis)*, and degeneration of lymphatic tissue.

Remember: Most chemotherapy drugs have been shown to be **teratogenic in humans** and should be avoided by pregnant women.

Methotrexate may cause ulceration of the oral tissues.

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PHARMACOLOGY

Cancer Chemo

Your patient with a history of breast cancer is undergoing chemotherapy with a drug called **cisplatin** *(Platinol)*. What are the expected **adverse effects** of cisplatin?

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- **Nausea and vomiting** *(75% to 100%; dose-related)*
- **Hair loss** *(alopecia)*
- **Xerostomia**
- **Changes within the tissues of the oral cavity such as mucositis**

Mucositis is a common reaction to cancer chemotherapy. It is an inflammation of the mucous membranes. During chemotherapy and radiation therapy, mucosal tissues begin to desquamate and develop into ulcerations. The mucosal integrity is broken and is secondarily infected by oral flora. Palliative treatment is indicated for mucositis. The antineoplastics such as 5-fluorouracil *(5FU)*, methotrexate and doxorubicin are commonly associated with the development of oral mucositis.

The **vasoconstrictor epinephrine** in local anesthetic injections must be used cautiously in patients taking which of the antidepressant drugs in order to avoid transient and significant increases in blood pressure?

- Tricyclic antidepressants *(i.e., Elavil)*
- Selective serotonin reuptake inhibitors *(i.e., Prozac)*
- Serotonin and norepinephrine reuptake inhibitors *(i.e., Effexor)*

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- **Tricyclic antidepressants *(i.e., Elavil)***
- **Serotonin and norepinephrine reuptake inhibitors *(i.e., Effexor)***

These two categories of antidepressant drugs significantly increase norepinephrine levels in tissues. In the presence of a vasoconstrictor administered via a local anesthetic injection, the patient could experience significant elevation of blood pressure due to the vasopressor actions of the combination.

The selective serotonin reuptake inhibitors *(i.e., Prozac)* have no such effect on norepinephrine in tissues and interaction with a vasoconstrictor like epinephrine is not an issue.

Which group of **antidepressant** drugs has the highest incidence of **dry mouth** *(xerostomia)*?

- Tricyclic antidepressants *(i.e., Elavil)*
- Selective serotonin reuptake inhibitors *(i.e., Prozac)*
- Serotonin and norepinephrine reuptake inhibitors *(i.e., Effexor)*
- Monoamine oxidase inhibitors *(i.e., Nardil)*

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- **Tricyclic antidepressants *(i.e., Elavil)***
- **Serotonin and norepinephrine reuptake inhibitors *(i.e., Effexor)***

These two categories of antidepressant drugs **induce significant dry mouth** in up to 75% of patients taking these medications. These effects are due to the secondary anti-cholinergic nature of these agents. Drug-induced dry mouth must be treated palliatively with artificial salivary substitutes.

The **selective serotonin reuptake inhibitors** *(i.e., Prozac)* have no secondary anti-cholinergic effects and therefore **do not cause** any significant dry mouth.

Name the **five major** categories of **antidepressant drugs** and give examples of drugs in each class.

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Tricyclic antidepressants:
- Amitriptyline *(Elavil)*
- Doxepin *(Sinequan)*
- Imipramine *(Tofranil)*

Serotonin and norepinephrine reuptake inhibitors:
- Venlafaxine *(Effexor)*
- Nortriptyline *(Pamelor)*
- Desipramine *(Norpramin)*

Selective serotonin reuptake inhibitors:
- Citalopram (*Celexa)*
- Escitalopram *(Lexapro)*
- Fluoxetine *(Prozac)*
- Paroxetine *(Paxil)*
- Sertraline *(Zoloft)*

Second generation miscellaneous:
- Bupropion (Wellbutrin)
- Trazodone (Desyrel)
- Nefazodone (Serzone)
- Mirtazipine (Remeron)

Monoamine oxidase inhibitors:
- Phenelzine *(Nardil)*
- Tranylcypromine *(Parnate)*
- Isocarboxazide *(Marplan)*

Which antivirals are classified as neuraminidase inhibitors?

- Acyclovir *(Zovirax)*
- Oseltamivir *(Tamiflu)*
- Zanamivir *(Relenza)*
- All the above

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- **Oseltamivir *(Tamiflu)***
- **Zanamivir *(Relenza)***

Oseltamivir *(Tamiflu)* and zanamivir *(Relenza)* inhibit influenza virus neuraminidase enzymes, potentially altering virus particle aggregation and release. Tamiflu and Relenza are both used to treat acute illness due to influenza *(A or B)* infection.

Acyclovir *(Zovirax)* is an antiviral that inhibits DNA synthesis rather than neuraminidase enzymes.

Granisetron *(Kytril)* and ondansetron *(Zofran)* are selective 5-HT_3 receptor antagonists used to treat what condition?

- Breast cancer
- Osteoporosis
- Emesis caused by cancer chemotherapy
- Mucositis caused by radiation therapy

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• **Emesis caused by cancer chemotherapy**

5-HT_3 receptor is a serotonin receptor which when activated during chemotherapy for cancer, causes emesis *(nausea and vomiting)*. Both granisetron and ondansetron are indicated for prophylaxis of chemotherapy-related emesis, prophylaxis of nausea and vomiting associated with radiation therapy, and prophylaxis and treatment of postoperative nausea and vomiting *(PONV)*. 5-HT_3 stands for 5-hydroxytryptamine type 3 receptor.

PHARMACOLOGY

Misc.

The following drugs belong to what pharmaceutical class of agents: pimecrolimus; sirolimus; tacrolimus;

- Immune modulators
- Monoclonal antibodies
- Immunosuppressants
- Interferons
- Colony stimulating factors

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• **Immunosuppressants**

Pimecrolimus *(Elidel)* is an immunosuppressant agent used for treatment of mild to moderate atopic dermatitis.

Sirolimus *(Rapamune)* is an immunosuppressant agent used for prophylaxis of organ rejection in patients receiving renal transplants.

Tacrolimus *(Protopic)* is an immunosuppressant agent used to treat moderate to severe atopic dermatitis in patients not responsive to conventional therapy.

PHARMACOLOGY

Misc.

The following drugs belong to what pharmaceutical class of agents: adalimumab; alefacept; infliximab; trastuzumab?

- Immune modulators
- Monoclonal antibodies
- Immunosuppressants
- Interferons
- Colony stimulating factors

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- **Monoclonal antibodies**

Adalimumab *(Humira)* is a recombinant monoclonal antibody that binds to human tumor necrosis factor alpha *(TNF-alpha)* receptor sites. It is used to treat active rheumatoid arthritis.

Alefacept *(Amevive)* is a monoclonal antibody used to treat moderate to severe plaque psoriasis.

Infliximab *(Remicade)* is a monoclonal antibody used to treat ankylosing spondylitis, Crohn's disease, and rheumatoid arthritis. Like adalimumab, infliximab works by binding to TNF-alpha receptor sites.

Trastuzumab *(Herceptin)* is a monoclonal antibody which binds to the extracellular domain of the human epidermal growth factor receptor 2 protein *(HER-2)*. It is used for the treatment of patients with metastatic breast cancer whose tumors overexpress the HER-2 protein and who have not received chemotherapy for their metastatic disease.

PHARMACOLOGY

Misc.

Your patient's medication history includes modafinil *(Provigil®)*. Your patient is probably using this drug in order to:

- Improve wakefulness during daytime sleepiness
- Improve salivary flow in dry mouth disorders
- Manage psychotic disorder
- Treat mental depression

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• **Improve wakefulness during daytime sleepiness**

Modafinil *(Provigil®)* represents a new class of central nervous system stimulants used to improve wakefulness in patients with excessive daytime sleepiness associated with narcolepsy and shift work sleep disorder. It also has an unlabeled use to treat attention deficit/hyperactivity disorder *(ADHA)*.

The exact mechanism of modafinil is unclear; however part of its action may be do to decreased GABA-mediated neurotransmission. It is also thought to increase high-frequency alpha waves while decreasing both delta and theta activity, thus increasing mental alertness.