PREVENTION OF THE RETAINED FETAL MEMBRANE SYNDROME (RETAINED PLACENTA) DURING INDUCED CALVING IN DAIRY CATTLE

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ABSTRACT

Sixty-six dairy cattle were induced to calve with dexamethasone treatment at 5 d prior to expected time of calving. Each animal was assigned randomly to one of two treatments, saline (2 ml) or $PGF_2\alpha$ (10 mg), which were administered within 1 h postpartum. With the saline treatment, 90.5 % of the animals had placental retention, whereas only 8.8 % of the $PGF_2\alpha$-treated animals had placental retention. The $PGF_2\alpha$-treated cows released the fetal membranes in 7.4 $\pm$ 1.35 h postpartum, whereas the saline-treated cows released the membranes in 98.3 $\pm$ 10.93 h postpartum. These data demonstrate that treatment with $PGF_2\alpha$ within the immediate postpartum period is effective ($P < 0.001$) in the prevention of placental retention in the dairy cow induced to calve with dexamethasone.

Key words: bovine, placenta, retention, prevention, prostaglandin

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INTRODUCTION

Retention of the fetal membranes (retained placenta) is a syndrome that can occur in many of the domestic species, although it is only a serious problem in dairy cattle (1,2). A range of 8 to 30 % of all spontaneous calvings by dairy cattle are reported to result in the retention of the fetal membranes (1-3). Induced calving has not been a useful cattle management practice due to the high rate of placental retention (4). Numerous studies have been conducted to determine the causes of both spontaneous and induced placental retention and to develop a treatment to prevent this condition (5,6).

We have recently reported that the bovine fetal placental component (fetal villi) will produce prostaglandins predominantly of the E series (PGE) when obtained from dairy cattle that subsequently retain the placenta (7). In contrast, villi from cattle that subsequently release the placenta produce prostaglandins predominantly of the F series (PGF; 7). This same synthetic defect is implied by the differences in the prostaglandin content of bovine placentomes collected from individual cows that had retained or released the placenta as reported by Leidl et al. (8). This difference was further suggested by the studies of Horta (9) in which fetal membrane retention was induced by aspirin treatment. The prostaglandin synthesis inhibition produced by the aspirin and the resultant retention of the placenta could then be reversed by the administration of $PGF_2\alpha$. These observations suggest a failure of the fetal placental villi to shift from PGE to PGF production when the placenta is retained. Therefore, an experiment was designed to examine the efficacy of using PGF treatment to prevent placental retention in the model system of induced calving which results in a high incidence of placental retention.

MATERIALS AND METHODS

Sixty-six Holstein cows and heifers in late pregnancy were housed in stanchion barns at the University of Maryland, College Park, and fed balanced rations of corn silage, mixed grain and alfalfa-timothy hay. All animals were induced to calve with dexamethasone (20 mg i.m.; Schering Corp., Rathway, NJ) at an estimated 5 d prior to the expected time of calving. Immediately following calving, all cows and heifers were assigned randomly to one of two treatment groups. Animals were given multiple intramuscular injections of saline (2 ml, n = 26) or prostaglandin $F_2\alpha$ ($PGF_2\alpha$; 10 mg; n = 40; Lutalyse, Upjohn Co., Kalamazoo, MI) immediately following calving (within 1 h postpartum), at 4 h postpartum and at 8 h postpartum, or as described below. The time of calving, time of injections, time of placental release, incidence of calving difficulty (dystocia),

sex of calf and incidence of twins were recorded for all animals. The hours until fetal membrane release were calculated as the time (h) from calving until spontaneous placental release. Many of the animals that retained the placenta did so until it was released by manual separation. Animals were considered to have retained the placenta if the fetal membranes were not released within 12 h postpartum. Data were analyzed for significant treatment differences via Fisher's Exact Test.

RESULTS

Early in this experiment, many of the treated animals (n = 15) released the placental membranes prior to the administration of the injections at 4 and 8 h postpartum. For these $PGF_2\alpha$-treated animals, 9 cows received only a single injection at 1 h, 5 cows received both the 1 h and the 4 h injections and 1 cow received all 3 injections. Each of the animals receiving 1 or 2 $PGF_2\alpha$ injections released the placental membranes while the animal that received all 3 $PGF_2\alpha$ injections retained the placenta. The 5 saline-treated animals during this period received all 3 injections and retained the fetal membranes. These data suggested modification of the original experimental protocol to include a single injection only. Therefore, the remaining animals (n = 46) in this experiment received only the 1 h postpartum injection. This change was not observed to alter the results significantly ($P > 0.05$). However, only the results for the 55 animals that were given single injections of either treatment within 1 h postpartum are presented in detail (Table 1).

It is clear from the data (Table 1) that the incidence of placental retention differs ($P < 0.001$) between the treatment groups. The rate of placental retention was 90.5 % for saline-treated cows as compared to 8.8 % for the $PGF_2\alpha$-treated cows. The mean length of time from calving to fetal membrane release was 98.3 $\pm$ 10.93 h for the saline controls and 7.4 $\pm$ 1.35 h for the $PGF_2\alpha$-treated animals.

The incidence of calving difficulty and sex ratio of the calves (male/female) did not differ between the treatments. Each of the treatment groups had 2 calvings which resulted in twins. In these twin calvings, the control group retained the fetal membranes while the treated group released the membranes in less than 8 h. Fourteen of the saline-treated cows required manual removal of the placental membranes, whereas only 1 of the $PGF_2\alpha$-treated cows required manual removal. The placental tissue was manually removed only from those animals that had retained their placentas for more than 4 d postpartum. Additional management data were not collected for these animals, and complete information concerning subsequent reproductive/production performance is not currently available.

For 5 additional animals, the first injection of $PGF_2\alpha$ was not administered until 2 to 3 h postpartum. In these animals, the placental membranes were observed to be retained for more than 12 h postpartum.

Table 1. Percentage of cows releasing the fetal membranes at various postpartum intervals

Interval (time to placental release)	Treatment Group	
	Saline (n = 21)	$PGF_2\alpha$ (n = 34)
≤ 8 h :	9.5	76.5
8 - 12 h :	0	14.7
12 - 24 h :	14.3	5.9
> 24 h :	76.2	2.9

DISCUSSION

This experiment demonstrates that the administration of $PGF_2\alpha$ within 1 h postpartum is effective in reducing the incidence of placental retention for at least the induced calving model. These results support earlier findings (7-9) which suggest that a deficiency of PGF or a lack of the conversion of PGE to PGF is responsible for placental retention. In contrast, Bosu et al. (10) reported higher maternal plasma concentrations of prostaglandin $F_2\alpha$ metabolite (PGFM) at 7 and 9 d prepartum in animals that retain the fetal membranes. These prepartum differences in PGFM appear minimal compared to the PGFM concentrations that they report (10) for the immediate peripartum period. The plasma PGFM results reported by Bosu et al. (10) were from cows at times that are considerably earlier prepartum than the prepartum PGF results reported by this paper and others (7-9). Bosu et al. (10) also reported higher PGFM concentrations at 1, 3 and 18 d postpartum in animals that retain the placenta. These plasma PGFM results (10) are, however, at later postpartum periods than the placental prostaglandin results (7,8) which demonstrated lower local placental PGF concentrations postpartum in animals that retain the fetal membranes. Our current results also suggest that it is the concentration of PGF during the immediate peripartum period which may be important to placental release rather than later concentrations. These increased plasma PGFM concentrations at 1 d and later postpartum (10) may be

related to trauma and irritation associated with retained fetal membranes, as suggested by Lindell et al.(11), rather than to the processes which normally regulate placental release.

Martin et al. (12) reported no difference in uterine contractions (1 to 6 h postpartum) between animals that retain or release the placenta prior to the time of placental release. This suggests that differences in concentrations of PGF during this immediate postpartum period (7) may be related to placental release rather than to uterine motility.

Overall, the data indicate a useful method for ameliorating the placental retention syndrome under at least the conditions of induced calving. The need exists to demonstrate its effectiveness in spontaneously calving animals as well and to conduct an adequate, large-scale field study. The time frame for treatment to be effective appears to be confined to a 1-h postpartum period. This may restrict success from a management point of view. Attempts with other treatment regimens will be necessary to improve the practical utility of the technique.

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