Cow - Calf Session

Dr. Don Hudson, Presiding

Parturition: A Mechanism Review: Induction, Intervention, and Calf Viability

Marshall R. Putnam, D.V.M., M.S.

Dept. of Medicine and Surgery, Food Animal Section College of Veterinary Medicine Oklahoma State University Stillwater, Oklahoma 74078

Abstract

The role of the practitioner in food animal practice involves the health care of individual animals as well as the management and care of herds or flocks. Health care of individual animals often affords the opportunity to assume a management role if the practitioner is alert and prepared. Practitioners should seek ways to increase their role in the management of food animal production as the cost of individual care is becoming prohibitive in all but a select few animals.

An adequate understanding of the parturition process allows practitioners to properly train clients in parturition management to maximize production and minimize losses. There is enough good work concerning the effect of parturition on productivity to make substantiated recommendations aimed at increasing the producers' profitability.

Conclusions

- 1. Provided that dilation is complete (membranes present at the vulva), delivery assistance can be given rather than allow parturition to continue and/or wait the recommended 2-3 hours after labor starts. This has been shown to:
 - (1) Not harm but possibly increase calf vigor.
 - (2) Decrease calf losses at birth.
 - (3) Increase the postpartum reproductive performance in the beef cow.
- 2. The accepted length for Stage 2 of labor *may* be shorter than the 2-3 hours now reported.
- 3. Induction of parturition is most reliably done with dexamethasone.
- 4. Proper management of sire selection, pre-breeding nutrition, and pre-partum nutrition can increase productivity.

5. Heifers should be fed to weigh 65% of their mature weight at breeding (15 months) and 85% of their mature weight at calving (24 mos).

Mechanism Review

Initiation

The hormonal control of parturition appears to be a cascade of events, each step building on the next, bringing about termination of the pregnancy and expulsion of the fetus. The signal that initiates the cascade begins in the fetus and creates a response from the maternal endocrine system. In the bovine animal, much of the detail in the cascade of events lacks documented proof but, using what is known about similar species such as the goat and sheep, one can postulate the events leading to parturition in the bovine.

By definition, the cow is a corpus luteum dependent species, thus she is dependent on the corpus luteum as the major source of progesterone to maintain pregnancy. To initiate parturition, the fetus must cause a decline in progesterone through lysis of the corpus luteum.

Fetal Role

The role of the fetus in timing the initiation of parturition through an intact pituitary-adrenal axis is well accepted in most (cow, sheep, pig, and goat) domestic species (14,30,31,35,36,37,40,51,55,56,57).

The control of the fetal pituitary-adrenal axis has best been described in the sheep. In the intact fetus, cortisol production at term appears to be a result of changes in maturation of the fetal adrenal plus an increasing release of ACTH from the maturing fetal pituitary (13, 56). The increasing release of ACTH acts on the fetal adrenal stimulating increasing levels of cortisol production as the adrenal gland matures (14,56). There is a gradual rise in the concentration of cortisol in the fetal plasma beginning 10 to 15 days prepartum, which culminates in a more rapid rise during the last two to three days of gestation (3). Similar changes in fetal cortisol levels are documented in the bovine animal (16, 38).

The rise in fetal cortisol acts as the signal to the dam that the fetus is ready for expulsion. Rising fetal cortisol levels also act to prepare the fetus for survival outside the uterine environment. Investigators have demonstrated that glucocorticoids accelerate lung maturity by the appearance of pulmonary surfactant (25). Glucocorticoids are also known to be involved in the maturation and biochemical differentiation of other tissues in the fetus (14). Thus the need for exogenous steroid priming of the fetus being taken by cesearean section before natural cortisol rises (last 8-10 days).

In initiating the events leading to parturition, fetal cortisol acts on the placenta. In sheep, the action of rising fetal cortisol on the placenta is well understood while corpus luteum dependent species have not received as much attention, and much of the action of fetal cortisol is still theoretical. Like the sheep, the cow experiences a rise in maternal estrogen and a fall in maternal progestins preceding parturition. A rise in fetal cortisol appears to trigger these maternal endocrine changes as in the sheep (16, 20).

Most of the work in the corpus luteum dependent species has been done in the goat. It has been proposed that the rising fetal corticol levels activate enzymes in the placenta as in the sheep, leading to increased placental estrogen secretion. After exposure to glucocorticoids, the caprine placenta contains the enzymes necessary for synthesis of estrogens which are rising dramatically as fetal cortisol increases (32, 55). Flint *et al.* (32) showed that the increase in fetal cortisol levels preceding parturition activates the 17-alpha hydroxylase enzyme system in the caprine placenta and increases estrogen production.

In the bovine animal, fetal cortisol increases dramatically during the last ten days of gestation (16, 38). Cattle are similar to the goats in placentation, enzyme systems, and endocrine control of parturition thus thought to respond to fetal cortisol similar to the goat (31).

Maternal Role in Parturition

The maternal role in parturition can be described as completing a process initiated by a signal from the fetus. The process carried out by the dam is seen as a fall in progesterone, a rise in estrogens, peaking prostaglandin levels just prior to parturition, and increases in oxytocin release just prior to and during parturition (35). Again, most of the work has been done in the goat as an example for the bovine animal because of the similarities in the two species.

Since the corpus luteum is the major source of proges-

terone in cattle, the fetus must alter maintenance and / or lyse the corpus to initiate parturition.

Luteal Support

Maintenance of the corpus luteum appears to fall into two categories in the caprine species: Placental origin and pituitary origin.

Placental Origin. From the placenta, there appears to be a substance caprine placenta lactogen (CPL) which is necessary for luteal support during pregnancy (12). CPL concentrations fall progressively during the last 15 days of pregnancy when corticosteroid concentrations were rising in fetal circulation. Similar, but more abrupt, changes were noted when corticotrophin was administered to the fetal goat raising the possibility that increasing fetal cortisol levels might inhibit the ability of the placenta to elaborate CPL (19, 21). If CPL is the placental luteotrophin in the goat, its withdrawal may act in conjunction with discrete prostaglandin F (alpha) (PGF) releases to initiate luteolysis (40).

In cattle, the structure of bovine placental lactogen has been reported (7).

Pituitary Origin. The maternal pituitary gland through LH secretion is also essential for luteal support in the pregnant goat. Hypophysectomy results in abortion while hypophysectomy followed by LH replacement resulted in the pregnancy being maintained (11).

Progesterone

The source of progesterone in both the caprine and bovine species is the corpus luteum (30,57). The action of progesterone in maintaining pregnancy is two fold. It inhibits prostaglandin release and blocks myometrial contractions.

The mechanism for blocking prostaglandin release is not known. but repeated doses of exogenous progesterone in cycling ewes resulted in an increase in prostaglandin release with each fall in progesterone. This indicates that progesterone blocks prostaglandin release and with progesterone decline prostaglandins are released (21).

The effect of progesterone on the myometrium to prevent myometrial contractions is mediated through controlling calcium ion transport across the cell membrane. At the present, calcium ions are the only known stimulant which directly triggers the actinomyosin-ATP interaction and thus initiates the cyclic myometrial contractile force of labor. Progesterone promotes binding of this "activator calcium" thus preventing myometrial contraction. Prevention of the activator calcium from initiating myometrial contractions occurs even in the presence of high levels of exogenous prostaglandin which normally lowers the threshold of excitement and allows the inflow of calcium ions into the cell (18).

Prior to parturition, the concentration of progestogens in

 \bigcirc

maternal blood plasma decreases gradually during the last 20 days of gestation and fall dramatically two to three days before parturition (54). The regression of the corpus luteum and dramatic fall of progesterone two to three days prior to parturition is the subject drawing the most interest in the literature.

Estrogens

The role of estrogens in the initiation of parturition is receiving more interest in the literature. The unconjugated estrogens first appear in significant amounts in maternal plasma around days 40 to 50 of gestation in the goat. Estrogens continue to increase steadily during pregnancy before undergoing a rapid increase over the last four to five days prior to parturition (15, 21, 23, 58, 61). The estrogens present in the fetal and maternal goats are mainly estrone and estradiol 17-alpha, both considered biologically weak, and variable amounts of estradiol 17-beta (58).

The source of rising estrogen concentration late in gestation is the placenta (55). Under the influence of fetal cortisol, the caprine placenta possesses the enzymes necessary for the production of estrogen. Although no actual increase of *In Vivo* estrogen production has been documented, the probability cannot be dismissed that the placenta is in fact producing estrogen, possibly from progesterone precursors (30, 55).

The action of estrogens in the control of parturition are varied. The possibility exists that estrogens play a dual role in control of the corpus luteum. A situation may exist in the goat where the progressive increase in estrogen (17-alph and estrone) concentrations may lead to a luteotrophic complex late in gestation. But, studies have shown that estradiol 17beta is luteolytic in the goat probably through enhanced prostaglandin production and release during falling progesterone levels (21). The action of estradiol 17-beta causing the release of prostaglandin may come from the estradiol 17beta traversing the placental barrier and acting on estrogen receptors in the maternal placenta that respond by increasing PGF production. This estrogen receptor population in the placenta and endometrium may be regulated by progesterone levels (55). Thus the event of falling progesterone levels would increase the estrogen receptor population in the placenta, enhancing the production and release of prostaglandins as estrogens rise. Similar changes in estrogen levels in the cow lead to the assumptions that the actions of estrogens are much the same as the goat.

Estradiol may also play an important role in oxytocin receptor population control in the placenta and myometrium (22). Roberts *et al* (48) have shown that in sheep the oxytocin receptor population in the endometrium and myometrium varies during the estrous cycle and that estrogen increases receptor concentration.

Prostaglandins

The role of prostaglandins in pregnancy and parturition

appears to be dual: lysis of the corpus luteum and contracture of the myometrium (56). The source of the prostaglandins appears to be the fetal membranes and maternal endometrium.

Prostaglandin lysis of the corpus luteum is still debated in the literature. Hoffman (36) states that the prostaglandin release appears to be a coincidence of the prepartum progesterone decline rather than a cause. Umo *et al* (61) have shown that the initial PGF increases occur 48 hours prior to term delivery and precedes the decrease in progesterone levels by 18 to 22 hours.

Prostaglandin synthesis and release appear to be under the control of changes in placental progesterone and estrogen production (56). As has been previously stated, progesterone blocks prostaglandin release, and estradiol 17beta stimulates production and release. In experiments by Liggins *et al* (41), the estrogen-induced release of prostaglandin was blocked by pharmacological amounts of progesterone (200 mg/day). However, the rise in the concentration of prostaglandins in the endometrium and maternal cotyledon was not suppressed indicating that progesterone appeared to inhibit release but not the synthesis.

The role of prostaglandin in myometrial contractions involves calcium ion transport. PGF lowers the excitation threshold allowing calcium ions to enter the cell and activate actinomyosin and ATP for myometrial contractions. Thus the reason for uterine inertia during parturition in hypocalcemia. The presence of progesterone blocks this effect, even in the presence of PGF by raising the threshold for calcium transport (18).

Prostaglandin also appears to soften the cervix. In the presence of low progesterone levels, infusion of prostaglandins into the arterial blood supply of the uterus and cervix of sheep resulted in cervical relaxation (29).

Oxytocin

The source of oxytocin is the posterior pituitary. Oxytocin plays an important role in the second stage of labor. Distension of the vagina and cervix causes the release of oxytocin (24, 28).

The release of oxytocin in response to vaginal distension is enhanced by estrogen and prostaglandin but inhibited by progesterone (50). The massive releases of PGF during labor allows some of the PGF to escape lung metabolism and stimulate oxytocin release in positive feedback cascade (21,33).

Oxytocin functions like PGF by causing an influx of the activator calcium into the cell.

Stage I

Stage I begins with the initiation of parturition by the fetus and ends when cervical dilation is complete allowing the membranes to protrude into the pelvis.

The events leading to this are initiated by the rising estradiol 17-beta levels increasing the production and release of PGF. Increasing PGF levels cause the estrogen sensitized myometrium to contract and force the fetal membranes against a softened cervix stimulating oxytocin release. These actions feed on each other until the fetus is expelled.

Stage II

With respect to management of parturition, Stage II is the most important. Stage II is characterized by the entrance of the fetus or fetuses into the dilated birth canal, rupture of the allantoic sac, abdominal contractions, and expulsion of the fetus through the vulva (49). Reported durations of Stage II fall within a range of 0.5-4.0 hours (49, 52, 53) with reported duration averages of 0.5 to 3.0 hours. Assistance is recommended if the duration of Stage II exceeds 2.0-3.0 hours.

Correct management of parturition should be aimed at reducing losses by controlling factors affecting the birth process. Prolonged or difficult (dystocia) parturition results in greater calf losses at birth and reduced postpartum reproductive performance. Dystocias were responsible for 60 percent of all deaths at or near birth and amounted to 38 percent of all losses including abortion, stillbirth, and postnatal death (43). Brinks *et al* (8) showed that beef heifers experiencing calving difficulty as 2-year-olds weaned 11 percent fewer calves the first year and 14 percent fewer calves per cow exposed the next year when compared to herdmates having no difficulty. Also, calves born the second year were 13 days later due to delayed return to estrus in the cows and averaged 46 lbs. lighter at weaning.

The *causes of dystocia* are multiple and complex but have been classified and ranked in order of importance: (1) disproportionate size between fetus and maternal pelvis accounting for over 50 percent of all dystocias, (2) malpresentation, posture, or position, (3) uterine inertia and incomplete cervical dilation, and (4) miscellaneous causes (47). Discussion will be limited to the most manageable cause, the conflict between fetal size and maternal pelvic opening.

Calf Birth Weight

Calf birth weight is the most important causative factor associated with dystocia. Burfening *et al* (9) showed a high genetic correlation (0.85) between birth weight and calving difficulty, with birth weight being a moderately inheritable trait. Calf birth weight is mainly a function of the calves' genotype with maternal environment accounting for some variability. Thus, if the maternal environment (size and nutrition) is optimum, the fetal birth weight will be expressed as its genetic potential from the sire and dam (47).

Sire selection can greatly affect birth weight and therefore perinatal losses. Data from the U.S. Meat Animal Research Center in Clay Center, Nebraska (1), demonstrates the effect

APRIL,	1983
--------	------

TABLE 1.	U.S. Meat Animal Research Center. Summary of Calving
	Difficulty by Breed of Sire in Two-Year-Old Hereford and
	Angus Dams (1).

Breed of Sire	(lb.) Av. Birth Wt.	% Difficult Births	% Dead at Birth
Jersey	58	20	4
Angu\$	66	41	8
Hereford	69	46	5
So. Devon	72	55	10
Limousin	74	75	9
Simmental	76	74	13
Charolais	77	77	14
Average:	69 lbs.	51%	8%

TABLE 2 U.S. Meat Animal Research Center. Summary of Difficult Births by Breed of Sire in Mature Hereford and Angus Cows (1).

Breed of Sire	(lb.) Av. Birth Wt.	% Difficult Births	% Dead at Birth
Jersey Angus Hereford So. Devon	71	5	2
Limousin Simmental Charolais	84	21	6
Average:	79 lbs.	14%	5%

of different breeds on dystocia and death loss at parturition in Hereford and Angus heifers (Table 1). The high birth weights from the larger breeds on small heifers resulted in very high dystocia and death rates.

The same sires were mated to mature adult Hereford and Angus cows. Even though the birth weights are higher, the incidence of difficult births is lower due to the increased size of the mature animals. But, again, in the sires producing the heavier calves (greater than 80 lbs), the dystocia rate was 21% versus 5% in the sires with an average birth weight of less than 80 lbs. The death rate for the large calves was 6% versus 2% for the smaller calves (Table 2). Thus a general conclusion can be drawn that heifers and small frame cows should not be bred to exotic breeds with large birth weights.

A review of the calving data from the Simmental Association showed that as the industry selects for greater yearling weights, the birth weights also increase due to the high correlation between the two weights. The report also pointed out that there are some bulls with high yearling weights yet relatively low progeny birth weights. These bulls had progeny performing above average at weaning and 365 days but with progeny birth weights below average (10). Thus, it is possible to select sires with above average progeny performance yet have progeny birth weights below average. This is especially important for first-calf heifers since the effect of birth weight is greater.

Maternal Environment.

Maternal environment influences the size of calves born regardless of the genetic potential. As stated, if the maternal environment is optimum, then the fetal birth weight will be expressed as its genetic potential but, if the maternal environment is restricted, the birth weight will be restricted accordingly. This is demonstrated in Table 3 showing the results of reciprocal crosses between South Devon (1200 lbs. mature wt.) and Dexter (615 lbs. mature wt.)

Dexter dams bred to South Devon bulls had calves only 6 lbs. above the Dexter purebred average while the Dexter bull sired calves 20 lbs. above the purebred average in South Devon dams (39). This demonstrates the effect of maternal genetics and environment in relation to the sire.

Where the maternal environment is severly restricted the birth weight will also be reduced and increased where the maternal environment is optimum. This does not consider the dystocia rate or death losses, only the birth weight.

Nutrition Effects: Fetal & Maternal

Fetal. The role of nutrition on dystocia and perinatal loss is a popular subject. Many producers believe that reducing the energy intake prepartum reduces the incidence of dystocia. Hammond (34) has demonstrated that the energy requirement for growth of the fetus once the cow is pregnant is second only to the energy requirement for the brain and nervous tissue of the dam. Thus, growth of the fetus will continue to occur even at the expense of maternal bone and muscle and fat disposition. This would indicate that reducing the energy intake of cows prepartum would have little effect on the birth weight of the fetus.

Researchers have proven that the only real effect of reducing the energy intake on heifers is that the subsequent cycling and pregnancy rates are reduced. Work by researchers at Ft. Robinson (60) showed the effects of feed level in the prepartum period on dystocia and death loss (Table 4).

There was no difference in the percent of dystocias or death rate even though birth weight was reduced 7 lbs (10%). But, the moderate group had excellent postpartum cycling rates while the low group had poor postpartum cycling rates.

Idaho State (27) researchers fed three groups of heifers 100%, 85%, and 75% of the NRC requirements (Table 5). The interesting points were: (1) all had good rates of gain (75% = 0.8 lbs/day), (2) average birth weights for all groups was 65 lbs. with no difference between the groups, (3) 75%

TABLE 3. Material Influence of Calf Birth Weight in South Devon-Dexter Crossbreeds (39).

Breed of Sire	Breed of Dam	Gestation Length	Birth Weight (lb.)
South Devon	South Devon	287 days	97
Dexter	Dexter	287 days	53
South Devon	Dexter	278 days	59
Dexter	South Devon	290 days	73
Two breed average South Devon mature Dexter mature cow v	cow weight averag	e is 1200 pound	75 s.

TABLE 4. Effect of Feed Levels on Birth Weights and Calving Difficulty at Ft. Robinson (60).

Feed level and weight gain					
120 days prepartum	No.	% Difficulty	Birth Wt.	% Deaths	
Moderate (150 lb. gain)	140	36%	70 lbs.	8%	
Low (35 lb. gain)	94	33%	63 lbs.	6%	

TABLE 5. Nutrition and Calving Performance, Idaho State University (27).

Nutritional Group	Average Daily Gain	Weight At Calving	Unassisted Births	Calf Weaning Wt.
(150 days preca	alving)			
100% NRC	1.3 lbs.	946 lbs.	70%	394 lbs.
85% NRC	1.2 lbs.	922 lbs.	65%	370 lbs.
75% NRC	0.8 lbs.	865 lbs.	54%	363 lbs.

TABLE 6. Effect of Restricting Prepartum Energy on Cow and Calf Traits (17).

	Energ		
Item	High	Low	Difference
Prepartum wt. change (lbs.)	79.4	-12.8	92.2
Birth wt. (lbs.)	67.3	62.9	4.4
Assisted births (%)	27.0	28.0	-1.0
Calves alive at birth	97	90	7
Milk production (kg/day)	10.6	11.0	-0.4
Weaning wt. (lbs.)	352	325	27
Percent in estrus by 40 days postpartum	41	26	15

NRC group had less unassisted births with 10% fewer calves weaned averaging 30 lbs under the other groups, and (4) the 75% NRC group also averaged 15 days later coming in to heat than the 100% group.

Corah *et al* (17) showed the effect of prepartum energy on cow and calf traits and the effects of high and low prepartum energy on calving (Tables 6 and 7). For 100 days prepartum,

he fed the high group 100% of the NRC requirment and the low group 65% of the NRC energy requirement. There was no difference in the assisted births with a slight decrease in the birth weights for the low group. In the low group there were fewer (7) calves alive at birth with a 27-lb. decrease in weaning weight and 15% fewer heifers cycling by 40 days postpartum in the low group.

The effect of low versus low-and-high prepartum energy in the last 30 days was lower birth weights in the group receiving low prepartum nutrition versus low-then-high prepartum nutrition. But, a higher number of calves survived to weaning and fewer calves scouring with less deaths and a shorter interval to first estrus occured in cows receiving low-high prepartum nutrition.

Maternal. Many of the factors associated with dystocia are already set at breeding. The growth rate of the heifer prior to breeding has been shown to be very important in reducing dystocia. Recommendations are that the heifers should be at least 65% of their mature weight at breeding and calf as 2-year-olds weighting approximately 85% of their mature weight (45). Bellows (5) has shown that heifers fed at a low rate of gain (0.6 lbs/day) to breeding have much higher calving difficulty as 2-year-olds. Also, fewer reach puberty at breeding and, when they did calve, their smaller pelvic opening created a greater instance of dystocia.

Calving the heifers at three-year-olds to gain greater size at parturition doesn't help. (Tables 8 and 9). This data shows that the mortality rate was the same even though the heifers had larger pelvic openings. The calves were larger; the number of dystocias was markedly less; but the mortality was the same. Even with the small birth weight (62 lbs) in heifers at Colorado State the number of dystocias was 83% because of the small pelvic opening (184 sq cm). (46) This shows the relationship between pelvic opening and fetal size. A combination of both factors is needed to achieve less dystocia and deaths not just a large pelvic opening or small calf alone.

This can be best achieved by breeding heifers as 2-yearolds (maximize lifetime productivity) at 65% of their mature weight to a bull selected for calving ease (low mortality and birth weights) and calve at 85% of mature weight.

Thus the effect of lower nutrition is a varied effect on birth weight, increased age at puberty, a significant reduction in postpartum reproduction potential, greater number of dystocias, and greater calf mortality.

The effect of protein on dystocia is still not clear. In 1979, Bellows *et al.* (4) showed the effect of high protein was to increase dystocia and weaning weight. But, in a report at the 1982 Western Section Meeting of the Animal Science Society, a similar study by Anthony *et al.* (2), showed no effect on high dietary protein on calf birth weight or instance of dystocia. More work needs to be done to clarify the effect, if any, of dietary protein on dystocia.

The effect of altering feeding schedule on the incidence of

TABLE 7.	Effect of Restricting Prepartum Energy Intake and Elevat-
	ing it 30 Days Prior to Calving (17).

	Continuous low*	Low-High**
	Energy	Level
Prepartum wt. change	-142	-22
Birth weight	59	76
Calf survival to weaning	71	90
Calf scours		
% affected	52	33
% mortality	19	0
Postpartum interval	50	42

*50% of energy requirement for last 100 days prepartum.

**50% of energy requirement for days -100 to -30 then switched to 117% for last 30 days prepartum.

TABLE 8. Calving Difficulty and Calf Losses in 2- and 3-year-old First-Calf Hereford and Angus Heifers.

	2-year-olds		3-year-olds	
Source	% Difficultues	% Deaths	% Difficultues	% Deaths
Miles City (1966-1969) (6)	42%	4%	17%	6%
Colorado State University (46)	83%	9%	_	_
Ft. Robinson (1963-1964) (60)	34%	7%	16%	8%

TABLE 9. Comparison of Calf Birth Weight and Dam Pelvic Area in Hereford and Angus Heifers.

	2-year-ol	2-year-olds		3-year-olds	
Location	Dam Pelvic Area (sq. cm.)	Calf Birth Wt. (lbs.)	Dam Pelvic Area (sq. cm.)	Calf Birth Wt. (Ibs.)	
Miles City	250	72	292	78	
Colorado State University	184	62	_	_	
Ft. Robinson	range: 150-220	65	range: 210-28	0 76	

night calving lacks solid reported research. Burfening (9) reported on personal communication from two researchers (Bellows and Rahnefeld) who were able to reduce night calving by 17%, 9%, and 13% over a controlled group by feeding at 10:00 P.M. rather than the traditional morning feeding.

State III

By definition, Stage III of parturition is the shedding of the fetal membranes and involution of the uterus. In the cow, the membranes are shed within 0.5-8 hours, after which they are considered retained. Involution of the normal uterus occurs in two stages: physical and microscopic. Physically palpable involution should occur in 14-21 days while microscopic involution (epithelization of caruncles) doesn't occur until 25-30 days postpartum (49).

Induction

Induction of parturition is a common management tool for individual animals as well as a herd basis to bring about parturition at a more timely and manageable fashion. Parturition is sometimes induced attempting to reduce birth weights, and decreasing dystocias. This has had varied success. Moody and Han (42) observed that when cows were induced from 0-15 days early, the birth weight was reduced 7.1% but with no reduction in the number of cows requiring assistance. They also reported a 40.5% increase in retained fetal membranes, a decrease of 22 lbs. in the weaning weight of the calves, and a 8.4% reduction in subsequent pregnancy rates. Wagner (59) reported no effect on fertility in cows induced to parturition early.

For induction of parturition, the cow should be 270 days of gestation or greater with milk present in the teats. Calf survival should be normal provided that induction doesn't occur too early.

The most reliable agent for induction of parturition is still dexamethasone, 20 mg given as a single intramuscular injection. Parturition usually occurs less than 70 hours postinjection with an average of 40-48 hours (59). The mechanism by which dexamethasone works is well understood. The exogenous steroid mimics the fetal cortisol and acts on the placenta from the maternal side in the same fashion as fetal cortisol.

Prostaglandins can also be used to induce parturition but with varied success. The action is probably through lysis of the corpus luteum but still not proven. The success of PGF appears to depend on the estrogen priming of the uterus and will have inconsistent results if the cow is not close enough to parturition for the estrogens to be rising from fetal cortisol priming.

Complications from induction are: increased calf mortality when done too early and increased instance of retained fetal membrane with its debateable results. Other agents which have been shown to cause abortions and should be used with caution in pregnant cows are estrogens, smooth muscle stimulants such as neostigmine and oxytocin, Rompun[®], and sodium iodide.

Intervention—Calf Viability

Timely intervention of Stage II is important in reducing calf and dam losses. The effects of dystocia on calf mortality and postpartum reproduction performance in the dam have already been discussed.

At present, intervention is recommended if Stage II has progressed up to 2-3 hours and no progress is noted (49,52). Recent work has shown that the duration of Stage II may be TABLE 10 Least-Squares Means of Calving Data (26).

Item	Number	Calving difficulty score	Duration labor ª (minutes)	Calf birth wt. (kg)	Calf vigor score ^b
Age of Dam					
Heifer	62	2.00**	54.1**	32.4	1.17*
Cow	65	1.41	22.5	35.0**	1.01
Gestation Feed L	evel				
Moderate	63	1.74	41.0	33.6	1.06
High	64	1.68	35.6	33.7	1.11
Obstetrical Assis	stance				
Early	67	2.13**	_	33.8	1.07
Late	60	1.28	38.3	33.5	1.11
Calf Sex					
Male	61	1.78	42.4	34.3°	1.10
Female	66	1.63	34.2	33.0	1.08
df/ms	116	.4	666.1	14.8	.1

a Stage II labor; late assisted dams only (ns = 24 and 31, heifers and cows, respectively).

b Score: 1 = live, vigorous calf; 2 = live, slow or weak calf; 3 = dead calf. cP less than .10

*P less than .05 **P less than .01.

TABLE 11.	Postpartum	Reproductive	Performance,	Main	Effects	(26).	
-----------	------------	--------------	--------------	------	---------	-------	--

ltems Number		% in estrus Postpartum by begin interval breeding (days) season		Services per conception	October pregnancy (%)	
Age of Dam						
Heifer	62	62.4**	78.4	1.09	78.2	
Cow	65	44.3	94.8**	1.15	86.9	
Gestation Feed L	evel					
Moderate	63	54.5	83.7	1.11	87.8	
High	64	52.2	89.4	1.13	77.2	
Obstetrical Assis	tance					
Early	67	52.4	91.4ª	1.07	89.5*	
Late	60	54.4	81.7	1.17ª	75.6	
Calf Sex						
Male	61	53.8	93.8*	1.12	80.0	
Female	66	52.9	79.4	1.12	85.0	
df/ms	116	354.9	0.1	0.1	0.1	

a P less than .10.

*P less than .05. **P less than .01.

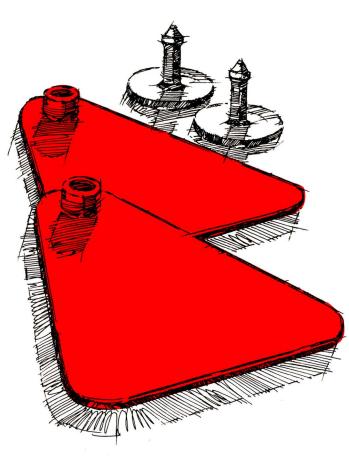
"P less than .01

TABLE 12	Pelvic	Area	and	Length	of	Stage	11	(44)).
----------	--------	------	-----	--------	----	-------	----	------	----

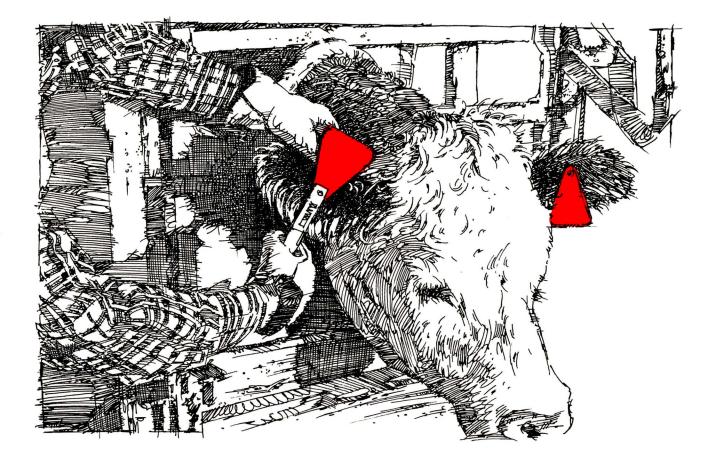
	Large Pelvic Area	Small Pelvic Area
Number of animals	23	23
Length of Stage II (minutes)	68.04	112.61
Number of Calves Not Nursing Within 2 Hours Postpartum	а З	7

a P less than .01.

A complete bovine herd health program should include convenient external parasite control.



New Wellcome® Insecticide Ear Tag. A major opportunity to improve your herd health programs.



The insecticide impregnated ear tag is a fast growing method of controlling cattle pests. So chances are, you'll hear the question this spring, "What about insecticide ear tags?" Now you have a good answer. Wellcome Insecticide Ear Tags. These tags provide an excellent opportunity to conveniently incorporate pest control as part of your total recommended herd health care program.

Traditional control measures, such as sprayers and backrubbers, are not popular dispensing items with veterinarians. Now insecticide ear tags fit conveniently into professional practice. In fact, in a three state test market last year, practitioners successfully included Wellcome Insecticide Ear Tags in their herd health programs.

Controls pests on beef and lactating dairy cattle.

More and more cattlemen are discovering the benefits of insecticide ear tags. It is probable that 25% of all pastured cattle will be tagged in 1983 and projections indicate this percentage will continue to grow. Whether you dispense tags or apply them yourself, you can participate in this trend using Wellcome Insecticide Ear Tags.

Season-long control.*

Wellcome Insecticide Ear Tags contain permethrin, a residual synthetic pyrethroid. Synthetic pyrethroids have a much higher degree of bioactivity than other insecticides.

The slow release of permethrin from Wellcome Insecticide Ear Tags produces the long-term effect. Specifically, season-long* control of face flies, hom flies and Gulf Coast ticks. They also control spinose ear ticks, and aid in the control of stable flies and house flies.

The insecticide rubs off the tag and onto the animal. The natural movements of the animal dispenses insecticide over its body, not just around the head.

Low toxicity.

Because of the high degree of insecticidal bioactivity only small amounts of permethrin are required for effective fly and tick control. Synthetic pyrethroids have a lower mammalian toxicity than other insecticides.

The ear tag formulation itself reduces the toxic hazard potential of external parasite control. Accidental overdosing of animals and contamination of the environment from spray drift or spillage are virtually eliminated.

Convenient application.

Wellcome Insecticide Ear Tags can fit perfectly into your schedule and be applied during your spring visit to the farm. One application then takes care of indicated external parasites for the whole season.

These tags utilize the dependable Allflex[®] Tagging System. Field use has shown this system to be virtually trouble-free. Many of your clients may already own Allflex applicators. Simply apply the tags in the ears of cattle according to label directions.

One tag per animal provides effective horn fly control. In cow/calf operations, you can achieve horn fly control by tagging the cows only.

Tag cows and calves with two tags each for effective face fly and/or tick control.

*Effective up to 5 months for control of face flies, Gulf Coast ticks and horn flies. This constitutes season-long control in most areas of the U.S.

More important points about Wellcome Insecticide Ear Tags.

Less trouble than other insecticide programs.

Other methods such as backrubbers and sprayers require expensive, maintenanceintensive application equipment. Wellcome Insecticide Ear Tags require only tagger pliers and the supplied button. And because one application provides season-long* control, ear tags save a significant amount of labor over dust bags, backrubbers and sprayers.

Not affected by rain or sunlight.

The insecticide in the Wellcome tag is incorporated into the plastic itself. It will not wash away. Permethrin is also stable in sunlight.

Designed primarily for pasture cattle.

Wellcome Insecticide Ear Tags primarily control pests of pasture cattle—horn flies and face flies. They work very well on pastured cattle. In confinement areas, however, where the principal pests are house flies and stable flies, ear tags should not be used as the only insect control measure.

Kills important disease vectors.

Wellcome Insecticide Ear Tags control face flies, proven capable transmitters of *Moraxella bovis*, generally considered the bacterium causing "pinkeye" (bovine infectious keratoconjunctivitis).

Kills irritating pests that can hamper production.

Annoying flies and ticks have been shown to reduce weight gains and milk production. Wellcome Insecticide Ear Tags control the source of the problem by providing continuous on-animal control.

The easy way to stock insecticide.

At two tags per animal, your clients could be asking for a large number of tags this season. The tag formulation means that sufficient insecticide to control a season's* pests on 100 cattle (at two tags each) is packed into one small case $(11'' \times 11'' \times 8'')$. Each case of 200 tags contains four display cartons, each containing five labelled plastic bags of ten tags and applicator buttons. Wellcome Insecticide Ear Tags are sold only to veterinarians. Allflex® Ear Tag Applicators are also available at your Wellcome Branch.



*Effective up to 5 months for control of face flies, Gulf Coast ticks and horn flies. This constitutes season-long control in most areas of the U.S.

WELLCOME ANIMAL HEALTH DIVISION BURROUGHS WELLCOME CO. KANSAS CITY, MO 64108 U.S.A.

shorter than now accepted. In 24 cows, Doornbos *et al.* (26) reported an average duration of 22.5 minutes and an average duration of 54.1 minutes in 31 2-year-old heifers. There tended to be less calf vigor with the increased length of Stage II. (Table 10)

In a group of 46 30-month-old hereford heifers, Putnam et al. (44) reported an average duration of 63.4 minutes in the 32 unassisted. The heifers were then grouped according to their pelvic area (Table 12). Unsurprisingly, heifers with the smaller pelvic areas had significantly longer intervals in Stage II. Within a 2 hour limit, seven calves were not able to nurse unassisted in the small pelvic area heifers while three calves needed assistance to nurse in the large pelvic area heifers. Both of these traits show lower calf vigor with the increased length of Stage II. These data need repeating. If the average length of unassisted Stage II is significantly shorter then dystocia would be occuring much sooner than we now recognize. More data is needed to further substantiate the effects of Stage II on calf viability.

Presently early assistance is not recommended unless obvious dystocia exists and is thought to do more harm than good (49,52). Doornbos et al. (26) examined the effect of early obstetrical assistance on cows and heifers. He grouped the cattle in late or early assistance groups. The late group was allowed to progress in parturition until parturition was completed or assistance was needed to save the calf. The early assistance group was allowed progression through Stage I into early Stage II to complete cervical dilation. The heifers were examined for dilation once the fetal membranes or parts were visible at the vulva, and the calf was then delivered. He reported that early assistance had not effect on calf viability or performance from birth to weaning. The most interesting findings were found by comparing the postpartum period for the early and late groups (Tables 10 and 11). Cows receiving early assistance showed a significantly higher number of animals cycling at the start of breeding season, a lower number of service per conception, and a higher fall pregnancy rate compared to the cattle allowed to calve unassisted until assistance was needed to save the calf.

Although unrepeated at this time these studies would indicate that the length of Stage II has an effect on calf viability and that timely and **properly administered** early obstetrical assistance is not harmful to the cow or calf. Early assistance seems to provide improved postpartum reproductive performance, with no detrimental, but a potential beneficial, effect on calf viability. These observations may warrant recommendations for obstetrical assistance once cervical dilation is complete (fetal membranes or parts visible at vulva) rather than "allow nature to take its course," although not practical in all situations. This philosophy provides prevention rather than treatment of a potential problem (dystocia) and could be applied in some management practices.

References

1. Anon. 1975, Germ Plasm Evaluation Program Progress Report No. 2, USMARC ARS-NC-22. 2. Anthony, R.V., R.A. Bellows, R.E. Short, R.B. Staigmiller, C.C. Kaltenbach and T.G. Dunn. "Effects of Dietary Protein Level on Prepartum Beef Heifers. Proc. West. Sec. Amer. Anim. Sci. (1982) 33:151. 3. Bassett, J.M., and G.D. Thorburn. "Fetal Plasma Corticosteroids and the Initiation of Parturition in the Sheep." Journal of Endocrinology, 44 (1969), 285-286. 4. Bellows, R.A., J.B. Carr, D.J. Patterson, O.O. Thomas, J.H. Kellen and W.L. Milmine. "Effects of Ration Protein Content on Dystocia and Reproduction in Beef Heifers." Proc. West. Sec. Amer. Soc. Anim. Sci. (1979) 29, 263. 5. Bellows, R.A. "Developing Replacement Heifers." Presented at: National Extension-Industry Invitational Workshop on Beef Cattle Reproductive Management. Oklahoma City, OK. October 30, 1978. 6. Bellows, R.A. "Calf Losses in Beef Cattle." Proceedings of the 5th Conference on Artificial Insemination of Beef Cattle (1971) p. 9. 7. Bolander, F.F., Jr., and R.E. Fellows. "Purification and Characterization of Bovine Placental Lactogen." Journal of Biol. Chem., 251 (1976) (, 2703-2708. 8. Brinks, J.S., J.E. Olson and E.J. Corroll. "Calving Difficulty and Its Association, with Sebsequent Productivity in Herefords." J. Anim. Sci. (1973), 36:11. 9. Burfening, P.J. "Effects of Dystocia on Future Reproductive Performance." Proceedings Soc. for Theriogenology (1981), 96-105. 10. Burfening, P.J. "Possible Effects on Growth With Selection for Lighter Birth Weights." National Sire Summary American Simmental Association (1976) A12. 11. Buttle, H.L. "The Maintenance of Pregnancy in Hypophysectomized Goats." Journal of Reproduction and Fertility, 52 (1978), 255-260. 12. Buttle, H.L., I.A. Forsyth, and G.S. Knaggs. "Plasma Prolactin Measured by Radioimmunoassay and Bioassay in Pregnant and Lactating Goats and the Occurrence of a Placental Lactogen." Journal of Endocrinology, 53 (1972), 483-491. 13. Challis, J.R., C.T. Jones, J.S. Robinson, and G.D. Thorburn. "Development of Fetal Pituitary Adrenal Function." Journal of Steroid Biochemistry, 8 (1977), 471-478. 14. Challis, J.R.G., J.Z. Kendall, J.S. Robinson, and G.D. Thorburn. "The Regulation of Corticosteroids During Late Pregnancy and Their Role in Parturition." Biology of Reproduction, 16 (1977), 57-69. 15. Challis, J.R.G. and J.L. Linzell. "The Concentration of Total Unconjugated Estrogens in the Plasma of Pregnant Goats." Journal of Reproduction and Fertility, 26 (1971), 401-404. 16. Comline, R.S., L.W. Hal, R.B. Lavelle, P.W. Nathaniels, and M. Silver. "Parturition in the Cow: Endocrine Changes in Animals with Chronically Implanted Catheters in the Fetal and Maternal Circulation." Journal of Endocrinology, 63 (1974), 451. 17. Corah, L.R., T.G. Dunn and C.C. Kaltenbach. Influence of Prepartum Nutrition on Reproductive Performance of Beef Females and the Performance of Their Progeny. J. Anim. Sci. (1975) 41:819. 18. Csapo, A.I. "The See-Saw Theory of Parturition." In Fetus and Birth, Ciba Foundation Symposium #47. Ed. by J.M. Knight and M. O'Connor. Elsevier, Amsterdam, 1977, pp. 159-210. 19. Currie, W.B., P.A. Kelley, H.G. Friesen, and G.D. Thorburn. "Caprine Placental Lactogen Levels of Prolactin-Like and Growth Hormone-Like Activities in the Circulation of Pregnant Goats Determined by Radio Receptor Assays." Journal of Endocrinology, 73 (1977), 215-226. 20. Currie, W.B., and G.D. Thorburn. "The Fetal Role in Timing the Initiation of Parturition in the Goat." In Fetus and Birth, Ciba Foundation Symposium #47. Ed. by J.M. Knight and M. O'Connor. Elsevier, Amsterdam, 1977, pp. 49-66. 21. Currie, W.B. and G.D. Thorburn. "Parturition in Goats: Studies on the Interactions Between the Fetus, Placenta, Prostaglandin F, and Progesterone Before Parturition of Term or at Parturition Induced Prematurely by Corticotrophin Infusion to the Fetus." Journal of Endocrinology, 73 (1977), 263-278. 22. Currie, W.B. and G.D. Thorburn. "Release of Prostaglandin F, Regression of Corpora Lutea, and Induction of Premature Parturition in Goats Treated with Estradiol 17-beta." Prostaglandins, 12 (1976), 1093-1103. 23. Currie, W.B., M.S.F. Wong, R.I. Cox, and G.D. Thorburn. "Spontaneous or Dexamathasone-Induced Parturition in the Sheep and Goat: Changes in Plasma Concentration of Maternal Prostaglandin F and Fetal Estrogen Sulphate." Mem. Soc. Endocrinology, 20 (1973), 95-118. 24. Debackere, M., G. Peeters, and N. Tuyttens. "Reflex Release of an Oxytocic Hormone

Copyright American Association of Bovine Practitioners; open access distribution.

by Stimulation of Genital Organ in the Male and Female Sheep Studies by a Cross Circulation Technique." Journal of Endocrinology, 22 (1961), 331-334. 25. DeLemos, R.A., D.W. Shermeta, J.H. Knelson, R. Kotas, and M.E. Avery. "Acceleration of Appearance of Pulmonary Surfactant in the Fetal Lambs by Administration of Corticosteroids." American Review of Respiratory Diseases, 102 (1970), 459-461. 26. Doornbos, D.E., R.A. Bellows and P.J. Burfening. "Effects of Obstetrical Assistance on Postpartum Reproduction in Beef Females. J. Anim. Sci (1981) (submitted for publication). 27. Falk, D.G., R.E. Christian, R.C. Bull and R.G. Sasser. "Prepartum Energy Effects on Cattle Reproduction." J. Anim. Sci. (1975) 41:267. 28. Ferguson, J.K.W. "A Study of the Motility of the Intact Uterus at Term." Surgery, Gynecology, and Obstetrics, 73 (1941), 359-366. 29. Fitzpatrick, R.J. "Dilation of the Uterine Cervix." In Fetus and Birth, Ciba Foundation Symposium #47. Ed. by J.M. Knight and M. O'Connor. Elsevier, Amsterdam, 1977, pp. 31-47. 30. Flint, A.P.F., and A.P. Ricketts. "Control of Placental Endocrine Funcation; Role of Enzyme Activation in the Onset of Labour." Journal of Steroid Biochemistry, 2 (1979), 493-500. 31. Flint, A.P.F., A.P. Ricketts, and V.A. Craig. "The Control of Placental Steroid Synthesis at Parturition in Domestic Animals." Animal Reproduction Science, 2, 1-3 (1979), 239-251. 32. Flint, A.P.E., E.J. Kingston, J.S. Robinson, and G.D. Thorburn. "The Initiation of Parturition in the Goat: Evidence for Control by Fetal Glucocorticoid Through Activation of Placental C-21 Steroid, 17-alpha Hydroxylase." Journal of Endocrinology, 78 (1978), 367-378. 33. Gillespie, A. "Prostaglandins and Human Labor." Mem. Soc. Endocrinology, 20(1973), 77. 34. Hammond, J. "Physiological Factors Affecting Birth Weight." Proc. Nutrit. Soc. (1944), 2:8. 35. Heap, R.B., A.K.A. Galil, F.A. Harrison, G. Jenkin, and J.S. Perry. "Progesterone and Oestrogen in Pregnancy and Parturition: Comparative Aspects and Hierarchial Control." In Fetus and Birth, Ciba Foundation Symposium #47. Ed. J.M. Knight and M. O'Conner. Elsevier, Amsterdam, 1977, pp. 127-150. 36. Hoffman, B., J. Schmidt, and E. Schallenberger. "Hormonal Mechanisms Involved in Control of Parturition." Current Topics in Veterinary Medicine and Animal Science, 4 (1979), 263-281. 37. Holm, L.W. "Prolonged Pregnancy." Advances in Veterinary Science, 11 (1967), 195. 38. Hunter, J.T., R.J. Fairchough, A.N. Peterson, and R.A.S. Welch. "Fetal and Maternal Hormonal Changes Preceding Normal Bovine Parturition." Acta Endocrinology, 84 (3), (1977), 653-662. 39. Joubert, D.M. and J. Hammond. "A Crossbreeding Experienced with Cattle, Special Reference to the Maternal Effect in South Devon-Dexter Crosses. Journal of Agri. Sci. (1958), 51:25-34. 40. Kennedy, P.C. "Interaction of Fetal Disease and the Onset of Labor in Cattle and Sheep." Federation Proceedings, 30 (1971), 110-113. 41. Liggins, G.C., R.J. Fairclough, S.A. Greives, J.Z. Kendall, and B.S. Knox. "The Mechanism of Initiation of Parturition in the Ewe." Recent Progress in Hormone Research, 29 (1973), 111-159. 42. Moody, E.L. and D.K. Han. "Effects of Induced Calving on Beef Production." J. Anim. Sci. (1975) 43:298 (abstract). 43. Patterson, D.J., R.H. Bellows, P.J. Burfening, R.E. Short, and J.B. Carr. "Incidence and Causes of Neonatal and Postnatal Mortality in Range Cattle." (Abstract). Journal of Animal Science, 49, suppl. 1 (1979), 325. 44. Putnam, M.R., L.E. Rice, R.P. Wetteman, K.S. Lusby and B.R. Pratt. "Planipart, (Clenbuterol) for the Postponement of Parturition and Alleviation of Dystocia in Cattle." Proc. Soc. Theriogenology (1982) (submitted for publication). 46. Rice, L.E. and J.N. Wiltbank. "Factors Affecting Dystocia in Beef Heifers." Journal of the American Veterinary Medical Association (1972) 161:1348. 47. Rice, L.E. "Dystocia in Cattle." (Master's Thesis, Colorado State University, 1969). 48. Roberts, J.S., J.A. McCracken, J.E. Gavagan, and M.S.Soloff. "Oxytocin-Stimulated Release of Prostaglandin F from Ovine Endometrium in Vitro: Correlation with Estrous Cycle and Oxytocin Reception Binding." Endocrinology, 99 (1976), 1107-1114. 49. Roberts, S.J. Veterinary Obstetrics and Genital Diseases (Theriogenology). 2nd Edition. Ann Arbor, Michigan: Edwards Brothers, Inc. 1971, 206-208. 50. Roberts, J.S., and L. Shore. "Effects of Progesterone and Estrogen on Blood Levels of Oxytocin During Vaginal Distension." Endocrinology, 84 (1969), 1076. 51. Ryan, K.J. "New Concepts in Hormonal Control of Parturition." Biology of Reproduction, 16 (1977) 88-94. 52. Schuijt, G. and L. Ball. "Delivery by Forced Extraction and Other Aspects of Bovine Obstetrics." In Current Therapy in Theriogenology. Ed. by Morrow, D.A. Philadelphia, Pennsylvania: W.B. Saunders Co. (1980), 247-257. 53. Sloss, V. and J.H. Dufty. Handbook of Bovine Obstetrics. Baltimore, Maryland: Williams & Wilkins, 6 (1980), p. 78. 54. Stabenfeldt, G.H., Bi. I. Osburn, and L.L. Ewing. "Peripheral Plasma Progesterone Levels in the Cow During Pregnancy and Parturition." American Journal of Physiology, 218 (1970) 571-575. 55. Thorburn, G.D. "Physiology and Control of Parturition: Reflections on the Past and Ideas for the Future." Animal Reproduction Science, 2, 1-2 (1979), 1-27. 56. Thorburn, G.D. and J.R.G. Challis. "Endocrine Control of Parturition." Physiological Reviews, 58, 4 (1979). 57. Thorburn, G.D., J.R.G. Challis, and W.B. Currie. "Control of Parturition in Domestic Animals." Biology of Reproduction, 16 (1977), 18-27. 58. Thorburn, G.D., D.H. Nicol, J.W. Bassett, D.A. Shutt, and R.I. Cox. "Parturition in the Goat and Sheep and Changes in Corticosteroids, Progesterone, Estrogens, and PGF." Journal of Reproduction and Fertility, suppl. 16 (1972), 61-84. 59. Wagner, W.C. "Parturition Induction in Cattle." In Current Therapy in Theriogenology. Ed. by Morrow, D.A. Philadelphia, Pennsylvania: W. B. Saunders, Co. (1980), 236-238. 60. Wiltbank, J.N. "Increasing Pounds of Calf Produced by Improving Reproductive Performance." 15th Livestock Research Roundup. N. Dakota Agri. Sta. (1964), p. 12. 61. Umo, I., R.J. Fitzpatrick, and W.R. Ward. "Parturition in the Goat: Plasma Concentrations of Prostaglandin F and Steroid Hormones and Uterine Activity During Late Pregnancy and Parturition." Journal of Endocrinology, 68 (1976), 383-389.