General Session I

Moderator - Erich Studer

The Normal Periparturient Cow

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Introduction

Life would be a lot easier for all of us if cows could produce milk without getting pregnant. Parturition is truly a mixed blessing of opportunity and frustration for producers and their veterinarians. In perfect compliance with Murphy's law, the periparturient period is a time when everything can, and often does, go wrong. This presentation will focus on managing reproduction in the abnormal postpartum cow from both a preventive and therapeutic perspective. We begin with a review of normal periparturient physiology.

Parturition

The cow and her calf spend the last month or so of gestation preparing for delivery. The endocrine events leading to parturition are also responsible for coordinating lactation, placental detachment, uterine involution and resumption of estrous cycles. It is an extremely important time for the cow in terms of production and reproduction in the next lactation.

During the last month of pregnancy, growth of the fetal adrenal cortex gives rise to a gradual increase in fetal corticosteroids. The increase in corticosteroids is responsible for the increased placental production of estrogens that occurs at this same time. Estrogens in turn are responsible for production of PGF_{2a}, oxytocin receptors[1] and gap junctions in the uterus during late gestation. The gap junctions are physical connections between the myometrial cells that enhance the propagation of uterine contractions. The effects of estrogens on the uterus are inhibited by the elevated levels of progesterone that exist at this time, thus preventing premature expulsion of the fetus.

During the last few days of gestation, something (fetal stress?) triggers the release of fetal ACTH causing a dramatic increase in fetal corticosteroids. This and the subsequent rise in estrogens result in the release of endometrial PGF_{2a}. The PGF_{2a} causes luteolysis, resulting in a rapid decline in progesterone and an increase in relaxin in the maternal blood. The estrogens, PGF_{2a} and relaxin cause the pelvic ligaments, cervix and birth canal to soften and expand in preparation for delivery. Signs of impending parturition become apparent and the cow enters the rather vaguely defined first stage of labor. At this time, uterine contractions are occurring at the rate of 4 to 5 every 10 minutes and the proportion of contractions propagating from the uterine tip to cervix is increasing.[2] The final cervical dilation occurs as the uterine contractions propel the chorioallantois through the cervix. As the fetus enters the vagina and the chorioallantois ruptures, a massive, reflexive release of oxytocin and PGF_{2a} occurs and second stage labor begins. Straining by the cow is coordinated with large, sustained uterine contractions. While the frequency of uterine contraction does not increase compared to first stage labor, the proportion of propagated contractions, the rate of propagation and the strength and duration of contractions are all increased.[2] Second stage labor usually lasts from one-half to one hour in the pluriparous cow. Heifers may take 3 to 4 hours to deliver their calf.

The third stage of labor encompasses expulsion of the fetal membranes and the initial stages of uterine involution, roughly the first few days postpartum. Complete involution of the uterus may take a month or more. Most cows expel the placenta within eight hours after delivery of the calf and membranes are considered retained sometime between 8 and 24 hours after delivery. The exact mechanism of placental separation is not known. The cellular and molecular mechanisms involved in placental separation are poorly understood. Placentomes become collagenized late in gestation while

leukocytes and binuclear giant cells invade the tissues. Infusion of collagenase into retained fetal membranes has been reported to promote expulsion of the retained placenta.[3] Expulsion of the detaching placenta and initial uterine involution are greatly assisted by continued strong uterine contractions. It is hypothesized that strong postpartum uterine contractions result in decreased blood flow to the endometrium and a resulting dilation of maternal crypts and a shrinking of chorionic villi. The contractions gradually decrease in strength and frequency over the first week postpartum but can be induced by treatment with 20 units of oxytocin during the first nine days.[4] Neither PGF_{2a} nor fenprostalene are as effective as oxytocin for induction of myometrial contractions in the early postpartum cow.[5] The benefits of hormonal treatment for retained placenta which are directed at increasing uterine tone are debatable as myometrial activity in cows with retained fetal membranes is already greater than in cows without retained membranes.[5]

The Postpartum Period

Complete involution of the postpartum uterus and resumption of estrous cycles are required for normal fertility. In the first week postpartum, the maternal portion of the placentome becomes necrotic and is expelled as part of the normal lochia. The caruncles are gone by about day 12 and the lochia ceases by days 14 to 16 postpartum. The uterus can be retracted for the first time between 10 and 20 days postpartum. By four weeks postpartum, the normal uterus has physically involuted almost completely. Histologic involution takes another 10 to 20 days to complete. In contrast to cows with a normal postpartum period, cows with retained placenta and metritis have altered neutrophil function,[6] longer endogenous prostaglandin release[7] and higher rates of mastitis.[8] Days open and services per conception are also greater in cows with an abnormal puerperium.

Bacterial contamination of the uterus at or shortly after delivery is the norm. More than 90% of cows have positive uterine cultures at two weeks postpartum but fewer than 10% are still positive as the cow enters the breeding period at 45 to 60 days postpartum. Persistent uterine infection can occur after retention of the fetal membranes, especially if an attempt is made to manually remove them.[9]

A healthy immune system plays a critical part in clearing bacteria from the postpartum uterus. It also has a role to play in policing the uterus after natural or artificial breeding. While antibiotics may assist in this process, there is substantial evidence that they are not a major factor in the recovery of most infected cows. An often overlooked aspect of the reproductive health equation is the integrity of the physical barriers to infection. Trauma to the perineum, vagina or cervix can lead to a chronic uterine contamination problem. Time and surgical reconstruction may be better alternatives to antibiotics for most of these cows.

After a brief period of refractoriness to GnRH, the anterior pituitary resumes its release of LH in response to hypothalamic or exogenous GnRH. Most dairy cows are capable of ovulating in response to exogenous GnRH by 14 days postpartum.[10] The first spontaneous postpartum ovulation usually occurs during the third week postpartum in most normal dairy cows. The timing of this ovulation is highly variable, however, being affected by cow health, uterine health, nutritional status and milk production, among others.[11] Usually, no behavioral estrus accompanies the first postpartum ovulation. Standing behavior occurs in most cows by the time of the third postpartum ovulation.

The corpus luteum that forms after the first postpartum ovulation is often small and produces low serum progesterone concentrations. It frequently maintains a shorter than normal diestrus period. If the first ovulation is delayed beyond the voluntary waiting period, this short interestrus interval can result in infertility. Cows that conceive at such an ovulation are likely to undergo luteolysis before maternal recognition of pregnancy can occur. Subsequent ovulations usually produce a normal diestrus. Ideally, cows would be ovulating for the third or greater time when first inseminated.

References

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