Therapy for Reproductive Problems in the Postpartum Cow

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Many problems beset the postpartum dairy cow and all of them may potentially decrease reproductive efficiency. This brief review will deal with retained fetal membranes (RFM) and metritis/endometritis and discuss various treatment approaches in light of recent research. It should be noted here that research often does not translate into obvious clinical recommendations. Two recent reviews dealing with RFM/metritis cite a combined total of 241 reports without reaching definitive conclusions about therapy (de Bois, 1982; Paisley et al., 1986). We will proceed from this rather uncertain foundation.

Retained Fetal Membranes

The ongoing controversy about cleaning cows will not be resolved here, unfortunately. Personally, I oppose manual removal of RFM and can justify it on aesthetic grounds. The problem is that the client frequently justifies attempts at manual removal on these same aesthetic grounds, just from a different vantage point (downwind). A look at the causes of RMF may shed some light on the present treatment dilemma. The incidence of RFM in normal herds ranges from 3 to 12% (Arthur, 1979). Among the proposed causes of RFM are: premature delivery, prolonged gestation, heat or other stresses, milk fever, selenium deficiency, vitamin A deficiency, infectious abortion, dystocia and twins. The mechanism of retention can vary with the cause from primary failure of the maturation and separation process to edema, hyperemia and necrosis of the placentomes to placentitis to uterine inertia (Grunert, 1984). Considering the variety of causes and mechanisms, it is not surprising that specific treatments have met with limited success. Interference with placental separation begins well before parturition and most therapy is designed to acutely squeeze or pull this tightly, chronically adhered placenta from the uterus. This approach is likely to be successful only in that 1 to 2% of RFM due to uncomplicated uterine inertia, such as occurs with milk fever.

Manual removal of RFM (cleaning cows) remains a controversial topic. Though widely practiced, there is little strong evidence to support its therapeutic value. Dryendahl (1977) reported a nonsignificant improvement in fertility in cows whose RFM were manually removed over those whose RFM could not be removed. It is important to note that fertility was decreased in both instances and that failure to remove membranes is not the same as allowing them to sit undisturbed. These data could just as easily be interpreted as contraindicating any intervention. What is needed, and often difficult to obtain, is a group of untreated RFM cows to serve as a control. Manual removal of RFM can cause additional uterine trauma and interfere with normal uterine defenses (e.g. phagocytosis) while introducing more contamination.

Alternative treatments for RFM involve uterotonic or antibacterial products. The limited value of prostaglandins, oxytocin, estrogen, ergonovine and calcium is not surprising in light of the low incidence of RFM due to uncomplicated uterine inertia. Additionally, Piper *et al.* and Martin *et al.* have shown that uterine muscular activity is not reduced in cows with RFM. Herschler and Lawrence (1984), however, reported a more rapid expulsion of RFM and a lower incidence of metritis in cows treated with fenprostalene (a PGF_2 analog).

Intrauterine antibiotics have been ineffective in clearing infections from the uterus of cows with RFM but they may decrease putrefaction and the subsequent odor. It is possible that interference with putrefaction can prevent placental separation and prolong RFM. Iodine and irritating antibiotic preparations also interfere with uterine phagocytosis. Ideally, antibiotics for intrauterine use would be nonirritating, broadspectrum, have limited systemic absorption and be effective in the presence of organic material. They should be particularly effective against the combination of Corynebacterium pyogenes and various anaerobes that apparently act synergistically to produce postpartum metritis and pyometra.

In the absence of a reliable therapeutic approach to the typical RFM cow I would suggest the following:

1. Emphasize prevention fostered by a better understanding of the causes of RFM. Dr. Jack Britt's presentation at this meeting should provide useful information along these lines.
2. When faced with a RFM cow, request that the client closely monitor appetite, production and temperature. Systemically ill cows with elevated temperatures can be treated with systemic antibiotics (e.g. penicillin). All RFM cows should be examined 2 to 4 weeks after parturition to monitor uterine involution and to detect and treat cases of metritis or pyometra as quickly as possible.

3. And finally, realize that a RFM cow is not a normal cow and it is unlikely that any present or forseeable therapy will convert her to normal status. Decreased reproductive efficiency can be expected in these cows. The old maxim "an ounce of prevention..." is all too true in this situation.

Metritis/Endometritis

Infection and inflammation of the postpartum uterus cover a spectrum from acute septic metritis with severe systemic signs to chronic endometritis characterized only by infertility. Obviously, approaches to treatment will depend on the type of metritis present. Very frequently, the more severe disease results from RFM and preventive measures should be directed at this as well as emphasizing sanitation at parturition.

Treatment is intended to evacuate uterine contents, bring the infection under control while promoting uterine defenses and providing support for cows with severe systemic disorders (i.e. fluids, antiinflammatory agents). Prostaglandin F_2 and its analogs, (PGF), oxytocin and estrogens have all been recommended for postpartum uterine evacuation. Oxytocin is the drug of choice in the first week postpartum while PGF is indicated for cows with a mature corpus luteum on the ovary. Estrogens have been reported to cause more serious metritis and salpingitis (Gustafsson and Ott, 1981) while others report that after the first week postpartum they promote uterine phagocytosis (Vandeplassche and Bouters, 1983). The efficacy of any single hormonal treatment (even repeated daily) to produce myometrial stimulation that results in an overall therapeutic effect has never been demonstrated.

Therapy to control infection has traditionally involved intrauterine infusion of antibacterial products. A serious drawback of this type of therapy is the need to withhold milk from treated cows. Other limitations include the anaerobic postpartum environment which inhibits the activity of the aminoglycosides, presence of resistant organisms, high concentration of organic debris and decreased absorption limiting treatment of the entire uterus. Perhaps a more serious problem is the potential for intrauterine antibiotic therapy to interfere with phagocytosis (Vandeplassche, 1984). Absorption of drugs from the postpartum uterus is low (Gustafsson and Ott, 1981). This can prevent effective levels of antibiotic from reaching the deeper uterine tissue. Additionally, no antibiotics are approved for intrauterine use in the cow in this country.

Olson et al. (1984) have made the following recommendations for treating uterine infections. In the immediate postpartum period (first 1 to 2 weeks), cows are treated locally with tetracyclines and, if needed, systemically with penicillin. Treatments are repeated daily as required. Uterine infections after the first 1 to 2 weeks postpartum are generally local in nature and intrauterine infusions of tetracycline are recommended. The use of GnRH at this time to promote ovulation and uterine cleansing during estrus is controversial. One researcher has found an increased incidence of pyometra in cows routinely treated with GnRH on day 15 postpartum (Etherington, 1983). Finally, Olson et al. recommend repeated intrauterine penicillin treatment for infections persisting into the postovulatory period.

The use of PGF alone has been promoted for treatment of uterine infections at all postpartum stages in cows with or without a mature corpus luteum (see Paisley et al. 1986). They propose that in addition to luteolysis, PGF may stimulate myometrial contractions to aid in uterine evacuation and may also promote phagocytosis by uterine leucocytes. They cite the additional benefit of not having to discard milk when PGF is used instead of intrauterine antibiotics.

In summary, therapy for RFM and uterine infections is still quite subjective. Very often, clinical trials to evaluate therapy are inadequately designed. Until a proven therapeutic approach is elucidated, it is important that we choose treatment options that are not harmful in themselves, are economically justifiable and do not risk contamination of the human food supply. As the economic realities of the dairy industry evolve, it is likely that farmers will develop a stronger interest in preventing these problems in order to maximize profitability rather than treating them to minimize losses.

References

1. Arthur, G.H. Retention of the afterbirth in cattle: A review and commentary. Vet. Ann. 19:26-36 (1979). 2. de Bois, C.H.W. Some aspects of the therapy and prophylaxis of retained placenta and puerperal endometritis in the cow. *In:* Factors affecting fertility in the postpartum cow. Karg, H. and Schallenburger, (eds.) Martinus Nijhoff Publishers, The Hague, 1982, pp. 479-509. 3. Dryendahl, I., Mattson, J., and Pehrson, B. Retained placenta in cattle—incidence, clinical data and effects on fertility. Zbl. Vet. Med. A, 24:529-541 (1977). 4. Etherington, W.G. The postpartum cow: Physiology, uterine involution and hormonal therapy. Spec. Ed. Comp. Cont. Ed. Pract. Vet. - Proc. Symp. Reprod. Mgmt. in Food Animals, St. Paul, 1983, pp. 16-21. 5. Grunert, E. Placental separation/retention in the bovine. Proc. Xth Intl. Cong. Anim. Reprod. and AI, Urbana-Champaign, 1984, IV:XI. pp. 17-24. 6. Gustafsson, B.K. and Ott, R.S. Current trends in the treatment of genital infections in large animals. Comp. Cont. Ed. Pract. Vet. 3:147-151 (1981). 7. Herschler, R.C. and Lawrence, J.R. A prostaglandin analogue for therapy of retained placenta. VMSAC. 79:79-81 (1984). 8. Martin, L.R., Williams, W.F., Russek, E. and Gross, T.S. Postpartum uterine motility measurements in dairy cows retaining fetal membranes. Therio. 15:513-524 (1981). 9. Olson, J.D., Ball, L. and Mortimer, R.G. Therapy of postpartum uterine infections. Proc. 17th Ann. Conv. AABP, Ames, IA, 1984, pp. 85-88. 10. Paisley, L.G., Mickelsen, W.D. and Anderson, P.B. Mechanisms and therapy for retained fetal membranes and uterine infections of cows: A review. Therio. 25:353-381 (1986). 11. Piper, E.L., Combs, K.J. and Peterson, H.P. Influence of corticoid induced parturition on postpartum uterine activity. VMSAC 73:1447-1449 (1978).

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