Dairy Session

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Embryonic Death & Early Abortion in Cattle—A Review

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My assignment today is to discuss embryonic death and early abortions in cattle. Dr. Miller will cover various aspects of abortions which occur during the remaining phases of the gestation period.

Recently I visited with a veterinarian who expressed the opinion embryonic death or early abortions in cattle are of little consequence and therefore is inclined to ignore their occurrence in his practice.

Based on experience, I find this approach quite unrealistic. It is my opinion early fetal loss should be given careful consideration in the conduct of all herd health programs.

To what extent do losses from early deaths occur? Reports range all the way from 2 per cent to 35 per cent. When a low level loss occurs in a large commercial herd, the situation may not always seem significant, however, if embryonic or early fetal death occurs in one or more valuable individuals, or the loss becomes a herd problem, the situation assumes considerable economic importance.

In my presentation I will attempt to include some principal causes, describe methods of diagnosis and discuss briefly ways of prevention and treatment.

You are aware there are a variety of causes of early fetal death. Some early deaths are associated with disease or infection; others are due to a variety of other factors.

The Venereal Diseases

Trichomoniasis This infection is not as frequently encountered today as it was 25 -30 years ago. However, the disease continues to exist and a brief review appears in order. The disease was quite widespread in both beef and dairy herds during my early years of practice. I would see at least a dozen herds a year and on many occasions an inkling of the cause was obtained from a visit with the owner even before observing or palpating the herd.

If all or some breedings in a herd are by means of natural service, pus discharges at the vulva are observed in some cows, irregular or prolonged intervals between heats are noted, and/or early abortions occur, a veneral disease, particulary "trick", should be suspected.

To verify this suspicion, palpation of the herd serves as a major factor in diagnosis. In view of this, I feel I can best describe the diagnostic methods used if I briefly recite some of my experiences.

The first herd I will describe was a beef herd with a history of breeding problems. Trichomoniasis was suspected. Early in the individual examinations I encountered a female that had been bred approximately three weeks previously. She was observed in heat and it was noted the secretion was slightly cloudy. I proceeded to gently milk the uterine horns and collect a few drops of the secretion at the vulva in a test tube. The "trick" protozoans were readily observed under both the low and high powers of the microscope.

In another beef herd where "trick" (trichomoniasis) was suspected, a case of pyometra following breeding was found in the seventh animal examined. Some of the pus was aspirated by means of an infusion tube and examined under the microscope. The sample proved positive. Incidently, once the pus produced by "trick" organisms is recognized, it cannot be easily mistaken for that produced by other organisms. It is light gray in color and has the consistency of slightly thickened milk.

In a dairy herd (we will call it **Herd A**) the history was as follows:

The owner, because he was experiencing serious breeding problems, sold his herd sire which was being used for natural service, and proceeded to have his cows artificially inseminated. After a few months, he experienced little improvement in conceptions. I was consulted at this point and the herd was found to be heavily infected with "trick" organisms. The herd had become infected in the following manner:

A heifer in the herd while in heat, jumped the fence which separated the herd from the neighbors'. She was serviced by the neighbor's bull before she was reunited with her herd mates. The heifer returned to heat 3 weeks later and at this

time was naturally bred to the herd "A" sire. I'm sure you have a suspicion what happened. The neigbor's bull was "trick" infected. He infected the heifer who in turn infected the Farm "A" herd sire who then infected the cows he subsequently bred, which by the way, included the majority of the females in the herd. This herd had a high percentage of pyometra cases caused by the "trick" organisms. Incidentally, pyometra following breeding is a common symptom of trichomoniasis.

Clean Herds Can Become Infected

You may have a client with a clean herd, a herd free of trichomoniasis, but that doesn't mean it cannot become infected while on a breeding health program. I'd like to refer to an outbreak that fits into this kind of situation.

This beef breeder had a healthy herd until he purchased a cow at a consignment sale. At about the same time he bought a high priced herd sire from a breeder in another state. The female was supposed to be with calf; but wasn't and returned to heat soon after she arrived at the farm. At this time she was naturally serviced by the new bull. The cow aborted approximately four months later. The fetus was submitted to a laboratory and found infected to the "trick" organisms. Fortunately only a limited number of females were subsequently bred to the bull, thus avoiding a herd infection.

Symptoms

Findings in The Female. In the female the "trick" organisms may produce metritis, pyometra, embryonic death or abortion. Infection tends to remain in the uterus for a period of about 2 to 6 months. Immunity tends to be built up, but the degree of immunity varies with the individual.

As was pointed out, the organism can be picked up in the secretion or from the pus of an infected uterus. It also can be found in the stomach of an aborted fetus or in the placental fluid a few days after abortion.

In The Bull. For diagnosis of the disease in the bull, one method is to collect samples of smegma either by inserting a long pipette into the bull's sheath or by flushing the sheath with saline solution. Samples may be checked under the microscope in the field or in the laboratory. To obtain an accurate diagnosis in a suspect individual, it is sometimes necessary to repeat the samplings or rely on other methods of diagnosis.

Another diagnostic method is to breed the suspect bull to a virgin heifer; start checking the uterine secretion in the female beginning about the 18th or 19th day following service.

Prevention and Treatment

Some management suggestions are:

Discontinue natural service and if possible, isolate or remove infected individuals from the herd.

Sexual rest for infected females is advisable and artificial inseminations should replace natural services.

Treatment-Females. Metritis or pyometra cases due to "trick" organisms may be treated similar to infections caused by other organisms.

Treatment - Bulls. Infected bulls should be slaughtered or given therapy. It is practical to treat only vauable herd sires.

Local Treatment. Local treatment has been successfully accomplished through the use of acriflavine, bova-flavine and/or tryptoflavine (1). The bull is given a pudental nerve block (as described by Dr. Lester Larson) or is tranquilized to expose the penis. The extended penis is thoroughly washed with warm water and a mild, non-irritating soap. Rinse membranes and dry. Then thoroughly massage into the mucus membranes bova-flavine ointment. Also, a small quantity of trypoflavine is injected into the urethra. Let the solution remain for about 5 minutes.

After treatment at least 5 negative samplings of the sheath should be made before the bull can be safely pronounced free of infection. The bull must be retreated if organisms are not completely eliminated.

Systemic Treatment. In this form of treatment, Dimetredogale (2) is given orally in bolus form or as a drench at the rate of 50 mg. per pound of body weight daily for 5 days. The drug may also be mixed in the feed. However, this latter treatment has been found not to be very reliable.

The drug may also be given intravenously in dosages varying from 10 mg/kg body weight daily for 5 days, or 50 to 100 mg/kg of body weight in a single injection.

VIBRIOSIS (now called CAMPYLOBACTER)

This veneral disease is caused by *Vibrio Fetus venerealis.* As in the case of trichomoniasis, this disease is transmitted primarily at breeding time and produces a temporary infertility. A high percentage of infected females may return to heat at prolonged intervals, indicating embryonic death. Early abortions also can occur. Pyometra development in vibrio infections either does not take place or occurs very rarely.

Confirmation of a tentative diagnosis is through identification of the organism in the laboratory from a freshly aborted fetus or from a sample of secretion collected at the cervix or obtained by means of uterine biopsy.

Samples secured from several suspect females in the herd are usually adequate for herd diagnostic purposes. Samples should be forwarded to the laboratory and cultured within 6 hours.

A third method of obtaining a sample for culturing is to naturally breed a virgin heifer to a suspect male and then take a cervical swab about 3 weeks later.

Unfortunately only a few laboratories are equipped to conduct tests for vibrio infection.

Prevention and Treatment. The vibrio organism is destroyed when infected semen is properly treated with antibiotics. Females often recover from infection in a few weeks or months without treatment. A relatively small number of females will, however, carry the infection through a normal pregnancy and continue to harbor the organism in the genital tract following calving.

The practice of annual vaccinations before breeding has been found effective in controlling the disease.

Other Causes

So much for venereal infections. Now let's move on to a

brief review of the various other causes of embryonic or early fetal death.

Early deaths are believed to be symptoms of such diseases as the upper respiratory viral diseases, leptospirosis, fungal infections, mucoplasma or uroplasma infections [3], etc.

The exact roles of some of these diseases in relation to early fetal death are not yet fully understood, hence the need for further study.

There is reliable evidence that some nutritional deficiencies or disorders contribute to embryonic or early fetal death. An example of a deficiency is vitamin A or its percursor carotene. Recent studies in Germany[4], in England, and limited field trials in this country [5], suggest that Beta-carotene may play a more significant role in fertility than vitamin A. Here also, more studies and observations are needed to establish specific conclusions.

Other suspected causes of embryonic death are severe stress, unsuitable environments, scar tissue of the uterine tract, etc.

Failure of the corpus luteum to produce adequate amounts of progesterone to maintain pregnancy is considered to be an important factor.

A healthy, normal cycling cow will occasionally produce an abnormal ovum which may or may not be capable of fertilization. If fertilization takes place, death of the embryo or fetus often occurs with subsequent absorption or abortion.

Limited research also suggests that a defective sperm is occasionally capable of fertilizing a normal egg. A normal zygote may be produced but such a union will more likely terminate in early death.

Abnormal embryonic development tends to result from polyspermency [6] (when more than one sperm cell enters the ovum).

There is also good evidence that too late breeding during the estrus cycle contributes to embryonic death. Studies reveal [7] that only about 30 per cent of embryos were found normal after 35 days when service was postponed to 12 hours after ovulation.

Limited observations in our practice have shown that a higher percentage than normal of embryonic death occurs when the corpus luteum is situated on the ovary opposite the pregnant horn.

I am sure I have overlooked mentioning other known or unknown causes of embryonic death and no doubt there are contributing factors that have not yet been suspected.

Some of the causes mentioned above are individualistic in nature and may not always be considered significant. On the other hand, those early deaths that result from disease or deficiencies may develop into herd problems that definitely need our attention.

Clinical Diagnosis

I would now like to digress a little and briefly discuss clinical diagnoses of embryonic death. This isn't always an easy assignment, but with experience, a positive diagnosis can be made about as easy as determining pregnancy.

Let's assume we are in position to palpate a cow that is pregnant 30-40 days. Normally the horn in which the embryo is located will be found slightly larger and contain somewhat more fluid than the non-pregnant horn. All structures, including the membranes, fluid and the amnionic vesicle can be palpated. The normal vesicle is round (pea like) has a firm wall and is larger at 40 days than at 30 days. The vesicle is usually located in the middle third of the uterine horn. Occasionally it is found near the tip or in or near the bifurcation (body of uterus).

If membranes and fluid are palpable but the vesicle is absent, you have a right to assume absorption or destruction of the embryo has taken place. Before such a determination is made, however, it is imperative the entire length of both horns first be carefully and gently palpated.

The ovary with the corpus luteum is invariably located on the side of the pregnant horn. However, it should be recognized that occasionally (about one out of 4000 pregnancies)[8] the corpus luteum will be located on the ovary opposite the pregnant horn.

When numerous palpations are made, expect occasionally to encounter a vesicle with a very thin wall which collapses with the slightest touch. This finding suggests embryonic death has occurred or is in the process of taking place.

When uterine palpations reveal fluids only — membranes and vesicle absent — you very likely are dealing with a case of hydrometra, mucometra or pyometra, depending on the consistency of the contents. With experience, these conditions can be differentiated without difficulty.

Now let's assume we are palpating a cow with a normal pregnancy of about 50 to 90 days. All structures can be readily palpated. The uterine horns will, of course, be correspondingly larger, contain more fluid and membranes than when palpations are made earlier. The vesicle also will be larger, oblong in shape and its wall will appear softer and more resilient.

If fetal death has occurred or is occurring, structural changes will be noted. Either the vesicle and embryo or young fetus will have disappeared partially or entirely or destroyed tissue will be present, which usually can be readily palpated. In a case of long standing, the dead fetus can be palpated as irregularly shaped, dessicated tissue.

In the process of fetal absorption, the fluid content of the uterus becomes diminished in volume and the uterine horns will have undergone contraction. Contraction of the cornua apparently occurs at a more rapid rate than shrinkage of the fetal membranes. As a consequence, the membranes become wrinkled and on palpation present a "squishy feel". This feel is very diagnostic of the condition in question.

As may be suspected, the various stages of defective development as well as the stage of gestation will present various deviations which, with experience, can usually be quite accurately determined.

Prevention and Control

Prevention and control of known or diagnosed causes of early death can usually be prevented or treated accordingly.

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For example, if disease, nutrition or stress are the responsible factors, it becomes a matter of accurate diagnosis, proper treatement, and changing or improving the feeding and management programs.

It's another story, however, when it is difficult or impossible to come up with a reasonable cause of early death in an apparently healthy individual. In this regard I would like to briefly discuss the controversial subject of preventing some embryonic or early fetal deaths through the use of repository type progesterone.

Back about 25 years ago I came across a young healthy cow that had twice lost her fetus at approximately 3 months. The cow was found pregnant at the following breeding and the owner remarked, "Isn't there something we can do so this cow can carry her calf to term?" I didn't have an immediate answer, but I did tell him about some research work carried on at the University of Wisconsin on embryonic death. Among other findings the researchers noted if the corpus luteum was enucleated in early pregnancy and 50 mg. of progesterone was administered daily, pregnancy could be maintained.

About this time Pitman-Moore Company began marketing Repositol Progesterone. To make a long story short, we treated the cow in question with this product and she carried her fetus to term. The next time she was found pregnant, the owner asked "shall we treat her again?" I indicated I didn't know. We went about our work in the herd and unintentionally overlooked the cow. In other words we overlooked treating her. She aborted at about the same stage of pregnancy, and naturally the owner was not happy.

Since that time we have treated hundreds of cows with repository type progesterone apparently with very satisfactory results.

In our program of treatment, we have been selecting cases in which we were unable to arrive at a reasonable cause. In other words, we employ progesterone treatments when early deaths cannot be traced to disease, deficiency or other suspect factors.

Briefly our program of treatment is as follows:

If a cow shows early evidence of embryonic death, we start the repository progesterone injections on the 4th or 5th day after service. The dose is 10cc (500 mg.) intramuscularly. The injections are repeated at intervals of 7 to 10 days. If the time of death of the embryo or fetus is suspected to have occurred at a later date, 50, 60 or 90 days following conception, we postpone the first injection to about the 27th or 28th day after service, provided a normal subsequent heat had not taken place. We continue the prevention treatments for about 50 to 60 days beyond the anticipated death period.

In the absence of adequate research, we use this treatment because it appears to be effective in a very high percentage of cases.

An alternative method of treatment in cases of this kind would be to employ the cow as a donor in embryonic transfer, particularly if the individual warrants the effort and expense involved.

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Dr. R. B. Miller's paper will appear in the 1982 Bovine Practitioner