Practice Tips and Related Topics

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Clinical Management of Early Fetal Death:

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I think most of you know, especially those who are in reporductive work, that embryonic death is a big subject and before I get into my practice tips I would like to make a few brief remarks. The first one is that embryonic death is a significant economic factor. Estimates from losses run all the way from 2 to 35%. My second remark is that there are a variety of causes. Twenty-five or thirty years ago in my practice, I would come in contact with probably a dozen or fifteen herds that were infected with trichomoniasis or vibriosis. It was my feeling at that time that embryonic death was primarily due to those two diseases. In the last ten years I can't recall coming in contact with one herd that has these diseases and I think that probably that is the same experience that you have had also. But, we do have embryonic death and I would just like to list a few of those factors or predisposing causes of embryonic death. First one would be diseases like IBR or BVD, possibly leptospirosis, and diseases that affect reproduction could be responsible for embryonic death, although more recent research work would indicate that they are not too important. Then we have stress factors. Hot weather, for example. I'm sure most people who work in this field, in hot weather, realize that conception is down, missing heat periods, but the embryonic death situation is up. Then we have nutrition. I doubt that there is any question that improper nutrition or inadequate nutrition might affect embryonic death. We used to think that vitamin A was important, but more recent work indicated that beta carotene now more or less supercedes vitamin A, in other words there is beta carotene in the corpus luteum whereas there is no vitamin A. Then we have abnormal development of cells, that is the embryonic cells, maybe half of the fertilized egg will develop normally and half will be abnormal. But when you have a situation like that you are apt to have embryonic death. Inheritance is a factor. Then there is the possibility of deficiency of progesterone being produced by the corpus luteum. Too late breeding will cause embryonic death. In our practice we have noted that when the corpus luteum is located on one ovary and the pregnancy is in the opposite horn, we find that the percentage of embryonic death is a little bit higher in those cases. There are other causes and I probably have skipped some and I'm sure there are some that we have probably still not run across. Can we do anything about it? Well, maybe we have part of an answer and maybe we haven't, anyway, I'd like to go back again for about 25 years.

When I came across a young healthy cow in a healthy herd that had lost two fetuses at approximately 3 months of gestation and we found the cow pregnant following this period, the owner said "Isn't there something that can be done to save this cow or so she can have a normal calf?" I really didn't know of any and then I did recall that the University of Wisconsin was doing some research work and among other things they found that if they would enucleate the corpus luteum in early pregnancy and give 50mg of progesterone daily, they could maintain pregnancy. Well about this same time also, Pitman-Moore came out with their reposital progesterone and to make a long story short, we treated this cow with repositol progesterone and she carried to term. Well, that doesn't mean too much, but anyway, the next time we found this cow pregnant the owner said "Shall we treat her again?" I said I did not know of any research work on it. Anyway, in our practice we go through a herd, that is we do the palpating and so on and then we go back and treat those that need it. We unintentionally forgot this cow and she aborted at the same time, so naturally the owner was quite perturbed. So, since that time we try to carry on a program of trying to prevent some of these embryonic deaths, whether we succeeded or not I'm going to let you decide. Our program was this, if we found that a cow had early embryonic death we would start the progesterone treatment on the fourth day after service. That is we gave 10cc (or 500mg) intramuscularly and then we repeated it every 10 days. Now if the embryonic death occurred later, let's say after 50, 60 or 90 days, then we would wait until the cow passed one heat period and start the progesterone injection on the 27th or 28th day and we tried to estimate the time that the embryo died and from that date we would go beyond 60 or 70 days and then we would stop.

Now, in treating these cows for embryonic death with progesterone, we would try to select our cases and by

selecting cases I mean we would eliminate any cow that might be diseased or might have a diseased reproductive tract, or we felt that the cow was not getting enough to eat with the proper food. In other words we treated those in which we really didn't know the cause. We have treated hundreds of cows during the last thirty years and we felt that we were getting benefit from using this type of treatment. Now, last year one of the speakers at the meeting said that there is no real good excuse for using a hormone to treat embryonic death. Well, he may be right and I'm not saying he is wrong. I'm not saying I'm wrong and when I say I, I mean there are quite a few veterinarians that I'm sure in this group who have used this treatment and found it successful. Some of you may not have found it successful, but I just wanted to bring this out because I think embryonic death is a very important situation with these valuable cattle and when you can save one, it is worth while.

Resistant Salmonellosis in Southern Alberta Feedlots

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Introduction

Reports on feedlot salmonellosis in North America over the past twenty years are scanty. During the period 1960-66, calf losses were reported in Arizona feedlots, stressors and inter-current diseases were considered to be important predisposing causes (9). Another report cites morbidity up to 30% in yearlings in a Colorado feedlot in 1961 (4). In the same feedlot in 1976, a mortality survey revealed 1.8% of total losses to be due to salmonellosis (5).

In Alberta, feedlot salmonellosis was not a problem until 1971 when a small number of deaths occurred in one feedlot. Dysentery was a prominent sign and at necropsy hemorrhagic enteritis was found. *Salmonella typhimurium,* resistant to chloramphenicol, tetracyclines, sulfonamides and streptomycin was consistently isolated. Disease continued at a low level for two years. In 1973 the use of an autogenous bacterin was reported to give good results and has been used since at this feedlot.

Losses in other feedlots continued to be very low and sporadic but in the spring of 1979, increased losses were experienced in two southern Alberta feedlots. A *Salmonella typhimurium* with a similar resistance pattern to the one mentioned previously was isolated. Antibiotic treatments which had been used with some success became relatively ineffective, losses mounted and the operators voiced much concern. An investigation was conducted to attempt to determine the causes of the outbreak and to suggest preventive measures.

History

Each of the two custom feedlots contained approximately 7,000 yearling cattle which originated either in the northern part of the province or in neighbouring Saskatchewan. Trucking distances from the various sales yards to the feedlots ranged from 320-800 km. Within two weeks of entry, animals were routinely branded and were vaccinated against infectious bovine rhinotracheitis, para-influenza-3 and clostridial diseases. The feed contained, for much of the feeding period, sufficient tetracycline to provide each animal with 75 mg. per day. The two feedlots were not particularly clean because of heavy rain the previous year. This had produced a build-up of mud or slurry in many of the pens. Furthermore, snow and rain during the spring of 1979 made the situation worse.

Death losses were normally between 1% and 2% but with the advent of salmonellosis, losses increased to between 2% and 2½%. In addition there were increases in cattle prices, in interest rates and in feed costs. Veterinary services and drugs became expensive and many treated animals developed chronic sicknesses. Finally, there was concern over the possibility of investigation by public health authorities. Losses continued at a lower level throughout the summer and fall of 1979 but rose to a second peak during the winter. Losses then gradually decreased in the spring of 1980 and finally stopped during midsummer.

Signs, Pathology and Microbiology

The common clinical signs were diarrhea and/or respiratory distress. Dysentery was less frequently noted. Necropsy studies conducted on many of the cattle with diarrhea or dysentery showed a severe fibrino-necrotic enteritis involving both large and small intestines. In these cases there was frequently a friable, yellow-grey, rope-like cast within the intestinal lumen. Necrosis and ulcers, sometimes associated with hemorrhage, were detected in the mucosa. Necrotizing or ulcerative abomasitis was often found along with the enteritis. In a small number of cases, ulcerations occurred in the omasal leaves adjacent to the omasal-abomasal junction.

Respiratory changes accompanied approximately half of the cases showing gastro-enteritis. A severe fibrinous pneumonia was most common while other findings included diphtheritic tracheitis (IBR) and necrotic laryngitis. Some cattle suffered a profuse thin yellow diarrhea; these did not have gastro-intestinal lesions but did have a severe fibrinous pneumonia. The feedlots were requested to submit to the laboratory dead animals or alternatively, intestine including ileo-cecal valve, liver and gallbladder, mesenteric nodes and lung from necropsied animals. These tissues were cultured and salmonellae were isolated from 46% of ileo-cecal valve tissues, from 42% of gallbladders and from 41% of mesenteric nodes. Only 5% of lungs yielded salmonellae.