Sudden Death Syndrome of Feeder Cattle: A Proposal for a New Approach

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Abstract

Sudden death syndrome (SDS) of feeder cattle is the occurrence of unexpected deaths of feeder cattle in their home feeding pen.[1] Cattle fitting this classification show an unclear cause of death, even after postmortem examination. Occurrence is sporadic and it is currently impossible to predict when or which cattle will be affected. The cattle are not observed moribund antemortem; they are rarely observed in the process of dying and they never leave evidence of antemortem discomfort or struggle.[2,3]

Cases of SDS are defined by the following criteria: animals are found dead with no recent sign of illness noted; necropsy findings indicate the animals did not die from any of the commonly recognized causes of death in feedyard cattle; deaths seem to occur in cattle which have been in the feedyard more than 45 days; post-mortem decomposition appears to proceed at an accelerated rate and it is more pronounced in the small intestine, liver and kidney than in surrounding tissues; abnormalities seen on post-mortem examination of the carcasses are generally referable to antemortem metabolic abnormalities, and these abnormalities are subtle and often in the process of being obliterated by the accelerated decomposition.[3,4] Incidence seems to vary between yards and with season or weather.[3,5]

Review

There are four investigations and three proceedings papers which directly address SDS. These efforts were undertaken either to document the efficacy of putative solutions[2,5,6] or to develop an experimental model in a controlled setting.[1,7,8]

Only one SDS-related article is published in the refereed literature.[2] This work by Jensen and Pierson, *et al.*, assessed the prevalence of the syndrome and characterized its associated pathology in feedlot cattle in northern Colorado.[9] The study surveyed 407,000 cattle for illness and death over a 14-month period. The cattle were in four northern Colorado feedlots from November of 1973 to December of 1974. The authors concluded the term "Sudden Death Syndrome" is a misnomer and "in some instances, [it is] a mask for neglect..." While this study was part of a landmark investigation of feedyard pathology and descriptive epidemiology, there are problems in the analytic process the authors used to generate their conclusions regarding SDS.

These results seem skewed by inclusion bias. Of the 407,000 cattle place in that period, 4,260 died. 1,358 (32%) of those died suddenly (i.e. found dead in the home feeding pen without recognition of premonitory illness). The researchers made a concerted effort to necropsy every dead animal during the study period, but they excluded autolytic carcasses and carcasses from animals dying on the weekend.[2] They necropsied 47% of all mortalities, (1,988/4,260), but they were 1.8 times more likely to necropsy cattle that died in the sick pen ($p < 0.0001$) than cattle that died in the home pen--the post mortem examination rates were 54% (1571/2902) and 31% (417/1358) respectively. In the analysis of the results, 43% (179/417) of the post mortems conducted on pen deads were excluded. The authors' conclusion that the major cause of pen deaths was pneumonia is not substantiated by their data. Only 27% (113/417) of the deaths were attributable to pneumonia, whereas 30% were attributable to other causes. Because consolidative, bacterial pneumonia is recognizable even in carcasses at an advanced stage of decomposition, it is fair to conclude that few if any of the remaining 43% were due to pneumonia.

The three non-refereed articles and the three proceedings papers about SDS are inconclusive. Of them, the three Cecil Reedy Workshops on SDS sponsored by the Academy of Veterinary Consultants in the mid-1970s[1,7,8] provide the most insight into SDS. These proceedings papers are comprised mainly of the conversations of feedlot practitioners, allied industry technical representatives and acedemicians. As such, these proceedings give insight into the thought and effort expended on SDS during the early 1970s.

Turner's article in 1971[5] included a field description of the problem and described vaccination as a means of control. However, the author's conclusions were not based on controlled field trials. In fact, the declining incidence of SDS attributed to the four-way clostridial booster program actually started before the institution of the program. Thus, it is likely that the diminished SDS incidence was due at least in part to causes other than the vaccination program.[3]

Coleman tested the efficacy of a four-way clostridial bactrin in protecting yearling steers from intramuscular challenge of injected *Clostridium sordelli* suspensions. Following the challenge, all of the non-vaccinates died with some lesions similar to those noted in SDS while the vaccinates survived with observable damage limited to local stiffness, swelling and tenderness. Interpretation of these results is complicated by the fact that the two groups were challenged with dissimilar *C. sordelli* suspensions. The suspension used on the controls was of known lethality, while that used on the vaccinates was concentrated ten-fold over that of the controls. Making a live culture ten times more concentrated does not make it ten times more virulent, in fact it may even destroy the virulence. From the information given in the article, it is impossible to know if the vaccinated steers received a lethal challenge and the sparing of the vaccinates' cannot be confidently attributed to the vaccine.

Don Williams (unpublished) described SDS and provided basic epidemiologic perspectives on its incidence over the period from January 1971 to December 1973 in three feedyards on the southern High Plains. In this paper, he proposed that SDS is the result of a small percentage of feedlot calves becoming hypersensitive to endotoxins that are released by gram negative bacteria dying in the rumens of grain-fed cattle; subsequent endotoxin exposure resulted in rapid death due to anaphylaxis. This theory explains well the sudden and sporadic nature of SDS, and some support of it was established by Tom Huber using an endotoxin assay derived from the horseshoe crab.[8] The theory weakens, however, due to Eyre's explanation in 1977 that the circulatory pattern of the bovine respiratory system precludes anaphylaxis from resulting in the cervical fascial hemorrhage and edema common to SDS carcasses.[7]

Experiences in Central Nebraska

Two feedyards in central Nebraska recently began recognizing SDS as an unacceptably high cause of loss. As a result, they agreed to provide us with retrospective data. The records are the most complete for 1992 and during that year 1% (611) of the cattle placed at risk died. Of those cattle that died in the pen, feedyard personnel classified 62% (273/437) as SDS based on the location and timing of the death and the outward appearance of the carcass. Consulting veterinarians necropsied 32% (139/437) of those pen dead cattle and found 56% (78/139) of the pen deaths attributable to SDS; 22% (30/139) attributable to respiratory disease, 2% (3/139) attributable to buller injuries and 4% (5/139) were too autolysed to make a diagnosis. The kappa value for agreement between the feedyard and necropsy classifications of SDS was 0.72 for the feedyard that recorded the pre-necropsy assessments.

While this data indicates the cowboys are probably overly eager to call a carcass an SDS case, some credence can be placed in the caretaker's classification of 273 of the 437 pen deaths as SDS cases. Assuming that the necropsied cattle were representative of the rest of the pen deads results in the estimate that 245 cattle died of SDS in these two feedyards in 1992. That is 0.4% of the cattle placed at risk and comprises 40% of the total death loss.

The numbers generated from this retrospective evaluation are similar to those reported in the aforementioned article by Jensen and Pierson. We therefore reject their conclusions regarding SDS and propose that SDS is an unresolved and costly feedyard problem which warrants further investigation. The study we are initiating represents a new approach to this old problem primarily because it will analyze the impact of various putative risk factors on the incidence of SDS after collecting detailed information from a broad cross section of feedlots.

Proposal

The objectives of this research are to:

1) document the occurrence of SDS in feedlots in the western high plains;
2) describe the post mortem lesions found in SDS cattle;
3) determine the incidence of SDS;
4) determine the association between SDS and various putative factors. Factors include management events, weather, feed and vaccination routines.

To fulfill these objectives we will work with feedyards and their consulting veterinarians and nutritionists throughout the western High Plains. Under the direction of their consulting veterinarians, the feedyards will document the occurrence of the SDS mortalities using a check-off necropsy form with a pictorial observation manual, which will allow us to accumulate accurate and detailed descriptions of post mortem findings. Using these records, we will determine the incidence of SDS and look for subclassifications based on common necropsy findings.

We will perform risk factor analyses to determine the associations between SDS and environmental changes, management factors and nutrition. We will determine populations at risk and analyze associated management events using feedlot records. Also, we will gather environmental parameters from local weather stations. Amassing this data will allow us to evaluate the contribution of a wide variety of factors to the development of these currently unexplained deaths.

Once we identify the factors associated with SDS, the information will allow feedyard management teams

to develop effective strategies to eliminate high-risk situations for SDS. The study will additionally contribute to the study of bovine physiology and nutrition; the definition of the risk factors associated with the occurrence SDS will provide insight into the physiologic responses of cattle to stressors while on high-energy rations. We propose that these insights will help nutritionists, microbiologists, physiologists and behaviorists refine their efforts to make beef production more efficient.

References

1. In: *Proceedings of the Academy of Veterinary Consultants*, March 19, 1976. Cecil Reedy Workshop on Sudden Death, vol. IV, no. 1, pp. 1-65. 2. Pierson RE, Jensen R, *et. al.* Sudden Deaths in Yearling Feedlot Cattle. *J Am Vet Med Assoc* 1976; 169:527-529. 3. Panciera RJ and Williams DE. Sudden Death Syndrome of Feeder Cattle. In: Howard JL. ed. *Current Veterinary Therapy. Food Animal Practice 2*. Philadelphia: WB Saunders Co, 1986; 955-956. 4. Anthony HD. Sudden Death Syndrome in Feedlot Cattle. *J Am Vet Med Assoc* 1972; 60:97. 5. Turner TN. The Sudden Death Syndrome in Feedlot Cattle. *Veterinary Medicine/Small Animal Clinician* 1971; 88:803-808. 6. Coleman JD, Hill JS, Bray HT, *et al.* Prevention of Sudden Death Caused by *Clostridium sordelli* in Feedlot Cattle. *Veterinary Medicine/Small Animal Clinician* 1975; 66:191-195. 7. In: *Proceedings of the Academy of Veterinary Consultants*, March 10, 1977. Cecil Reedy Workshop on Sudden Death, vol. V, no. 1, pp. 1-57. 8. In: *Proceedings of the Academy of Veterinary Consultants*, March 14, 1975. Sudden Death Syndrome Discussion, vol. III. no. 1, pp. 20-35. 9. Jensen R, Pierson RE. *et al.* Diseases of Yearling Feedlot Cattle in Colorado. J *Am Vet Med Assoc* 1976; 169:497-499.

Abstract

Increased reproductive losses in cattle infected with bovine pestivirus around the time of insemination

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Unmated heifers seronegative to bovine pestivirus were used to investigate the effects on conception and embryo-fetal survival of pestivirus infection around the time of artificial insemination. The reproductive performances of three groups were compared; the control group did not become infected during pregnancy, group 1 heifers were infected by contact with a persistently infected cow and calf four days after insemination and group 2 heifers were infected intranasally nine days before insemination. Conception rates and embryo-fetal survival were monitored by serial serum progesterone assays, transrectal ultrasonography and manual palpation of the uterus. The conception rates (determined 20 days after insemination) of 60 per cent (nine of 15) and 44 percent (eight of 18) for groups 1 and 2 were lower than the 79 percent (11 of 14) achieved by the control group. The group 1 heifers subsequently experienced significant embryo-fetal loss, resulting in a pregnancy rate (determined 77 days after insemination) of 33 per cent (five of 15), significantly lower than the control group's 79 per cent (11 of 14). The pregnancy rate of the group 2 heifers (39 per cent, seven of 18) was also significantly lower than that of the controls, largely as a result of the group's poor conception rate. All the heifers diagnosed pregnant 275 days after insemination were induced to calve. No persistently infected calves were born.