Beef: Feedlot Session II

Management of Bovine Respiratory Disease

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The Pathology of Treatment Failure

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It is implied in the title of this paper that death of a feedlot steer or heifer from pneumonia, despite therapy, is to be viewed as a failure of treatment. While it is true that a dead animal can hardly be touted as a resounding success, it is perhaps equally unreasonable to view fatal termination of an overwhelming bacterial infection as our failure. It is at least equally reasonable to ascribe the death of the animal to hopelessly severe initial disease, to preexistant, concurrent or superimposed complicating diseases, or to mistaken diagnosis and thus inappropriate therapy. In this paper I will review some of the published information relevant to so-called treatment failure, and add to it some of my own opinions based upon postmortem examination of feedlot cattle.

What are the lesions in dead feedlot cattle clinically suspected of shipping fever?

Approximately 70 percent of these cattle have severe bronchopneumonia involving 40-60% of total lung mass. Most have fibrinonecrotic pneumonia typical of pasteurellosis and yield a more-or-less pure culture of *P. hemolytica* that often has multiple drug resistance. Some (up to 20% of the total) have suppurative bronchopneumonia with fibrous adhesions, from which a variety of bacteria can be cultured. In some of these the treatment history suggests that these are treatment-modified versions of fibrinous pneumonia, in which therapy has altered the host: parasite balance to buy more time for neutrophil infiltration (IE.pus), fibrinolysis and liquefaction of necrotic lung. A similar progression from fibrinous to suppurative pneumonia is seen in pigs with that species' equivalent of pneumonic pasteurellosis, *Hemophilus (Actinobacillus)* pleuropneumoniae.

About 20% of necropsied feedlot cattle have a disease other than pneumonia as the cause of death. This group, which includes those with diagnoses such as bovine virus diarrhoea, septic peritonitis, and hemophilus septicemia, could perhaps be removed from the category of "treatment failure" and placed in the unpopular category of "diagnosis failure!"

Only 10% of fatalities are shown to have concurrent disease such as I.B.R., B.V.D. or bovine respiratory syncytial virus (B.R.S.V.) infection that may explain the unduly poor therapeutic response. The lesions of I.B.R. and the mucosal disease variant of B.V.D. are well-known and usually are easily seen at postmortem examination. (The role of noncytopathogenic B.V.D. infection in the pathogenesis of feedlot pneumonia remains controversial and, in my opinion, unproven.) In contrast, the lesions of B.R.S.V. are subtle and usually require histologic examination. Even then, the more severe lesions of pasteurellosis will easily obscure the bronchiolitis caused by the virus unless care is taken to submit a portion of "normal" lung for examination. With experience and the help of repeated histologic quality control, the lesions of B.R.S.V. may be detected grossly as undue meatiness and resilience of dorsal caudal lung lobes in a manner similar to atypical interstitial pneumonia.

Are fatalities, if they occur in uncomplicated and adequately treated cases of pasteurellosis, necessarily "treatment failures"?

It is estimated that 3-5% of treated shipping fever cattle die, and most appear to have typical pneumonic pasteurellosis with bacterial isolates sensitive to the employed therapy. It may be that these deaths are preventable by earlier institution of therapy, more aggressive therapy or better nursing care, but is it possible that some or most of these are doomed from the start? Based on subjective criteria, about 25% of fatal cases have lesions older than suggested by the clinical history and are thus good candidates for the category of late initiation of therapy (or late detection). The remaining 75% have lesions that more-or-less match the clinical history and seem not to be the victims of delayed therapy. Despite therapy, 50-60% of lung mass is involved and a considerable proportion of that mass is dead. Before concluding that such cases are failures of treatment (implying that earlier or different therapy would have been successful), consider:

1. Following a heavy challenge, the necrosis and fibrinous inflammation develop and progress with astonishing speed. Even with the most astute clinical detection, therapy may be too late to save the lung.
2. The necrotic lung may not be penetrated by the antibacterial agent employed, or the altered lung environment may impair antibacterial efficacy.
3. Even if therapy destroys the offending agent, the self-perpetuating cycle of necrosis and inflammation will not immediately cease. As in all necrosis and inflammation, release of chemical mediators from damaged tissue, damaged leucocytes and activated plasma will continue to exert noxious local and systemic effects. The old saying "The treatment worked but the patient died" may be more true than we usually think! It is probably true that some cases—in very susceptible cattle or those subjected to unusually massive challenge—are doomed despite our best efforts. Considering the massive destruction in the lungs of the typical fatal case, perhaps we are better advised to wonder why therapy **works** instead of why it occasionally seems to fail!

In conclusion, postmortem examination of apparent treatment failures provides few surprises. The majority of cattle have classical fibrinous pneumonia or its sequel of suppurative bronchopneumonia indistinguishable from cattle dying without benefit of treatment. Only a small percentage have proven concurrent disease such as I.B.R., B.V.D. or B.R.S.V., although careful examination may reveal the actual involvement of these agents to be higher than is currently thought. Delayed detection or therapy seems to have occurred in about one quarter of fatalities, but it is not known whether earlier treatment of these cases would have prevented death or not. The concept of the "hopeless case" needs to be investigated.