SOME REMARKS ON SHIPPING FEVER IN CANADA*

By G. R. CARTER**

Shipping fever, stockyards pneumonia or transit fever, has been a disease problem of cattle in Canada for many years. It is certainly one of the more important causes of losses in beef cattle. It is proposed in this discussion to present the author's views on the nature of shipping fever as it occurs in central Canada. It is certainly evident from the various remarks we hear about this disease that a great deal of confusion still prevails.

Shipping fever of cattle is understood to be the occurrence of pulmonary infections during or after transit. These infections are occasionally complicated by intestinal infections resulting in diarrhoea or dysentery and occur in Canada as a consequence of the following: (1) movement incidental to local sales and shows, and (2) transportation of "feeder and stocker" cattle from western to eastern Canada. The latter traffic takes place for the most part during the fall and early winter. It is the shipping fever seen in the "stockers and feeders" which is of the greatest economic importance.

Cattle shipped from western Canada for the "stocker and feeder" market range in age from six months to two years of age. They are out on range prior to being rounded-up and transported to the nearest stockyards. Some cattle pass through sales before reaching the stockyards. The time spent in the stockyards prior to loading is variable. As soon as these cattle leave the range they move into a radically different environment. The disruption in their feeding and watering habits alone is extreme.

It would be interesting to know just how many young range cattle have chronic pulmonary lesions before leaving western Canada. The figure would probably be high. I have observed lesions of chronic pneumonia in a considerable percentage of normal veal calves at slaughter in southern Ontario. Although bacteria can occasionally be isolated from these lesions, many of the affected lungs appear to be bacteriologically sterile (1). It is not unreasonable to postulate that many of these chronic processes result from low-grade virus infections. My observations suggest that the western cattle most severely affected are those that have had chronic processes in their lungs prior to shipment. It should be appreciated that the animal with chronic pneumonia may be a shedder of large numbers of pathogenic organisms.

With large numbers of cattle crowded together during shipment, some of

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which may already have pulmonary infections, it is readily seen that the dosage of potentially pathogenic organisms to which healthy cattle are exposed in both the stockyards and railway cars must be enormous. All of the unfavourable circumstances referred to above become aggravated during transit to eastern Canada. Besides the crowding, irregular feeding and watering, there is the constant jostling and incessant vibration. It is no wonder that occasionally cattle die of shipping fever during transit to eastern Canada. In fact, it is surprising when one views the whole matter that more animals do not succumb in transit.

Certainly the most striking feature of the description so far is the severe stress to which cattle are subjected after leaving the range. This is no doubt the most important feature of shipping fever. Although it has been common knowledge for many years that physical stresses predispose the animal to infections, it is only recently that the mechanisms involved have been worked out.

When stress is considered in relation to pulmonary disease one immediately thinks of the normal flora of the respiratory tract and the potential pathogens it contains.

In the human being we would suspect the streptococci, the influenza bacilli, or the ubiquitous pneumococci. In the domesticated animals we think first of the pasteurella species. These species, particularly *Pasteurella multocida*, are present in the upper respiratory tract of approximately 10 per cent of normal cattle (2).

It is of interest that Cavallero and Sala (3) have shown that latent infections of *P. multocida* in rats are made acute and fatal infections as a result of inoculating cortisone, a substance released in relatively large amounts during stress.

The stresses to which the western cattle are subjected do not cease until they have adjusted to new environments on farms in eastern Canada. Perhaps the greatest number of shipping fever cases are seen in the eastern stockyards prior to shipment of cattle to farms.

When thinking about shipping fever, it should be remembered that we can not necessarily draw an analogy between this disease and clear-cut diseases of one cause, like hog cholera and rinderpest. One observation which indicates that shipping fever is not comparable with an epizootic, like hog cholera for example, is the fact that shipping fever in the Toronto stockyards is almost non-existent when there are no western cattle passing through the yards. That is, without a continuous supply of susceptible stressed animals, the disease disappears. Such would not be the case with our clear-cut epizootic diseases.

Investigation has shown that the most notorious opportunists in cattle incidental to shipment are: *P. multocida* and *P. hemolytica* (4, 5). However, other organisms occur not infrequently, e.g. corynebacteria and pleuropneumonia-like organisms (4, 5). Although viruses and rickettsia have not yet

been conclusively demonstrated it would seem, in view of the remarks above on chronic pneumonia, that in some instances these agents are the forerunners of pasteurella infections. The conclusion is then that the shipping fever seen in western cattle is a pulmonary infection of varying aetiology initiated for the most part by stress incidental to transit.

It is well known that shipping fever can spread from "feeders and stockers" to native cattle that have undergone no apparent stress. The author has observed this on numerous occasions. These outbreaks are of interest in that any animal in the herd, regardless of age, may be affected. The morbidity is high but if treatment is begun early the mortality is either low or nil. I have recovered *P. hemolytica* from many of the outbreaks of this kind which I have studied. This organism has been shown to produce mild infections in calves not subjected to any particular stress. However, severe infections were produced in calves that had chronic pneumonia at the time of exposure to *P. hemolytica* (1). Because infections could only be produced with primary cultures of *P. hemolytica*, the question of exaltation of virulence would seem to be important. Again, native cattle with pulmonary lesions at the time of exposure are usually severely affected.

The shipping fever seen incidental to livestock shows has not been investigated thoroughly but we do know that *P. multocida* and *P. hemolytica* are frequently implicated. There is considerable evidence that a viral or rickettsial agent may be involved in some outbreaks. Here again it is likely that the aetiology may vary with outbreaks.

Doubtless the tendency to employ the epizootic concept of disease was responsible for calling shipping fever haemorrhagic septicaemia. We know now that although some pasteurella infections terminate as a haemorrhagic septicaemia, the infections caused by *P. hemolytica* do not. Simply because haemorrhagic septicaemias occur occasionally in shipping fever, we have no right to refer to the disease as haemorrhagic septicaemia. It follows likewise that bacterins and antisera prepared for the prevention of haemorrhagic septicaemia are not always adequate for the prevention and treatment of shipping fever.

It would appear that shipping fever will continue to be responsible for enormous losses to the livestock industry unless measures are taken to reduce the stresses incidental to transportation. Because of the wide variety of potential pathogens to which cattle are susceptible, it would seem an almost impossible task to effectively immunize cattle against the infections to which they may fall prey during shipment.

It is of interest that the treatment of shipping fever with sulphamethazine and streptomycin is generally efficacious. This would indicate that although the infection may not be entirely bacteriological, it is in most instances predominantly so. When no response is obtained from the treatment just referred to, terramycin is given intramuscularly each day for three or four days. Dr.

D. C. Maplesden, who is in charge of the Ambulatory Clinic at the Ontario Veterinary College, suggests that treatment on numbers of shipping fever cases should be based upon clinical signs rather than on elevation of temperature, say to 104°F. Realizing that chronic pulmonary lesions predispose the animal to particularly acute pneumonia, he recommends auscultation of the thorax of each case.

Unfortunately we have no bacterin prepared specifically for the prevention of shipping fever as it occurs in Canada. The "so called" haemorrhagic septicaemia bacterins do not contain organisms of the species *P. hemolytica.*

The views advanced in this discussion may be summarized as follows:

1. Much of the confusion in regard to shipping fever has been due to our thinking of it as a clearly defined epizootic disease of one cause.

2. The shipping fever observed in the "feeder and stocker" cattle shipped from western to eastern Canada results for the most part from the stresses incidental to transit. Cattle with chronic pulmonary infections, perhaps viral in nature, are particularly susceptible to shipping fever.

3. *P. multocida* and *P. hemolytica* are frequently observed in the pulmonary infections of shipping fever. Other agents may be involved, e.g. corynebacteria and pleuropneumonia-like organisms and the aetiology in some instances may be multiple.

4. Although haemorrhagic septicaemia may be seen in shipping fever occasionally, there is no justification in using this term as a synonym for shipping fever.

RESUME

Cet article parle de la pasteurellose bovine (shipping fever) au Canada. Les causes déterminantes de la maladie agissent dans deux cas: 1) le transport concomitant aux ventes et aux expositions et, 2) le transport des bestiaux d'élevage ou d'engraissement de l'ouest à l'est du Canada. L'auteur est d'avis que la maladie est surtout attribuable au stress durant le transport. Dans la plupart des cas étudies, *P. hemolytica* et *P. multocida* ont paru jouer un rôle important dans les infections pulmonaires. L'auteur rapporte aussi que des PPLO (pleuropneumonia-like organisms) ainsi que des corynebactériums ont été isolés. On a découvert que les animaux les plus gravement atteints étaient les porteurs de lésions de pneumonie chronique. De plus, l'auteur émet l'opinion que même si dans certains cas des virus et des rickettsies peuvent se trouver à l'origine de certaines explosions de fièvre du transport, il rests vrai que les pasteurellas en sont les agents étiologiques principaux.

En ce qui regarde le traitement, l'auteur est d'avis que l'état du poumon, tel que révélé par l'auscultation, renseigne infiniment mieux sur l'opportunité d'une médication que la présence d'une température de 104°F ou plus.

A la clinique de l'Ecole Vétérinaire d'Ontario, on a utilisé avec une bonne marge de succès la sulfaméthazine, la streptomycine et les antibiotiques à large spectre d'efficacité. Quant aux bactérines commerciales, attendu qu'aucune d'elles ne contient de *P. hemolytica*, on met leur efficacité prophylactique en doute.

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SEROLOGICAL DIFFERENTIATION OF COW POX AND VACCINIA VIRUSES

A technique that has been used extensively of late in analytic studies of both antigens and antisera is based on their diffusion into gels, one or several zones of precipitation developing. Gispen (J. Immunol. 74: 134, 1955) observed that the diffusion pattern obtained in this way for vaccinia antigen with standard vaccinia antiserum consisted of 3 to 6 zones or fractions, two zones being predominant. When cow pox antigen was tested against vaccinia serum, one of these major zones was absent. Gispen also noted differences in the patterns for ectromelia, rabbit pox and fowl pox viruses.

EFFECT OF CHEMICAL DISINFECTANT ON JOHNE'S BACILLUS

Vardan (Amer. J. Vet. Res. 15: 159, 1954) found that the following chemical disinfectants prevented the growth of *Mycobacterium johnei* after 15 min. *in vitro*: cresols, sodium orthophenylphanate, alcohol, mercury bichloride, calcium hypochlorite and methylene blue. The following chemicals in the concentration used, did not prevent the growth of these organisms after 60 min. exposure: sodium hydroxide, quaternary ammonium compounds, basic fuchsin, crystal violet and brilliant green. On the basis of his findings, Vardman concluded that cresolic disinfectants, in the dilutions recommended by the U.S. Bureau of Animal Industry for premises infected with *M. tuberculosis*, would also be satisfactory for *M. johnei*.