Bluetongue in Cattle: A Review

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Historical Aspects

Bluetongue, an arthropod-borne viral disease, occurs in sheep, cattle, goats and wild ruminants (2, 6, 9, 12, 13, 16, 17). Although recognized as a disease entity in sheep in South Africa about a century ago (19), the first epizootic of bluetongue disease in cattle was not reported until 1933, when clinical infection occurred in a small percentage of cattle over an extensive area of South Africa. Although lesions in the affected cattle resembled foot and mouth disease, after sheep inoculation and immunological tests, the definitive diagnosis was bluetongue (1, 10). The disease was given several names including "seerbuck" or "sore-mouth", "ulcerative stomatitis", "pseudo-foot-and-mouth disease", "epizootic catarrh" and "malarial catarrhal fever" (6, 9).

In 1934, Bekker *et al* experimentally infected calves with bluetongue virus and succeeded in producing clinical signs and epithelial lesions similar to those currently observed in animals with natural bluetongue infections (1, 10). Spreull, in 1905, had experimentally infected two calves and a two-year-old ox with bluetongue virus from the blood of infected sheep and observed "a very slight (clinical) reaction" but no lesions in one calf (27). Most researchers to date have found experimental infections in cattle to produce only mild clinical signs and lesions, if any, unless immunosuppressant agents are used to lower the animal's resistance to the virus (4, 16, 20).

Following the initial outbreak in cattle, bluetongue became widespread on the African continent, and has occurred in Cyprus, Pakistan, Japan, Israel, Turkey, Spain, Portugal and the United States (5, 6, 9, 16, 20). It was first isolated from clinically affected cattle in the United States in 1959 (16, 17).

Leudke *et al* in 1970 reported the sequelae of abortion and the birth of calves with congenital deformities (i.e. dwarf-like conformation, crooked legs and gingival hyperplasia) to cattle naturally infected with bluetongue in early pregnancy (21). Also in 1970, bluetongue was presumptively incriminated as a cause of hydranencephaly in calves which were naturally infected *in utero* with a virulent strain. Affected calves, born to dams from areas in which a bluetongue epizootic had previously occurred were free of virus but had antibodies to the bluetongue virus prior to receiving colostrum (23). Similar tetratogenic effects of bluetongue virus had been previously recorded in sheep (4, 13, 21).

Etiology

The agent of bluetongue is a reovirus belonging to the orbivirus subgroup and is in fact the orbivirus "type-virus" possessing the following characteristics (3, 18, 24):

- is arthropod-borne and biologically transmitted by insect vectors of *Culicoides* spp. (11).
- is nonenveloped (naked) and therefore shows relative stability to lipid solvents (ether and chloroform).
- is resistant to sodium deoxycholate and sodium carbonate.
- is heat stable (6) but labile at pH 3.0.
- is susceptible to 3% sodium hydroxide solution and to organic iodides.
- has cubic symmetry and consists of 32 capsomeres which appear large and doughnut-shaped in negative contrast preparations.
- the genome consists of double-stranded RNA.
- ranges from 60 to 90 nm in size.
- is infectious but not contagious (5) and is not spread by contact (6, 9).
- has been isolated from bovine semen (16).
- predominant mode of its release from infected cells is by cell lysis.
- has antigenic independence from other types of viruses.
- bluetongue virus *in vivo* maintains close association with the erythrocytes of infected cattle (16).
- is epitheliotropic (5).

Bluetongue virus has historically undergone antigenic drift, with strains of low virulence becoming highly virulent for cattle (4). It is therefore possible that bluetongue may evolve into a more severe clinical illness than is presently observed.

Animals which are experimentally infected show a slight reaction compared with more severe clinical signs sometimes observed in the field (4, 5, 6, 7, 9, 16). However, calves inoculated experimentally and given an immunosuppressant agent develop marked clinical disease (21). This suggests that the bluetongue virus may be acting in consort with some unknown etiological agent(s) and stress and individual susceptibility factors may be involved (4, 5).

Transmission of the virus may be achieved ex-

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perimentally by blood inoculation and under natural conditions occurs entirely via the bites of at least four *Culicoides* species (sand flies, midges) (2, 4, 5, 6, 9, 11, 12, 13, 15, 26).

The epizootiology of insect-borne viral diseases requires an efficient vector, a readily available susceptible host and a virus reservoir (12, 13, 15). There is a marked climatic, seasonal and geographical prevalence of bluetongue (9, 12, 15, 25, 26). Most cases in moderate temperature zones occur in late summer and early autumn (e.g. in South Africa and the United States) when the vector population is highest. However, in more tropical areas (Kenya), outbreaks may occur at any time (12, 16, 17). The disease is most prevalent in wet seasons and in low-lying areas, conditions which favor insect multiplication (2, 9, 12, 13, 17, 18).

Cattle are much more attractive to *Culicoides* spp. than are sheep and this factor may enhance the importance of cattle as carriers (4, 5, 12, 13, 17, 25). The biological transmission of bluetongue between cattle and sheep by the same culicoid vector has been demonstrated experimentally (22). Cattle and many species of wild ruminants may act as reservoirs of bluetongue virus (6, 7, 9, 15, 17, 20). There is evidence that the bluetongue virus may overwinter in both cattle and *Culicoides* spp. but this aspect of the epizootiology has not been confirmed and other reservoirs may be involved (5, 7, 11, 12, 17). The persistence of bluetongue virus in cattle was initially demonstrated by Spreull in 1905 (27) and more recently researchers have found that the virus can persist in a viremic state in cattle for as long as three years (16). Therefore in endemic areas the extent of bluetongue infection is frequently unknown (4).

The disease may be spread into a previously bluetongue-free area via the vector or by the introduction of a reservoir host. Once bluetongue enters a country, there is little hope of eliminating it (13, 15, 17).

Clinical Diagnosis

The clinical signs and lesions of bluetongue seen in cattle are similar to those observed in sheep and include:

- an initial stiffness which "warms-out" with exercise.
- pyrexia (40 to 41°C).
- ulcerative lesions of the tongue, dental pad, gingiva, oral mucosa and lips.
- excessive salivation with long, stringy strands of saliva hanging from the mouth.
- a "burned" and cracked appearance to the muzzle.
- copious serosanguinous nasal exudate which later becomes mucopurulent and may plug the nostrils necessitating oral breathing.

As the disease progresses, severe lameness due to coronitis may develop together with ulcers of the teats and a subsequent decreased milk production. In chronic cases, debilitation and cracking and sloughing of the skin may occur (1, 2, 4, 5, 6, 7, 10, 16, 20).

The lesions of bluetongue may be confused with infectious bovine rhinotracheitis, bovine virus diarrhea, parainfluenza-3 infection, vesicular stomatitis, malignant catarrhal fever, mycotic stomatitis, rinderpest, photosensitization and foot and mouth disease, thus a reliable diagnosis cannot be made in the field (4, 5, 16). Laboratory diagnosis (serological or virological examination of suspect material) is therefore required (5, 14, 15, 16).

Vaccines

The first vaccine against bluetongue was developed by Theiler in 1906 for use in sheep. Theiler vaccinated sheep with infected blood in which the virus had been attenuated by serial passage in the same species (28). Today, vaccination is routinely carried out in sheep in South Africa using an egg-attenuated polyvalent live virus vaccine containing a number of strains of virus (2). A live virus vaccine is used for sheep in the United States (4).

There is little information regarding vaccination for bluetongue in cattle. Erasmus expresses the need for a vaccine in cattle since cattle play a major role in the epizootiology of bluetongue and the creation of an immune cattle population could be significant in the control of the disease (13). However, there is no guarantee that vaccination would prevent both clinical disease and infection. Since the prevalence of bluetongue and the effect of ovine vaccines on cattle are both unknown, no vaccination program against bluetongue in cattle had been promoted in the United States.

A unique feature of the virus is the existence of at least 16 antigenic strains (recognized by virus neutralization) which vary widely in their virulence in cattle (13, 18, 20). Infected cattle do not appear to develop a significant immunity, however, any immunity is strain-specific and there is no cross-immunity to other strains (2, 13, 16). Therefore, any vaccine developed for use in cattle would have to be polyvalent in order to be effective (6, 12, 13).

Current Status and Significance

The most significant damage inflicted by bluetongue is economic (13, 14, 15, 16). Economic losses are mainly a result of embargoes and stringent testing regulations imposed on the exportation of cattle and cattle semen from infected areas (4, 14, 16). Less extensive direct losses include a decrease in milk production, loss of weight and condition, and the loss of calves and fetuses due to abortion or malformation (16).

Cattle generally are resistant to bluetongue (9, 20) and hence its prevalence in cattle is relatively low (4, 9). The disease is infective for cattle (2),

and although infection is usually inapparent (4, 5, 6, 7, 9, 10, 16), the clinical illness does occur naturally but is uncommon and usually followed by spontaneous recovery (1, 2, 4, 15, 16, 18). However, the severity of bluetongue disease is unpredictable (20) and is influenced by the virus strain, dose, host physiology, breed and other external factors (26). The virus may cause fatalities in a highly susceptible population (2). Outbreaks of the disease are sporadic and the morbidity is variable but usually low (about 5%) (4).

Once bluetongue becomes established in a given locality, complete eradication seems impossible, the disease becomes endemic (13, 15) and therefore most bluetongue-free countries have strict regulations to prevent the introduction of the disease (14, 15). However, the disease was successfully eradicated from the Iberian peninsula by a rigorous quarantine, slaughter and vaccination program (15). To prevent the entry of bluetongue into a country which has effective natural barriers against uncontrolled livestock entry, quarantine measures and serological testing of all ruminants from endemic countries, and adequate treatment of aircraft and other vehicles to prevent the accidental introduction of infective insects must be effected (2, 14).

Most international trade is based on a complement-fixation test for bluetongue (8, 14) and all cattle require two negative tests before entering Canada. Bluetongue regulations in Canada apply to all cattle unless they are imported for immediate slaughter. Bluetongue is a reportable disease under the Animal Disease and Protection Act in Canada. Reactor cattle are destroyed and compensation is paid to the owner.

The Canadian Situation

There have been no publications on the occurrence of clinical bluetongue disease in cattle or sheep in Canada. Although bluetongue has been serologically diagnosed in Canada in imported cattle under quarantine, it has not been isolated from cattle of Canadian origin, and prior to 1975 no Canadian cattle had tested serologically positive. In 1975, 9470 cattle imported from the United States were serologically tested in western Canada and several hundred reactors were found, none of which showed clinical evidence of infection. The reactor animals were destroyed and cattle of Canadian origin, in contact with the reactors, were tested. These animals were all serologically negative except for cattle from a single ranch in the lower Okanagan Valley in British Columbia. Subsequent testing of cattle within a 2400 square mile area revealed an average serological morbidity of 10 to 12%. This outbreak is likely a function of the environmental singularity of the Okanagan Valley. The rest of Canada is believed to be bluetongue-free on the basis of negative results on a nation-wide serological survey. Virus has not been isolated from either reactor domestic or wild ruminants or the vector (*Culicoides variipennis*).

Canada had traditionally been considered free of bluetongue together with the United Kingdom, Ireland, New Zealand and Australia. Currently the bluetongue-free countries e.g. the U.K., Australia, Spain, Greece, East Germany, etc. have placed embargoes on Canadian cattle and semen. Exports have been regained to some other countries as a result of quarantine measures and controls exerted.

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BOOK REVIEW

Atlas of Veterinary Ophthalmic Surgery. S. I. Bistner, G. Aguirre and G. Batik. Published by W. B. Saunders, Toronto. 1977. 302 pages. Price $31.90.

This book should fill a distinct void in small animal veterinary medicine. There are references in the book to ophthalmic surgical problems in the equidae but the emphasis is on small animals – primarily the dog. The book brings together much of the knowledge of the last ten years and the stress is, as the title states, on surgery.

The first thirty-five pages discuss the principle of instrumentation, storage, sterilization, delivery and cleaning. The preparation of the surgical area, selection of suture material, cryosurgical equipment, irrigating solutions are covered very thoroughly.

The chapters in sequence then discuss in depth anesthesiology, plastic surgery, canthoplasties, cosmetic and therapeutic lid surgery, entropion, ectropion and the nasolacrimal system. Cornea anatomy with further discussion of repair, physiology, diseases and surgery is well done. Lens surgery is right up to date including phacofragmentation and ultrasonics.

Glaucoma, another disease with its attendant surgical procedures, has been pulled together and takes up twenty pages in chapter 10.

The appendices include breed predisposition to eye diseases, excellent color plates of various eye diseases and a listing of manufacturers.

The principle author Dr. Steve Bistner is an authority on eye diseases in animals. Formerly on staff at Cornell University, he is now at the University of Minnesota. Dr. Gustavo Aguirre is Assistant Professor of Ophthalmology at the School of Veterinary Medicine in Philadelphia. George Batik, M.S. from Cornell University did the excellent illustrations on the surgical techniques.

Any minor criticism of this book would only be that there is not enough emphasis on sedation and protection of the eyes postoperatively and that the embryo ophthalmic surgeon should realize that when he is splitting an eyelid or incising an eyeball he better know what he is doing.

At the price the book is a good investment. *J. A. Hutchison.*