I. Vesicular Stomatitis

Vesicular stomatitis (VS) is caused by a rhabdovirus. It usually occurs in warm weather and may be insect-borne, but the method of transmission is not clear.

The lesion begins as a raised, flat, pale papule only a few millimeters in diameter. These papules become hyperemic and develop into 2-3 cm vesicles that coalesce. The vesicles may rupture and become superficial erosions. These erosions may still have an epithelial flap, which helps to differentiate the lesions from other erosions such as those that occur in bovine virus diarrhea. The lesions heal in one to two weeks, unless there is secondary bacterial infection or trauma caused by milking. Usually the viremia persists longer than the lesions, however there are no systemic signs. Lesions usually are confined to the oral cavity but can occur on the feet and teats.

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Malignant Catarrhal Fever—Case Histories

Larry Hutchinson *D.V.M.*
Extension Veterinarian
The Pennsylvania State University
University Park PA 16802

Incidence

- Sporadic single cases in North America, Europe, Australia, New Zealand
- Enzootic form in Africa (African or wildebeest type?)

Clinical Picture

- 3—8 week incubation period
- nasal—ocular signs: mucopurulent nasal discharge; muzzle and buccal necrosis; dyspnea; panophthalmitis; blindness
- nervous signs, extreme depression, incoordination, weakness, tremors
- lymphadenopathy
- G—I, signs from constipation to profuse diarrhea
- persistent high fever is characteristic
- contact with sheep

Case Histories

Farm A: 135 head purebred dairy herd in Central Pennsylvania; show cow purchased January, 1978; she

was hauled in a truck (Sept. '78); truck had to be used to haul sheep and goats; cow became ill December 5; nasal discharge, corneal opacity, blindness, anorexia, febrile for 11 days until death.

Post-Mortem: muzzle and tongue erosions and necrosis; lung - emphysematous; lymphadenopathy; keratitis.

Histopath: perivascular cuffing in cerebrum, cerebellum, and spinal cord. lymph nodes - hyperemia and hyperplasia.

No other cases have occurred in this herd.

Farm B: Report by Pierson et. al., JAVMA, 8/15/73 Colorado feedlot; of 231 steer and heifers, 87 died within 68 days; 100% mortality of visibly sick cows; panophthalmitis, diarrhea, leukopenia, fever; sheep on same farm but not in direct contact.

Farm C: Western Pennsylvania dairy herd; 55 head, Holsteins and Guernseys; May and June 1976-8 cows died; 1 cow died in each of the months of July, October, November and December.

Signs: high fever, lacrimation, keratoconjunctivitis, blindness, incoordination, some cows aborted; diarrhea, some cows showed hemorrhagic cystitis.

Autopsy: two cows posted at P.S.U.; one cow posted at Ohio State University; 1 head examined at P.S.U.: pneumonia, emphysema; hemorrhagic cystitis, hemorrhagic areas of small intestine, non-suppurative meningoencephalitis, CNS perivascular cuffing; vasculitis; 1 cow-hepatitis and nephritis. One or more cows recovered. 17 serum samples submitted to Plum Island - negative for African-strain MCF virus neutralizing antibodies; sheep contact on farm; a new ram was purchased from West Virginia shortly before outbreak; no young stock were affected.

Farm D: 120 cow dairy herd; northeastern New York; high fever; Keratitis; ("looked like peracute **IBR**"); depression, incoordination; lymph node hypertrophy; 2 sheep on farm had free access to adult cattle; several pigs had contact with cattle; sheep and swine were asymtomatic; mortality - 1/3 of adult cattle; some recoveries; Plum Island - several positive titers to African-Strain MCF.

Bovine Leukemia Virus: Transmission and Diagnostic Tests

Simon J. Kenyon, *Ph.D., MRCVS*
Assistant Professor of Medicine
New Bolton Center
University of Pennsylvania
Kennett Square, PA 19348

I. Expression Of BLV In The Individual Animal

BLV infection in cattle takes the form of a persistent infection of lymphoid cells. Infection of cells other than lymphocytes or lymphoid tumor cells has not been demonstrated. Although the viral genome is integrated into the DNA of many host lymphocytes, actual expression of the virus or its antigens is rare. BLV has not been observed budding from *fresh* lymphocytes or tumor cells of BLV-infected animals, nor have BLV virion antigens been detected in the same fresh tissues. Thus in BLV-infected cattle, expression of the virus is repressed. This is in striking contrast to FeLV infection in the cat where plentiful expression of FeLV occurs and FeLV antigens are easily detected by a fluorescent antibody test performed on fresh blood smears.

Although direct detection of BLV or its antigens in freshly-harvested bovine cells is not possible, all cows infected with BLV have persistent antibody titers to a number of BLV antigens, the most important of which, from the point of view of current diagnostic tests, are the envelope glycoprotein (gp70) and the major internal virion antigen (p25). Detection of these antibodies forms the basis of the most widely applied diagnostic tests for the presence of BLV infection in cattle.

Another consequence of BLV infection is the development in some cows of a persistently elevated peripheral blood lymphocyte count. This persistent lymphocytosis (PL) is a genetically determined response to BLV infection, and before the development of serological tests for BLV, it was widely used to indicate the presence of lymphosarcoma risk in individual animals or within herds.

II. Diagnostic Tests For BLV Infection

A. Hematologic Keys

These are based on the presence of PL. Normograms for the expected maximum lymphocyte counts in different