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TECHNICAL BULLETIN 18

APRIL 1957

Sporadic Bovine Encephalomyelitis

Veterinary Department

AGRICULTURAL EXPERIMENT STATION
South Dakota State College of Agriculture and Mechanic Arts
College Station, Brookings, South Dakota



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Sporadic Bovine Encephalomyelitis

G. S. HARSHFIELD¹

Sporadic bovine encephalomyelitis (SBE) is a specific infectious disease of cattle caused by an agent belonging to the psittacosis-lymphogranuloma group of viruses. This disease is not of frequent occurrence in this country, so many livestock men and veterinarians are not familiar with it. In herds where SBE has occurred it has sometimes accounted for serious loss, not only in deaths but also in weight loss.

SBE is of importance to the veterinarian from the standpoint of differential diagnosis. In a few herds it has been mistakenly diagnosed as shipping fever. However, it is an infection that is more apt to be confused with other diseases affecting the nervous system.

Outbreaks of SBE in eastern South Dakota and western Minnesota have afforded material for observations and study of the disease. This bulletin reports the results of the studies at this laboratory together with reference to contributions by others.

HISTORY OF SBE

McNutt (6) in 1940 provided the first description of SBE, having observed cases in several herds in Iowa. The first herd providing material for experimental study was owned by a Mr. Buss and the names "Buss encephalitis" and "Buss disease" were used to identify the disease in the earlier studies. These terms are still in use as synonyms for SBE.

In this and later studies reported by Iowa workers (6, 7, 8, 9, 10, 17) the infectious nature of the disease was proven and the causative agent, although not identified, was considered to be a virus. The disease syndrome differed sufficiently

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Acknowledgement for assistance on this project is given Mary S. McCarty, Elaine Kerner, John McAdaragh, technicians, and Drs. T. A. Dorsey, J. B. Taylor, A. B. Hoerlein, and Carl E. Rehfeld, who are or have been members of the staff during the period covered in this report.

from other encephalitides of cattle to conclude that this was a disease not previously recognized.

In 1941, Boughton (1) reported the occurrence of SBE on 12 ranches of the Edwards Plateau region in west Texas. The outbreaks had occurred over a period of several years. Price and Hardy (16) in a later report stated that SBE is "rather well distributed" in west-central counties of Texas where it is called "brain fever" by ranchmen. Texas workers confirmed the findings of McNutt on the infectious nature of the disease.

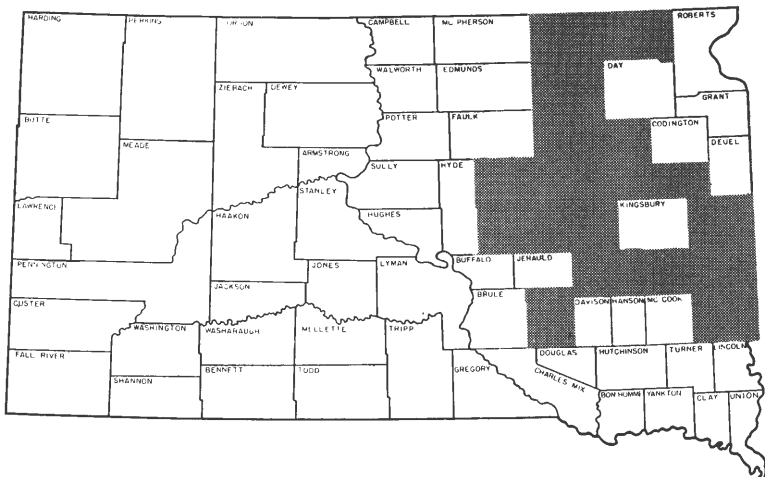
Sporadic bovine encephalomyelitis has been diagnosed in herds of South Dakota and western Minnesota since 1945. The first published report (4) in 1951 recorded diagnosis in eight herds on widely separated farms. In later reports (3, 5) additional outbreaks in this area are recorded. SBE now has been diagnosed in 24

herds over the 12-year period. Through the cooperation of Dr. H. A. Wenner, University of Kansas School of Medicine, and Dr. R. W. Menges, Public Health Service, Federal Security Agency, virus strains recovered from South Dakota outbreaks as well as strains from cases occurring in Missouri were studied and identified as belonging to the psittacosis-lymphogranuloma groups (11, 12, 13, 19, 21).

OCCURRENCE AND DISTRIBUTION

Geographic Distribution. A review of published reports provided data regarding the geographic distribution of SBE in the United States. The following 15 states were listed one or more times: California, Delaware, Idaho, Illinois, Indiana, Iowa, Kansas, Maryland, Michigan, Minnesota,

Figure 1. Areas in gray are counties in South Dakota in which sporadic bovine encephalomyelitis has been diagnosed.



Missouri, Oklahoma, South Dakota, Texas, and Wisconsin.

An outbreak of encephalomyelitis in cattle in Japan, which was similar to SBE in the United States, was reported in 1954 (14, 15). The agent causing the disease was recovered and shown to have the characteristics of the virus strains in this country.

In South Dakota. Sporadic bovine encephalomyelitis has been diagnosed in 21 herds in South Dakota and in 3 herds in western Minnesota by workers at this station. The South Dakota herds were located in 14 counties (see figure 1). It is believed that SBE has wider distribu-

tion, both within the state and nationally, than is indicated by the diagnosed outbreaks. Histories of cases have been provided in several instances which were suggestive of SBE but materials were not made available for laboratory study. Experience both in natural herd outbreaks and in transmission studies has demonstrated that many cases show only mild symptoms and would not be detected under average herd management.

SBE can occur at any season of the year. Although all of the laboratory diagnoses were made in the months from April to December (see table 1), the histories provided

Table 1. Morbidity and Mortality in Herds Affected with SBE, 1945-1956

| HERD NO. | DATE OF DIAGNOSIS | | DURATION OF OUTBREAK | ANIMALS IN HERD | NUMBER | |
|-------------|-------------------|------|-------------------------|--------------------|--------|--------|
| | MONTH | YEAR | | | SICK | DEATHS |
| 1 | July | 1945 | 4 wk. | 17 | 7 | 7 |
| 2 | May | 1946 | 2 mo. | 88 | 5 | 2 |
| 3 | August | 1946 | 1 mo. | 16 | 8 | 5 |
| 4 | May | 1947 | 4 wk. | 125 | 32 | 16 |
| 5 | April | 1949 | 5 mo. | 120 | 17 | 9 |
| 6 | July | 1949 | ----- | 60 | 9 | 5 |
| 7 | October | 1949 | 3 wk. | 40 | 4 | 2 |
| 8 | November | 1949 | ----- | 134 | 15 | 3 |
| 9 | December | 1949 | 4 wk. | ----- | 2 | 2 |
| 10 | April | 1950 | 2 wk. | 35 | 2 | 1 |
| 11 | May | 1951 | 5 wk. | 95 | 5 | 3 |
| 12 | June | 1951 | 6 mo. | 150 | 14 | 2 |
| 13 | July | 1951 | ----- | 13 | 4 | 2 |
| 14 | October | 1951 | ----- | ----- | 2 | 1 |
| 15 | April | 1952 | 1 wk. | ----- | 1 | 1 |
| 16 | May | 1952 | 3 mo. | 95 | 5 | 3 |
| 17 | August | 1952 | 1 wk. | ----- | 1 | 1 |
| 18 | September | 1952 | 4 mo. | ----- | 9 | 3 |
| 19 | July | 1953 | 6 mo. | ----- | 12 | 2 |
| 20 | July | 1954 | 1 mo. | 30 | 3 | 3 |
| 21 | June | 1954 | 3 mo. | 80 | 9 | 5 |
| 22 | August | 1954 | 2 wk. | ----- | 3 | 2 |
| 23 | September | 1956 | ----- | ----- | 2 | 2 |
| 24 | October | 1956 | 3 wk. | 105 | 8 | 1 |

by veterinarians and owners indicate that some of the outbreaks started in the first quarter of the year.

Morbidity and mortality data are not available on all of the infected herds. In 16 of the herds with a total of 1,203 cattle of all ages, the morbidity was 147 animals (average 12.2 percent) and the mortality 69 animals (average 5.7 percent of the herd, 47 percent of the affected animals). The proportion of the animals in the herd showing signs of infection varied widely in different outbreaks, ranging from 5 to 50 percent. The death loss was also variable, ranging from 6 to 100 percent of the sick animals.

Both young and mature animals have shown symptoms in outbreaks of SBE. It is worthy of note that the highest morbidity and mortality occurred in young animals of 1 year or less in age. In one outbreak in a herd of 125 head, there were 32 clinical cases of which 16 died. There were no deaths among 9 cases in mature cows, 7 deaths in 14 cases in heifers and steers, and all of the 9 calves which developed symptoms died. In another herd of 17 calves, 7 became sick and died.

The duration of outbreaks has varied in the different herds. In each of two herds only one clinical case developed with deaths occurring within a week. In larger herds and with greater spread of the infection, the duration was lengthened to several weeks or several months, with new cases appearing at intervals of a few days to 3 to 4 weeks.

There have been no confirmed reports of the spread of SBE to neighboring farms in the South Dakota outbreaks. There was a possible recurrence on one farm the next year but this was not verified by clinical or laboratory investigation.

The epizootiological features of SBE outbreaks in South Dakota have not differed greatly from those observed and reported by McNutt (9) in Iowa. He also noted that morbidity and mortality was greatest among animals of younger age, with a mortality of about 50 percent of affected animals. However, he does point out that the disease may persist in a herd for several years, causing an occasional loss. One farm is cited where SBE was diagnosed for 5 consecutive years. Boughton (1) reported the occurrence on 2 successive years in a herd in Texas. Price and Hardy (16) mention that the infection may disappear from a herd spontaneously but reappear several years later. They indicate that the disease is more mild with a mortality of less than 10 percent of affected calves in the Texas herds.

SYMPTOMS

As the mode of spread and route of infection in natural outbreaks has not been determined with certainty, the incubation period under field conditions is not known. McNutt and Waller (10) gave the range from 4 to 27 days. In South Dakota trials with experimentally infected calves, the time from exposure to the first rise in body temperature varied with the route of

administration of the virus. When the virus was given per orum, the incubation period was 12 to 14 days; when inoculated subcutaneously or intraperitoneally it was 5 to 7 days, and with intravenous and intracerebral inoculation, 2 to 4 days.

The first detectable symptom of SBE is a rise in body temperature ranging from 103° F. to 107° F. Usually the temperature remains elevated throughout the course of the disease, gradually returning to normal within 7 to 10 days in case of recovery. The temperature may be found subnormal in fatal cases as death approaches.

The appetite is not immediately affected. Calves may eat well and appear active for a day or two after the first temperature rise but gradually become depressed and less interested in food. As depression and inappetence progresses, loss of weight becomes evident. When the animal is standing one can notice the gaunt appearance and the "tucked up" abdomen.

Some of the cases, especially young calves, become prostrate soon after appearance of symptoms. If assisted, they may be able to stand but show poor muscular coordination when forced to move. The incoordination is most noticeable in the fetlock joints as evidenced by weakness and "knuckling over" when walking. Either front or hind limbs may be more affected. The weakness causes the animal to weave from side to side.

Some animals are unable to stand alone. A few have shown opisthotonus and these were in-

variably fatal cases. Others may remain down for several days but eventually make a recovery.

Symptoms such as circling, pushing, and excitability which frequently characterize other diseases of the nervous system are generally not seen in SBE. Depression and incoordination are the main symptoms denoting encephalomyelitis.

Symptoms involving the respiratory and digestive systems have been seen in a few cases of SBE but have not been of certain diagnostic significance. These symptoms were observed as a mild catarrhal nasal discharge and diarrhea sometime during the course of the disease.

The clinical course of the disease is from 3 to 4 days to about 3 weeks. The severity of the symptoms is not a dependable clue on which to base a prognosis. Prostrate animals may make a complete recovery although the prognosis in severely affected young calves is poor.

In inoculated calves, symptoms have generally been mild. Only one animal which was inoculated intravenously with SBE virus went down and was unable to stand, even when assisted to its feet. A necropsy was made before the disease was allowed to run its course. In all inoculated calves, regardless of the route of administration of virus, an elevation in temperature marked the onset of infection. They continued to eat, although the food intake was sometimes markedly reduced for 3 to 4 days during the peak of the infection. Weight loss and depression

were noticeable in most experimentally produced cases, but incoordination and other signs of nervous involvement were rarely observed. Body temperatures above 103° F. persisted for 7 to 10 days. Animals not sacrificed for experimental data recovered.

PATHOLOGY

Gross Lesions. Although the name encephalomyelitis has received general usage in designating this disease, lesions involving the nervous system are not as evident at necropsy as those involving the serous membranes of the body cavities. However, in cases which terminate after only 2 or 3 days duration or in those sacrificed in the earlier stages of the illness, the sero-

sal changes are also minimal or absent.

If the examination is carried out after 4 or 5 days, a generalized peritonitis is a constant lesion. There is an increase in serous fluid with a stringy network of fibrin over the omentum and among the coils of the intestine. Congestion and small hemorrhages of the peritoneum are observed accompanying the exudation.

A slight enlargement of the spleen has been seen at necropsy in earlier stages but not in cases of longer duration. A thin plastic film of fibrin often covers the capsule of both the spleen and liver. No gross kidney lesions have been associated with SBE. Upon examination of cases which have been sick for about 10 days or longer, flattened plaques or irregular shaped masses of fibrin may be found in the peritoneal cavity instead of fibrinous strands. Lymph nodes in the body cavity are noticeably enlarged and on cutting are edematous. Remarkably, fibrous adhesions are slight or absent in those cases killed for necropsy in later stages of SBE.



Figure 2. Thoracic wall of calf 13 days following inoculation with SBE virus. Dark area in center is hemorrhagic with a layer of fibrin over a part of the inflammatory area.



Figure 3. Left lung of calf. Apical, cardiac, and lower portion of diaphragmatic lobes are partially covered with fibrin.

The pleural cavity and also the pericardial sac often contain serous or serofibrinous exudate similar to that found in the peritoneal cavity (see figures 2 and 3).

Except for congestion and edema of meningeal coverings, gross lesions of the central nervous system are usually absent.

Histopathology. The microscopic findings involving the serous membranes are fibrinous peritonitis, pleuritis, pericarditis, and epicarditis. A cellular reaction is found in those surface areas in which there is infiltration of mononuclear and oc-

casional neutrophil and eosinophil cells, new fibroblasts and endothelial proliferation (see figure 4). Leucocytes are found in the fibrinous exudate adhering to the surface.

In the parenchyma of the liver and kidney small foci of lymphocytes have been found (see figure 5). The occurrence of these lesions has been irregular and it is not known that they represent tissue reaction to the SBE virus.

Meningeal infiltration with predominately mononuclear cells is observed over the entire brain and is

Figure 4. Capsule of spleen showing cellular infiltration, fibroblastic and vascular proliferation of the serosal covering. X80

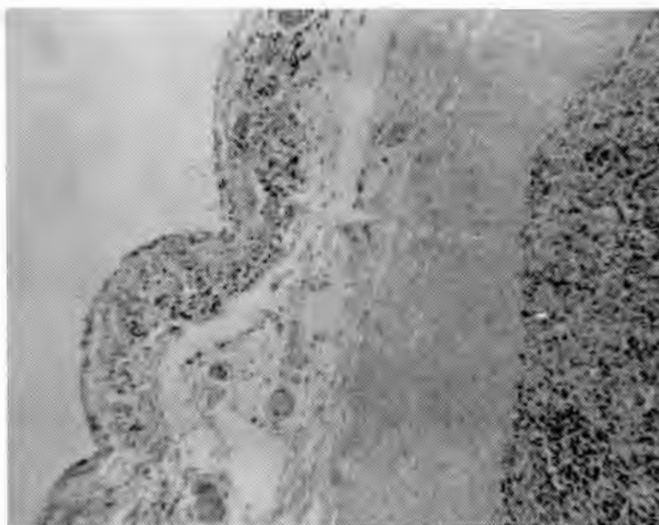
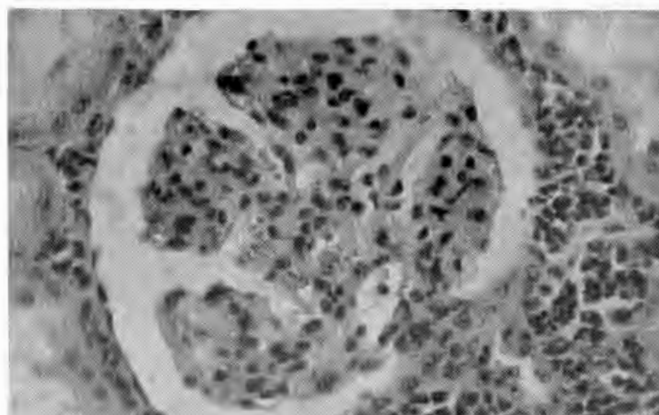


Figure 5. Interstitial infiltration with lymphocytes in the kidney. Such lesions are of irregular occurrence in SBE. X340



especially marked at the base of the brain and over the cerebellum, extending into the sulci (see figure 6). Inflammatory lesions are found microscopically in all parts of the brain and at different levels of the spinal cord. These lesions are manifested by endothelial proliferation and perivascular infiltration, focal abscessation and encephalomalacia, neuronal degenerative and necrotic changes (see figures 7, 8, 9, 10). In the brain, vascular and focal lesions are encountered in all parts, although the intensity of the cellular reaction appears less in the cerebrum than in the medulla and cerebellum. Both gray and white matter are involved.

Focal areas are associated with liquefaction, abscess formation, and degenerative changes in neurons in the area. The infiltrating cells in these lesions are principally mononuclear cells although neutrophils are often abundant in tissues collected from cases in earlier stages. In the cord, the inflammatory lesions involve the gray matter (see figure 11). Menges, Harshfield, and Wenner (13) state that the lesions of the central nervous system are suggestive of secondary changes following generalized vascular damage.

ETIOLOGY

An infectious agent, which Wenner et al. (19) identified as belonging to the psittacosis-lymphogranuloma group of viruses, is the specific cause of SBE. Minute round elementary bodies can be demonstrated in infected tissues by appropriate staining methods, although

they are never numerous in exudates from field or experimentally produced bovine cases. Macchiavello's stain has been the most satisfactory for demonstrating elementary bodies. They are found with difficulty or not at all in tissue sections stained with hemotoxylin-eosin. By the Macchiavello-method they stain pink-red in color and are found singly and in small clumps free in the exudate or contained within macrophages.

Elementary bodies are more numerous in preparations made from infected yolk sac membranes from chicken embryos inoculated with SBE virus. McNutt (9) mentioned finding coccus-like bodies in tissues from SBE cases. Although not identified, these probably were elementary bodies.

Isolation of the Infectious Agent.

By guinea pig inoculation, virus was demonstrated in a variety of tissues from field cases. Spleen, liver, and brain have been tissues of choice. In guinea pigs inoculated intraperitoneally with infected tissue emulsions, serofibrinous peritonitis and sometimes pleuritis was produced within 3 to 6 days. Few of the inoculated animals died from infection but the lesions were evident at necropsy 6 to 10 days after inoculation and virus was present in the exudate and visceral organs. Liver and splenic tissues were chosen for additional inoculations or for storage.

Strains of SBE virus from five South Dakota outbreaks have been adapted to chicken embryos following guinea pig passage. Embryos

Figure 6. Meningeal infiltration with predominately mononuclear cells over the cerebellum. X80



Figure 7. Large area of necrosis in the medulla. Blood vessels near the area of softening show perivascular infiltration. X32

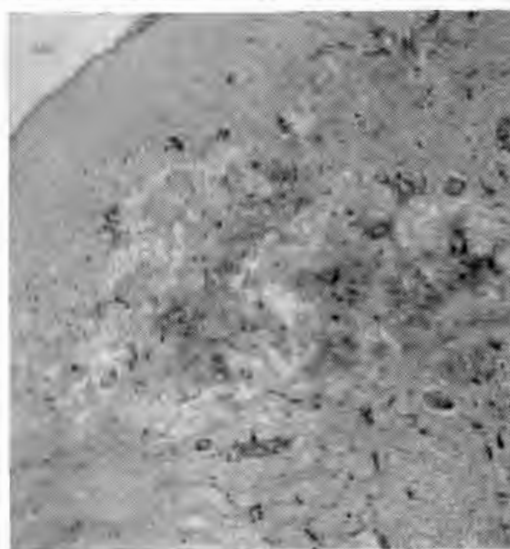
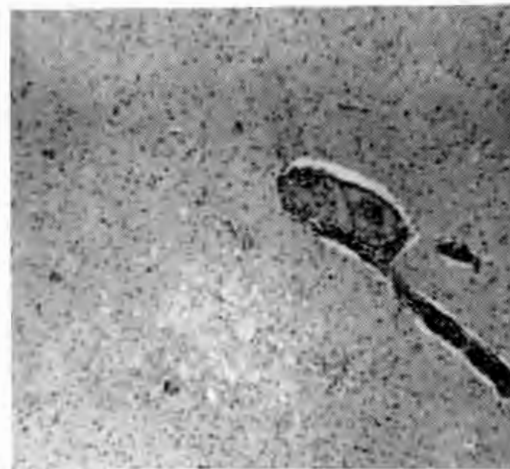


Figure 8. Cerebrum. Perivascular infiltration with mononuclear cells. A small area of softening can be seen near the vessel. X80



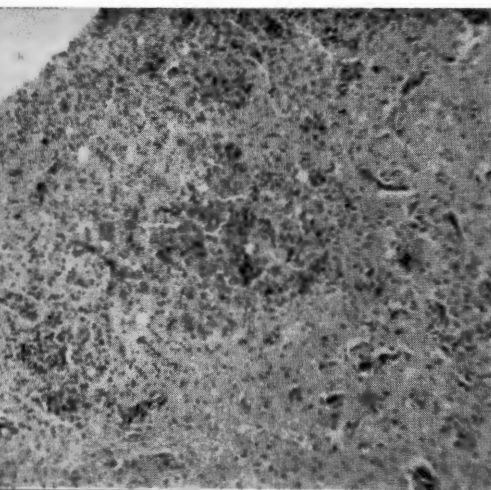


Figure 9. Abscess in the thalamus. The infiltrating cells in this lesion were predominately polymorphonuclear. X80

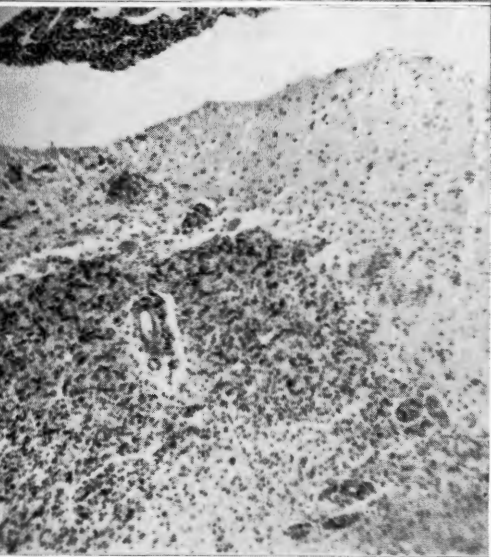


Figure 10. Area of the cerebellum with perivascular infiltration. Note the meningitis. X80

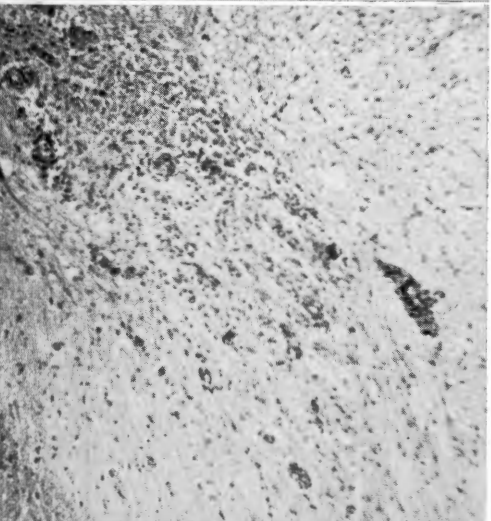


Figure 11. Lesion in the ventral horn of the gray matter of the spinal cord in the lumbar region showing diffuse and perivascular cellular infiltration. X80

were killed in 3 to 8 days when inoculated via yolk sac after a 7-day preliminary incubation period. On the first embryo passage from guinea pig tissues, the death pattern was usually irregular but embryo mortality occurred in about 5 days and was generally 100 percent on continued passages. Virus was present in high concentration in the yolk sac membrane and yolk material. Embryo LD₅₀ determinations on yolk sac material for three strains were as follows:

H strain 96th embryo passage
10^{-6.5}

S strain 34th embryo passage
10^{-6.7}

L strain 32nd embryo passage
10^{-6.8}

Properties of the Virus. The SBE virus has not been grown aerobically or anaerobically in bacteriological media. When subjected to heat in a waterbath at 56° C., pathogenicity of the virus for chicken embryos was retained at 2 minutes. At 5 minutes some inactivation had occurred, while at 10 minutes it was completely inactivated. The virus

was no longer infective for chicken embryos following 5-minute treatment with 2 percent and 5 percent sodium hydroxide, 5 percent compound cresol, or 0.3 percent quaternary ammonium compound (paraisobutyl phenoxy ethoxy ethyl dimethyl benzyl ammonium chloride). The agent resisted freezing. Yolk material retained infectivity for over 1 year at -20° F. Lyophilized yolk material stored at -20° F. for 3 years retained complete pathogenicity. Storage of infective tissues at 45° F. was less satisfactory as pathogenicity was gradually lost during the first month.

Susceptibility to antibiotics has been tested in chicken embryos. The virus retained infectivity for the embryo when treated with penicillin, dihydrostreptomycin, and chloramphenicol. Results with chlortetracycline and oxytetracycline are given in table 2.

It can be noted that both tetracycline compounds exerted some action toward preventing infection in embryos. Results were irregular with oxytetracycline, while a relatively large dosage of chlortetracy-

Table 2. Antibiotic Action of Tetracycline Compounds Against SBE Virus in Chicken Embryos

| MILLIGRAMS PER EGG | CHLORTETRACYCLINE | MILLIGRAMS PER EGG | OXYTETRACYCLINE |
|-----------------------|-------------------|-----------------------|-----------------|
| 0.0 | 30/31* | 0.0 | 26/26 |
| 0.1 | 10/10 | 0.062 | 1/10 |
| 0.2 | 20/20 | 0.125 | 2/19 |
| 0.5 | 12/30 | 0.25 | 3/27 |
| 1.0 | 5/28 | 0.5 | 5/22 |
| 2.0 | 1/10 | 1.0 | 3/17 |
| 5.0 | 1/8 | | |

*Numerator—embryo deaths; denominator—embryos inoculated.

cline was required. Although penicillin and dihydrostreptomycin failed to prevent infection, they have been found useful in the laboratory to reduce contaminants in infective materials for inoculation work.

Wenner et al. (19) reported irregular results with penicillin in susceptibility tests. Price and Hardy (16) have studied the antibiotic susceptibility of a SBE strain, employing a number of antibiotics not included in the trials at this laboratory. They noted pronounced activity with tetracycline, erythromycin, chlortetracycline, carbomycin and penicillin.

Stearns and McNutt (17) demonstrated virus in Berkefeld filtrates. Wenner et al. (19) reported that virus was present in filtrates obtained with fritted glass and Berkefeld N filters. At this laboratory filtrates obtained with Selas filters with pore radius less than 20 microns (XFF porosity) were noninfective for chicken embryos. Electron microphotographs of elementary bodies showed them to be spherical bodies of approximately 375 millimicrons (14).

SUSCEPTIBILITY OF OTHER SPECIES TO SBE

The SBE viral agent caused active infection in guinea pigs. Intraperitoneal inoculation resulted in serofibrinous peritonitis and sometimes pleuritis but not encephalitis. There was an increase in fluid which was frequently blood tinged as a result of hemorrhages in the body wall and diaphragm. Fibrin

was found adherent to the coils of the intestine and a plastic fibrinous exudate usually covered the capsules of the liver and spleen. Both the liver and spleen were enlarged. If the thorax was involved, it contained an accumulation of fluid which coagulated after opening the cavity.

Few guinea pigs were killed by the virus. When death occurred it was usually from the accumulation of pleural fluid.

By the intracerebral route, encephalitis was produced. Subcutaneous inoculation resulted in an extensive tissue reaction around the inoculation site. Although infection was evident as early as 3 days following intraperitoneal inoculation, lesions were most pronounced at 6 to 8 days.

McNutt (9) reported the results of inoculation of other animal species. In rabbits, an unapparent infection was produced but virus was recovered 7 days following inoculation. He carried out a limited number of trials with sheep, swine, goats, and turkeys and did not produce apparent disease. One of nine chicks inoculated intracranially died of encephalitis.

Wenner et al. (19) tested the pathogenicity of the virus in rabbits, swiss mice, cotton rats, hamsters, and rhesus monkeys. Encephalitis, but not peritonitis, was produced in rats, hamsters, and monkeys by intracerebral inoculation. Peritonitis was produced in hamsters in this laboratory on intraperitoneal inoculation.

Chicken embryos were killed by

yolk sac inoculation of SBE virus. The principal change noted was an increase in the size of the yolk sac brought about by absorption of fluid from the allantoic sac. The yolk material was thinner in consistency and was easily withdrawn with syringe and needle.

IMMUNITY

Animals which recover following infection with SBE are immune to further challenge with SBE virus. This resistance to challenge has been demonstrated in both cattle and guinea pigs.

Trials have been conducted to obtain some measure of the immune status by neutralization of active virus with serum from convalescent calves. These trials were conducted by using constant amounts of serum with serial 2-fold dilutions of virus and by using serum dilutions with a constant virus dilution. The serum-virus mixtures, after a period of 1 to 2 hours incubation, were inoculated into embryonated eggs. The results of serum neutralization trials have been very irregular and the procedure has not provided a satisfactory measure of immunity.

No reaction was obtained in SBE convalescent guinea pigs with lymphogranuloma venereum skin test antigen (Frei antigen).

Wenner et al. (21) reported results of complement-fixation (CF) tests on serums from a series of experimental calves using bovine encephalitis, lymphogranuloma venereum (Lygranum), and ornithosis antigens. CF antibodies were demonstrated in serums collected after recovery.

In another paper Wenner, Menges, and Carter (20) gave additional information on CF antibodies in bovine serums. Cattle known to have had SBE, cattle known to have been exposed, and cattle with no history of either infection or exposure, were included in the studies. From this survey they concluded that CF antibodies in titers of 1:8 or above are present in 50 percent or more of adult cattle in the area of the survey in midwestern United States.

The authors recognized that the serologic response in cattle might in some cases have resulted from some agent other than the SBE virus, for a group relationship exists among members in the psittacosis-lymphogranuloma venereum group of viruses. Such an agent may exist in the virus first recovered from feces of apparently normal cattle by York and Baker (22).

Serum samples collected prior to exposure and in convalescent stages from calves used in experiment at the South Dakota laboratory were checked for antibodies by direct complement-fixation with 6BC psittacosis antigen by Dr. John P. Delaplane, Department of Veterinary Microbiology, Texas A and M. No significant serum titers were obtained in serums prior to exposure but convalescent serums demonstrated titers of 1:4 to 1:64.

Immunization Trials. Since animals have demonstrated resistance to a second infection following recovery from SBE, an attempt was made to stimulate immunity by vaccination with virus inactivated with

formalin. Guinea pigs and calves so treated were still susceptible when challenged with active SBE virus.

York and Baker (22) described the virus which they recovered from cattle feces as being a member of the psittacosis-lymphogranuloma group and without pathogenicity for cattle when exposed by various routes. In guinea pigs inoculated intraperitoneally it produced lesions closely resembling those produced by SBE virus.

At the South Dakota laboratory, a search has been made for virus with like characteristics in cattle in the area. A virus (or viruses) was demonstrated by guinea pig inoculation in feces of 5 of 7 calves which had been inoculated with SBE virus and in 12 of 14 normal calves.

Using strains recovered from normal calves, it was found that they failed to produce more than a mild febrile reaction and no other clinical response when inoculated into calves by intraperitoneal or subcutaneous routes. Upon necropsy, the calves showed no lesions involving serous membranes as is characteristic of SBE.

An experiment was conducted with guinea pigs to determine whether a virus strain recovered from the feces of a normal calf would stimulate resistance to challenge with SBE virus. Eleven animals were inoculated intraperitoneally with the fecal strain. Two of them were killed for necropsy 7 days post inoculation and serofibrinous peritonitis was present.

Three weeks after the inoculation, three animals were challenged with the same fecal strain and six with SBE virus given intraperitoneally. No lesions were found in the body cavities of the three challenged with the virus from feces and four of those challenged with SBE virus were likewise negative at necropsy. Two of them showed a mild peritoneal reaction.

In a trial with calves, three animals each received two intraperitoneal inoculations of virus strains from feces of normal calves spaced 3 weeks apart. They were challenged with SBE virus 3 weeks after the last injection. Upon necropsy 12 days later, lesions were found involving the peritoneum and pleura which were characteristic of SBE.

SPREAD OF SBE

The manner in which SBE is introduced into a herd and the mode of spread from animal to animal are not well understood. It has been demonstrated that in psittacosis or ornithosis to which SBE is related etiologically, the virus is eliminated in body excretions. Latent infections are of frequent occurrence with psittacosis and these also eliminate virus in the feces. It was logical, therefore, to suspect that similar conditions for spread of SBE occur. In several of the herds in which SBE was diagnosed, there was a history of additions from outside sources within a few months prior to the outbreaks, suggesting that carriers were a possible source of infection.

Search has been made for virus

in nasal excretions, urine, and feces of calves infected by various routes with SBE virus. These excretions were inoculated intraperitoneally into guinea pigs which were examined for lesions after a 6- to 10-day period.

Virus has not been demonstrated in nasal excretions by this procedure during active stages of infection or during convalescence. Virus was demonstrated in one urine specimen obtained 19 days after perorum inoculation of a calf and in another 15 days following intraperitoneal inoculation. The number of examinations of nasal excretions and urine has involved too few animals to properly assess their importance in shedding of virus.

Virus of the psittacosis-lymphogranuloma group has been demonstrated in fecal specimens from 5 of 7 calves that had been inoculated with SBE virus; but 12 of 14 calves not known to have been exposed also eliminated virus in feces. Inoculation of guinea pigs has not provided a suitable means of differentiating between the non-pathogenic and SBE virus.

Only a few of the strains from feces were inoculated into calves. Virus isolates from feces of an animal at 5 and 6 weeks after it had been inoculated with SBE virus were used to expose two calves. When challenged later with SBE virus both were immune. Another calf was inoculated with virus from feces of an animal that had shared a pen with a SBE-infected animal for 30 days. This calf was also immune when challenged later. None of the

calves inoculated with virus strains recovered from animals which had not been exposed to SBE resisted challenge SBE virus so that they were presumably not SBE strains.

In addition to excretions, blood and various tissues have been examined for virus by guinea pig inoculation. Blood samples collected during the febrile stage from six calves experimentally infected showed presence of virus, but no virus was recovered from blood during convalescent stages. Virus has been recovered irregularly from liver, spleen, kidney, and bile specimens collected from animals sacrificed during convalescent stages.

DIAGNOSIS

Only a tentative diagnosis of SBE can be made by clinical signs. A slight stiffness and possibly lessened appetite from 1 to 5 days may constitute the only outward symptoms, but a febrile reaction should be detected if SBE is responsible. Such mild cases are often not brought to the attention of veterinarians unless there are other animals exhibiting more marked symptoms.

Stiffness, incoordination, "knuckling" at the fetlocks when forced to move, or being down and requiring assistance to rise are signs indicating a possibility of SBE. Cerebral symptoms such as circling, pushing, or aggressiveness are generally absent in SBE. An animal may be unable to stand but still will eat and drink. A history of new cases in a 2- or 3-week period showing similar symptoms further supports a tentative diagnosis of SBE.

The diagnosis of SBE can be confirmed on post mortem examination if generalized serofibrinous peritonitis and pleuritis are found without complicating pneumonic or abdominal visceral lesions.

In the laboratory, intraperitoneal inoculation of guinea pigs with suspensions of liver, spleen, or brain tissue is a useful procedure for demonstrating the presence of SBE virus. Elementary bodies may be demonstrated in smears of exudate stained by Macchiavello's method, although they are never numerous.

Complement fixation tests with serums from recovered cases and antigen prepared from SBE or related viruses have value in identifying the disease with the psittacosis-lymphogranuloma group.

TREATMENT

Veterinarians have reported disappointing results in treatment of animals affected with SBE. Unfortunately the infection is often well advanced when the diagnosis is made so that any treatment is less effective than if initiated in early stages. It is difficult to evaluate treatment in SBE, as recovery may be expected in some animals that appear hopeless.

No studies on treatment of field or experimentally produced cases have been made at this laboratory. Results of antibiotic susceptibility trials indicated that chlortetracycline and oxytetracycline might have therapeutic value. Price and Hardy (16) found several additional antibiotics exerted activity against the virus and cite favorable

clinical response when erythromycin or when tetracycline followed by penicillin were used intramuscularly in affected calves. The body temperatures returned to normal within 12 hours and recovery was apparently complete within 10 days.

Although transmission of SBE has not been successful in stable and pen exposure trials, isolation of sick animals is recommended as a preventive measure in infected herds.

SUMMARY

Sporadic bovine encephalomyelitis is a specific infectious disease caused by a virus of the psittacosis-lymphogranuloma group. In addition to encephalomyelitis, serofibrinous serositis affecting peritoneal, pleural, and pericardial surfaces is a constant pathological finding and pathognomonic of SBE.

SBE has shown an average morbidity of about 12 percent with an average mortality of nearly 50 percent of affected animals. Cattle of 1 year or less of age are more susceptible and mortality is greatest in calves. The disease has not been recognized in other farm animals.

Truly sporadic in occurrence, SBE has been diagnosed in 24 herds over a 12-year period at this laboratory. According to available literature, SBE has been recognized in 15 states in this country and in Japan. It is probable that the infection has wider distribution than these recorded occurrences indicate, for some cases can easily escape detection because of mild clinical signs. Although not proven, unapparent infections may occur in

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cattle. There is limited evidence that virus may be eliminated in feces of animals for several weeks after the infection.

The viral agent is of relatively large size. Elementary bodies have been demonstrated in exudates. The virus produces infection in guinea pigs causing peritonitis and sometimes pleuritis on intraperitoneal inoculation. It adapts to growth in chicken embryos with enlargement of the yolk sac and death of the embryo. The virus has remained viable in frozen and lyophilized states.

A viral agent of the psittacosis-lymphogranuloma group, which appears to be a separate virus from that of SBE, can be recovered from

the feces of cattle that are apparently normal.

Inoculated into guinea pigs it produced lesions indistinguishable from SBE and stimulated partial resistance to SBE challenge. In cattle it did not stimulate sufficient resistance to protect against SBE. A vaccine prepared of formalinized SBE virus did not provide protection in guinea pigs.

Treatment of SBE has received but limited experimental study. On the basis of antibiotic sensitivity studies, so-called "broad spectrum" antibiotics such as chlortetracycline, oxytetracycline, tetracycline, erythromycin, and carbomycin offer the most promise as therapeutic agents.

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