A SURVEY OF CENTRAL NERVOUS SYSTEM DISEASES OF MICHIGAN CATTLE

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ABSTRACT

A SURVEY OF CENTRAL NERVOUS SYSTEM

DISEASES OF MICHIGAN CATTLE

By

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Diseases which affected the central nervous system of Michigan cattle during a 7-year period were surveyed. Seasonal, age, breed and sex relationships, the pathologic changes and the pathogenic mechanisms of the conditions encountered were discussed. Those diseases occurring most frequently were listeriosis, polioencephalomalacia (PEM), infectious thromboembolic meningoencephalitis (TEME) and lead poisoning.

Listeriosis occurred most frequently in Holstein female cattle over 2 years of age. It was most prevalent in the first quarter of the year and was associated with feeding silage. *Listeria monocytogenes* has been proven the causative agent. Microscopically there were lesions of limited distribution consisting of microabscessation, glial nodules and perivascular cuffing with lymphocytes.

Polioencephalomalacia occurred most frequently in Holstein calves 0 to 6 months of age. It was most prevalent in the first quarter of the year. The occurrence of this disease was associated with stress and a high dietary intake of stored foods. Treatment of affected animals has implicated thiamine in the pathogenic mechanism. Microscopically there was a laminar necrosis of the cortical gray matter.

Infectious thromboembolic meningoencephalitis occurred most frequently in steer calves of beef breeds 6 to 12 months of age. It was most prevalent in the fourth quarter of the year and was associated with stress and possible carrier animals. *Hemophilus somnus* has been proven the causative agent. Microscopically, foci of thrombosis and vasculitis with an accompanying response were found randomly throughout the brain and meninges.

Lead poisoning occurred most frequently in Holstein female cattle. There were 2 high risk age groups, the very young and older animals. It was more prevalent in the second and third quarters of the year and was associated with pasture conditions where access to lead compounds was enhanced. Microscopic lesions were nonspecific.

Encephalitis due to *Escherichia coli* septicemia, rabies, malignant catarrhal fever, and a group of miscellaneous encephalitides were observed but were of low frequency.

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INTRODUCTION

Bovine encephalitides in Michigan have become increasingly important during recent years. Feedlots and dairy farms have encountered epizootics of central nervous system diseases which have perplexed the owner and the veterinarian alike. The clinical signs of these diseases are often so similar that it makes a positive clinical diagnosis difficult. The diagnostician approaches the problem of a bovine with central nervous signs in terms of a differential diagnosis. In addition to the clinical signs, the gross and microscopic lesions and bacteriologic and virologic determinations are paramount in arriving at a definitive diagnosis.

It is expected that encephalitides will continue as important diseases and economic factors in feedlots and dairy operations. This research will attempt to bring together the present knowledge of these various conditions as it relates to pathology. Additional information will be important in the diagnosis, treatment and control of these and the identification of new diseases.

OBJECTIVES

The objective of this research was to survey the central nervous system (CNS) diseases of cattle as they have occurred in Michigan during the preceding 7 years.

The specific aims were:

1. To establish seasonal, age, breed and sex incidences of specific diseases.

2. To review the pathologic changes and the current concepts of pathogenesis of each disease.

3. To establish a routine which will predicate an accurate diagnosis of CNS disease.

4. To compile the information contained in the past records and present it as useful and meaningful data.

LITERATURE REVIEW

There are many conditions which affect the CNS of cattle. Some are neurotrophic and selectively attack neural tissue producing pathologic changes limited to the brain and spinal cord. Others have no predilection for nervous tissue but produce CNS lesions and clinical signs by their action on the adventitial cells of the blood vessels of the brain and meninges. Some diseases result in CNS lesions because of their septicemic nature, the primary infective site being in other organ systems. Some conditions are metabolic in nature, a deficiency of an essential ingredient or the presence of a histiotoxic substance resulting in a degenerative change.

The CNS is enclosed in unyielding bony cavities and is additionally encompassed by the dense fibrous dura mater. It is composed primarily of nerve-cell bodies, their dentritic and axonal fibers and the supporting neuroglial cells. It has limited numbers of adventitial cells and perivascular fibroblasts which accompany the blood vessels supplying the parenchyma. Because of these physical restrictions the CNS is limited in the ways it can respond to toxins, deprivations and infectious agents. The resulting similarity of clinical signs and neuropathologic lesions emphasizes the importance of a gross and microscopic examination of the CNS in the differential diagnosis of these conditions.

This literature review will pertain only to those diseases which produce CNS lesions in the cattle of Michigan. It is not intended to cover the total manifestations of each disease process but to review briefly the history, the classical gross and microscopic changes in the CNS and the theories of the pathogenic mechanism of these diseases.

Listeriosis

Listeriosis was first described by Murray *et al.* (1926). They isolated the organism from the liver of sick rabbits and guinea pigs and after observing large numbers of monocytes in the peripheral blood of infected rabbits named it *Bacterium monocytogenes*. Pirie (1927), in South Africa, isolated an identical bacterium from the liver of gerbils dying from a plague-like disease. He named the organism *Listerella hepatolytica*, choosing the generic name to honor Lord Lister, a pioneer in the field of bacteriology. The 2 genus and species names were later combined to *Listeria monocytogenes*.

Gill (1931), in New Zealand, was credited with the first isolation of *L. monocytogenes* from farm animals. He observed the disease in sheep which he called "circling disease", a name still often applied to listeric encephalitis of ruminants.

In the United States the disease and lesions were first described in sheep by Doyle (1932). The infection in cattle was first diagnosed by Jones and Little (1934) in New Jersey. Fincher (1935) reported the infection in New York cattle and Olafson (1940) reported listeriosis in New York sheep. Since that time the disease has been widely reported throughout the United States and in foreign countries.

Listeriosis has been listed as an emerging disease by the World Health Organization of the United Nations. Three international symposia on listeric infections have been held to exchange information on the

increasing disease problem. Gray and Killinger (1966) reviewed world literature on *L. monocytogenes* and listeric infections and cited many references on history, the organism and the infection in man, animals, fowl and fish.

Olafson (1940) observed that outbreaks of listeriosis had a seasonal incidence, the peak of infection occurring February through May. Feeding of silage was associated with these outbreaks. Olafson noted that in some cases the disease abated following removal of silage from the ration. Gray (1960) isolated *L. monocytogenes*, of the same type, from the brain stem of affected sheep and from the silage consumed by those sheep. Palsson (1963) reported that in a 5-year period in Iceland listeriosis occurred in 177 of 307 flocks of sheep fed grass silage; it occurred in only 5 of 347 flocks not fed silage. Jensen and Mackey (1949) investigated 20 infected farms for recurrence of the disease. Nine of those experienced infection in new animals the following year. Seasonal, age, breed and sex incidence were not given. Gibbons (1963) stated that *L. monocytogenes* was distributed widely among wild rodents and that carrier birds or animals may be the source of the organism in contaminated silage.

Jubb and Kennedy (1970) and Jensen and Mackey (1971) listed 3 clinical manifestations of listeric infections; they were: 1) encephalitis or neurolisteriosis with clinical signs of disturbance in the central nervous system, 2) gastrointestinal septicemia with lesions in many visceral organs, including liver, spleen and lungs, and 3) placentitis with abortion commonly occurring during the second half of gestation.

Cordy and Osebold (1959), in a study of natural cases of listeriosis, suggested that encephalitis occurs by way of a hematogenous route, the

bacteria lodging in the reticular formation of the brain stem with lesions developing in the midbrain, pons and medulla with subsequent extension to the meninges, ependyma and occasionally the eye. Asahi *et al.* (1957) and Borman *et al.* (1960) demonstrated that the organism could ascend the trigeminal and facial nerve trunks to produce a meningoencephalitis with lesions confined to the brain stem in the region of the respective nerve nuclei, trunks and branches. It was assumed that the organism reached peripheral branches of the nerves by way of wounds in the oral or nasal mucosa. This indicated that the encephalitic form developed as a local infection.

Jubb and Kennedy (1970) and Smith *et al.* (1972) stated that gross neuropathologic lesions usually were not discernible. Occasionally the meninges were thickened and grayish foci of malacia were found in cross sections of the medulla. Histopathologic changes were limited to the brain stem and consisted of foci of gliosis, microabscessation and lymphocytic perivascular cuffing and meningitis.

Polioencephalomalacia

Polioencephalomalacia (PEM) in cattle was first reported by Jensen *et al.* (1956) in Colorado and Wyoming. The exact cause of the condition was unknown. Discounting the opinion of Wyoming workers that PEM was due to chronic selenium poisoning, the authors suggested the possibility of some unknown intoxication as the cause. The reported incidence was higher in female Hereford cattle which predominated that area and was higher in the 12- to 18-month age group. Polioencephalomalacia occurred more frequently during January among feedlot cattle and more frequently during July among pastured cattle. The neuropathologic lesions observed were located in the cerebral cortex and in the granular

layer of the cerebellum. Specific sulci and adjacent gyri of the cerebral cortex were studied. Gross lesions consisted of multiple yellowish foci of necrosis and separation of affected from viable tissue. Microscopically there were necrosis of neurons, proliferation of endothelial cells and accumulation of phagocytic cells with gliosis at the junction of necrotic and viable tissue. Variable amounts of necrosis in the granular layer of the cerebellum were also observed.

Terlecki and Markson (1959) described a similar condition in Britain which they called cerebro-cortical necrosis (CCN). Later (1961) they published a detailed description of the condition. The exact cause of CCN was unknown but the authors noted that the cerebral necrosis resembled the ischemic-anoxic type. The reported incidence in Britain was higher in cattle in the 3- to 6-month age group and occurred most frequently during the winter months. Cerebro-cortical necrosis was seen in all local cattle breeds and their crosses without apparent sex bias and under different systems of management and feeding. The neuropathologic lesions were located in the cerebral cortex and cerebellum. Gross lesions consisted of a bilateral yellowish discoloration of the cerebral gyri especially those in the dorsolateral regions. In some cases a distinct line of separation was evident between the cortical gray matter and the subjacent white matter. Microscopically, there were laminar necrosis of the cortical gray matter, capillary proliferation and infiltration of microglial cells especially at the periphery of the lesions. Cerebellar lesions consisted of necrosis of the Purkinje cells and moderate to severe rarefaction of the granular layer.

The pathologic terms polioencephalomalacia and cerebro-cortical necrosis, though originating from different geographic areas, apparently

designate the same disease. Until the cause is determined, Jensen and Mackey (1971) suggested naming the condition polioencephalomalacia, a descriptive term denoting necrosis of the gray matter of the brain.

Little and Sorensen (1969) reported on PEM in Minnesota. They reported a seasonal prevalence in January, February and March and that calves 3 to 5 months of age were most commonly affected. The feeding of grain, especially moldy cob corn, appeared to be associated with the disease.

Davies *et al.* (1965) observed field cases which recovered after treatment with large doses of thiamine and theorized that PEM was caused by an acute deficiency of thiamine.

Although largely empirical and clinical, Spencer (1969), Jarret (1970), Herrick (1971) and Jensen and Mackey (1971) suggested supporting evidence for a thiamine deficiency as the causative mechanism of PEM. Thiamine has a central role in metabolizing carbohydrates; a deficiency of thiamine results in high blood accumulation of pyruvate and lactate and theoretically could cause cerebral edema resulting in ischemia and neuronal necrosis. The role of thiamine in the mechanism of early brain edema and swelling as it relates to the pathogenesis of PEM remains unknown.

Courville (1958) studied human cases involving acute to chronic cerebral anoxia and concluded that the essential cause of laminar necrosis of the cerebral cortex was a lack of oxygen. He also concluded that this lesion would develop under the appropriate circumstances regardless of the precise mechanism by which the amount of oxygen reaching the affected area was reduced.

Edwin *et al.* (1968) and Edwin *et al.* (1968) reported an increased thiaminase activity in the contents of the digestive tract of PEM affected ruminants.

Loew *et al.* (1970) described an unidentified thiamine destroying activity in rumen contents of PEM affected cattle in Canada. Edwin and Jackman (1970) tentatively identified the enzyme thiaminase I as the causative agent.

Davies *et al.* (1968) isolated a thiaminase producing fungus, *Acrosperia macrosporoides*, from moldy straw which was being consumed by PEM affected calves. The possibility of subclinical PEM was suggested. Loew *et al.* (1970) further supported this theory by demonstrating thiamine destroying activity in rumen contents of apparently normal cattle.

Loew and Dunlop (1972) found that very large doses of thiaminase compounds were needed to experimentally produce PEM but they were unable to demonstrate increased quantities of these in the gastrointestinal contents or tissues of naturally occurring cases, Loew *et al.* (1972) concluded that if destruction of thiamine in the gastrointestinal tract was of significance in PEM, then it was more probable that it was due to bacterial action rather than to thiaminase.

Jubb and Kennedy (1970) stated that PEM lesions originate as a circulatory disturbance. The areas of involvement had a distribution related to the field of supply of the middle cerebral artery and, to a lesser extent, the posterior cerebral artery supplying the cerebellum.

Infectious Thromboembolic Meningoencephalitis

Infectious thromboembolic meningoencephalitis (TEME) of cattle was first reported by Griner *et al.* (1956) in Colorado. The cause was thought to be secondary involvement of the brain and meninges resulting

from a wide variety of primary disease conditions. The disease was described as a random dissemination of infected emboli in the blood vessels of the brain and its meninges. Multiple macroscopic and microscopic foci of inflammation and liquefactive necrosis from localization of infectious emboli were described. Hereford cattle 1 to 2 years of age were most commonly affected but they were the predominant breed and age in that area.

Kennedy *et al.* (1960) described an epizootic of bacterial encephalitis in a California feedlot which began in October and extended through February. The diagnostic gross lesions were confined to the brain, where single or multiple hemorrhagic areas of malacia were described. Microscopically, these lesions had evidence of a bacterial vasculitis which led to thrombosis and infarction. The disease was experimentally reproduced using an isolate recovered from animals having characteristic lesions. The organism was identified as a *Hemophilus*-like bacterium.

Weide *et al.* (1964), reporting on encephalitic diseases found in Kansas feedlots, stated that a condition characterized as infectious embolic meningoencephalitis was the most common encountered. Streptococci and staphylococci were isolated, and the condition was again suggested to occur associated with bacterial infections in other parts of the body. The sex ratio of the disease was approximately 1:3 in favor of heifer calves. The significance of this was not determined. Most affected cattle weighed 750 to 900 pounds. The disease was not observed in adult or non-feedlot cattle.

Bailie *et al.* (1966) reported isolation of organisms similar to those isolated by Kennedy but suggested that the organisms were *Actinobacillus actinoides*-like. The condition was primarily found in

feedlot cattle weighing 750 to 1100 pounds and 1 to 3 years of age. Breed susceptibility was not observed. Fifty-two heifers and 26 steers were affected.

Shigidi and Hoerlein (1970) of Colorado and Bailie (1969) in Kansas studied the bacteriologic properties of the etiologic agent. Bailie suggested the name *Hemophilus somnus* for the organism.

Panciera *et al.* (1968), after observation of field cases in Oklahoma and Texas, called attention to the acute respiratory manifestations and chronic joint and muscle involvement in cattle infected with *H. somnus*. Most episodes of disease occurred in cattle confined to feedlots but cattle pastured on winter wheat were occasionally affected. The latter usually became ill following assemblage and transport to wheat pasture.

Dillman (1969) reported a polypoid tracheitis syndrome occurring in chronically coughing feedlot cattle which was associated with chronic *H. somnus* and *Pasteurella multocida* infection.

Olander *et al.* (1970), observing TEME in Indiana feedlots, reported a seasonal incidence during October through January. They suggested the possibility of inapparent carrier animals being responsible for spreading the disease.

Brown *et al.* (1970) outlined the epidemiology and pathogenesis, gross and histopathologic lesions, serologic procedures and the differential diagnosis of the *H. somnus* complex.

Lead Encephalopathy

Lead poisoning has been recognized in most domesticated animal species and in man. Reports of naturally and experimentally induced lead toxicosis are many; those in animals have been reviewed by McIntosh (1956). Buck (1970) stated that lead is one of the most

common causes of poisoning in livestock and companion animals in the United States.

While most authors referred to the clinical manifestations of lead toxicosis, very few reports described the neuropathologic lesions. Kradel *et al.* (1965) were first to attribute specific brain lesions to lead poisoning. They reported a laminar cortical necrosis of the cerebral cortex with endothelial proliferation, malacia and an eosinophilic meningeal infiltration. Little and Sorensen (1969) described necrosis located mainly in the superficial laminae of the cerebral cortex. These were usually small and focal but could form a laminar lesion. They were among the first to describe differences in the neuropathology of PEM, TEME and acute lead poisoning. Jubb and Kennedy (1970) and Smith *et al.* (1972) also gave reference to CNS lesions of lead toxicosis. These included cerebral edema and laminar cortical necrosis of the ischemia-anoxic type.

Christian and Tryphonas (1971) gave a detailed description of the gross and microscopic brain lesions of naturally and experimentally produced lead poisoning in cattle. They described a symmetrical laminar cortical necrosis selectively localizing at the tips of the gyri. They attributed the basic pathogenic mechanism of lead toxicosis to dysoria, an increased permeability of the blood-brain barrier. Lead created a state of prolonged metabolic dysoxidosis presumably by interfering with porphyrin metabolism. This affected the permeability of capillaries, resulting in dysoric encephalopathy. Dilatation of the capillary bed, activation and proliferation of endothelial cells, swollen astrocytes, hemorrhage, transudation and formation of cavities with preservation of neurons in affected areas were interpreted as being the results of altered capillary permeability.

Allcroft and Blaxter (1950) found that 0.2 to 0.4 gm./kg. of lead as the acetate, carbonate or oxide was lethal for calves up to 4 months old. Older animals were slightly more tolerant. Metallic lead was determined by Allcroft (1950) to be an unlikely cause of poisoning. He gave a calf 400 gm. of lead shot over a period of 4 months without producing evidence of ill effects. Eighty-four percent of the lead shot was recovered at slaughter 5 months after the last dose was given.

Encephalitis due to *Escherichia coli* Septicemia

Calf scours is a complex disease, but the primary infective agent has been generally considered to be *Escherichia coli*. Reisinger (1965) suggested the possibility of a synergistic action between a virus and *E. coli*. The relative importance of virus in this disease has not been fully assessed. A similar disease was reported by McBryde (1934) in suckling pigs, by Marsh and Tunnicliff (1938) in lambs, and by Dimock *et al.* (1947) in foals. There was no reported evidence of natural interspecies transmission of this disease.

Calf scours has been a common problem during the first few days of life and has been frequently fatal. Reisinger (1965) attributed 90 percent of all dairy calf diarrhea mortalities to this disease complex. Ensminger *et al.* (1955) ranked calf scours second in overall importance, without regard to ages, among beef cattle diseases. Blood and Henderson (1968) stated that the incidence of this disease decreased as husbandry methods were intensified. Oxender *et al.* (1973) stated that 2 periods, birth and the first 14 days of life, were critical to successful calf raising. These 2 periods accounted for the majority of calf mortalities on Michigan dairy farms.

Bacterial invasion to cause calf scours was usually related to some upset in the physiological equilibrium of the calf. Blood and Henderson (1968), Oxender *et al.* (1973) and Speicher and Hepp (1973) listed the following important factors in dairy calf mortality: the stress of crowding, poor sanitation, substitute diets, chilling, malnutrition of the dam, and failure of the calf to ingest colostrum. Blood and Henderson (1968) stated that the disease was most prevalent during the winter months and, when established in a group of calves, animal passage enhanced the virulence of the infection. Stress, investigated by Lee and Phillips (1948) and Moll (1965), may be the "triggering" mechanism to initiate the disease. Whether stress "triggers" a susceptibility to a specific pathogenic serotype or merely causes debilitation that allows the normal *E. coli* flora of the alimentary tract to become pathogenic needs more investigation.

The gross lesions of calf scours were minimal in comparison with the pathophysiological manifestations in the live calf. McSherry and Grinyer (1954) attributed much of the clinical picture to electrolyte and acid-base imbalances. Smith *et al.* (1972) stated that lesions observed in calves dying of acute colibacillosis were composed of a mucoid enterocolitis that may contain flecks of blood, edematous mesenteric lymph nodes, and an acute synovitis in one or more joints. Cachexia and dehydration were marked. Calves that had been sick for several days may have shown secondary pathology due to systemic dissemination of infection. Fincher (1963) and Moll and Brandly (1955) have shown that bronchopneumonia, purulent arthritis, omphalitis, purulent otitis media and meningoencephalitis may occur. *Escherichia coli* may be cultured from the blood and major body organs as well as from the alimentary tract.

Rabies

Rabies is a viral encephalitis which has been recognized since ancient times. There were many reports concerning this disease, but it was not the intent of this thesis to review all of its various aspects. It is important to note that the classical work of Pasteur, cited by Merchant and Packer (1961), which showed that the virus could be modified so that it would produce immunity without the hazard of producing the disease, was one of the milestones in medicine. Merchant and Packer (1961) reported that the rabies virus was neurotropic and establishment of infection depended on inoculation of the virus into a wound either by biting or contamination with infected saliva. Johnson (1965) demonstrated that the virus traveled from the site of infection to the CNS by way of the peripheral nerves.

A report from the National Research Council, Subcommittee on Rabies (1973) stated that the rabies virus was pathogenic for all mammals and that the virus was currently classified as a member of the rhabdovirus group.

Babes (1892) described focal proliferations of glial cells occurring in rabies. These changes, later called Babes nodules, were a fairly constant finding but were not considered to be specific. Negri (1903) described spherical, intracytoplasmic inclusion bodies having specific tinctorial characteristics. These inclusions, now called Negri bodies, have since been considered pathognomonic of the disease. Jubb and Kennedy (1970) stated that Negri bodies were found most commonly in the neurons of the hippocampus in carnivores and in the Purkinje cells of the cerebellum in herbivores.

Lapi *et al.* (1952) reported that specific lesions of rabies developed earlier and more consistently in the gasserian ganglion

than elsewhere in the nervous system and were sufficiently characteristic to permit a presumptive diagnosis when Negri bodies were not demonstrated. These lesions consisted of focal proliferations of Schwann cells surrounding ganglion neurons, mild infiltrations of lymphocytes and plasma cells and proliferation of Babes nodules.

Jubb and Kennedy (1970) reported that there were no gross lesions present in cases of rabies. Microscopic lesions consisted of inflammatory and degenerative changes in the pons, hypothalamus and cervical spinal cord. Perivascular cuffing with lymphocytes, focal gliosis (Babes nodules) and the presence of Negri bodies within the cytoplasm of neurons were typically found.

The diagnosis of rabies in the absence of demonstrable Negri bodies has been aided by a mouse inoculation technique reported by Tierkel (1959). The development of the fluorescent antibody technique for the diagnosis of rabies as reported by McQueen (1959) further improved the accuracy of diagnostic procedures and reduced the time necessary to report the status of a suspected animal brain.

Malignant Catarrhal Fever

Hutyra *et al.* (1949) referred to the observation and description of malignant catarrhal fever (MCF) as early as the eighteenth century. They stated that it was sporadic and world-wide in occurrence, had a high mortality rate, and, although transmissible, was not contagious.

Blood and Henderson (1968) stated that malignant catarrhal fever was caused by a nonfilterable herpesvirus which could be experimentally transmitted only by blood transfusion or node-to-node transfer of lymphoid tissue. This suggested that the virus was firmly attached to the leukocytes. The mode of natural transmission has not been discovered.

Jubb and Kennedy (1970) suggested ingestion as the probable route along with a possibility of arthropod vectors. Sheep have served as natural reservoirs to cattle. This finding was substantiated by Berkman *et al.* (1960) in Michigan.

Blood and Henderson (1968) stated that clinically the disease is characterized by a catarrhal mucopurulent inflammation of the upper respiratory and alimentary epithelia, keratoconjunctivitis, encephalitis, rapid dehydration and enlargement of the lymph nodes. This clinical picture has sometimes been divided into 4 syndromes: the peracute, the intestinal, the head and eye, and the mild form, the head and eye syndrome being the typical field observation.

Jubb and Kennedy (1970) stated that malignant catarrhal fever has 2 pathognomonic lesions which serve to differentiate it from similar diseases. These were: 1) vascular lesions consisting of a fibrinoid necrotizing vasculitis with cellular accumulations in the adventitia and 2) destruction of lymphoid tissue with a concomitant proliferation of primitive lymphoreticular and connective tissue cells. These vascular and lymphoid lesions constituted the fundamental pathologic changes in all tissues and were the basis of all other described lesions. Meningeal plasma exudation was also a characteristic finding.

Miscellaneous Encephalitides

In addition to those conditions already reviewed, many others can also involve the CNS. Jubb and Kennedy (1970) listed the following main divisions of CNS disease: congenital abnormalities, increased intracranial pressure, cerebral swelling and edema, lesions of blood vessels and circulatory disturbances, traumatic injuries, degenerative lesions, metabolic nutritional and toxic lesions, inflammatory lesions (pyogenic, viral and parasitic) and neoplastic disease.

Miscellaneous conditions from each of these divisions were occasionally encountered. Because of their low frequency a literature review was not done.

Michigan Cattle Population

To understand any predilection for season, age, breed or sex of these various diseases some information is needed on the breakdown of Michigan's total cattle population and common management practices of Michigan farmers.

Cattle, by classes, on Michigan farms are given in Table 1. The 1972 figures indicated that 42.2% of the cattle population were dairy cows and replacement heifers; 11.7% were beef cows and replacement heifers; 19.2% were cattle on feed for market; 25.4% were calves weighing less than 500 pounds; and 1.5% were bulls.

Research Report 183 (1972) from the Michigan State University Agricultural Experiment Station gave this account of the dairy cow numbers in Michigan. In 1966 Grade A producers accounted for 53% of the dairy herds and 75% of the dairy cows. By 1971 these percentages had increased to 70% Grade A producers and 88% of the dairy cows. Table 2 gives the percentage distribution of Grade A herds by breed in 1968. These figures indicate that the Holstein breed comprised the bulk of Michigan's cattle population.

Since Michigan has been predominantly a dairy state, the marketing of milk and milk products, from a financial point of view, has been of the greatest importance to the majority of Michigan farmers. The amount of milk produced in most milk sheds has been greater during the spring months and lower during the fall months. In order to compensate the dairyman who maintained a high fall production when milk was needed,

Table 1. Livestock on farms, by classes, Michigan - January 1, 1965-1972

Species and Class	1965	1966	1967	1968	1969	1970	1971	1972
Cattle and calves*				Thousands				
Classification by sex and age								
Milk animals:								
Cows, 2 years and over	653	614	558	525	509	509	---	---
Heifers, 1-2 years	186	166	153	145	139	135	---	---
Heifer calves	189	176	168	160	152	151	---	---
Beef animals:								
Cows, 2 years and over	136	136	122	116	119	123	---	---
Heifers, 1-2 years	78	78	63	67	68	70	---	---
Other calves	304	280	259	242	244	254	---	---
Steers, 1 year plus	204	195	190	183	192	209	---	---
Bulls, 1 year plus	19	18	17	16	16	17	---	---
Classification by sex and weight								
Cows and heifers that have calved	---	---	---	---	---	587	599	611
Beef cows	---	---	---	---	---	118	137	138
Milk cows	---	---	---	---	---	469	462	473
Heifers, 500 pounds and over	---	---	---	---	---	268	273	282
For beef cow replacement	---	---	---	---	---	40	42	43
For milk cow replacement	---	---	---	---	---	169	169	177
Other heifers	---	---	---	---	---	59	62	62
Steers, 500 pounds +	---	---	---	---	---	218	240	234
Bulls, 500 pounds +	---	---	---	---	---	21	22	23
Calves, less than 500 pounds	---	---	---	---	---	374	393	392
All cattle and calves	1,769	1,663	1,530	1,454	1,439	1,468	1,527	1,542

*Classification by sex and age discontinued in 1971; classification by sex and weight started in 1970.

Source: Michigan Agricultural Statistics, MCRS, July, 1972.

Table 2. Percentage distribution of Grade A herds by breeds, 1968

Breed	State Total (%)
Holstein	86.8
Guernsey	2.5
Jersey	1.5
Brown Swiss	.4
Mixed and other	8.8
Total	100.0

Source: Research Report 96, Michigan State University Agricultural Experiment Station, January, 1970.

Michigan markets have operated on the base-surplus plan for paying for fluid milk. The base period was established during the lower production months, August through December. A dairyman's base payment was established by the average amount of milk produced during the base period. This encouraged the dairyman to produce a more uniform supply throughout the year. Milk produced in excess of the allotted base amount was paid for on the surplus milk value which was lower. In practice the dairyman would attempt to have the majority of his cows freshen during the base period to ensure his income and to keep from receiving a lower price should he produce a surplus of milk while not in the base period. To accomplish this a dairyman would group heifers for breeding and attempt to rebreed some cows to have them freshen during the base period. Speicher (1973) found that 66.3% of the dairy calves were born during the base period in quarters 3 and 4.

Of the 19.2% of the total cattle population which were on feed for market, many were imported from other states. Michigan health regulations governing admission of livestock for feeding and grazing required that animals under 18 months of age be accompanied by an official interstate health certificate or permit and be placed under feeder quarantine separate and apart from dairy and breeding cattle until tested for brucellosis and tuberculosis or slaughtered. Table 3 gives inshipments of feeder cattle during 1967-1971 indicating a definite rise in feeder cattle imports in April and again from August through December.

Table 3. Inshipments of feeder cattle, Michigan, 1967-1971

Month	Feeder Cattle				
	1967	1968	1969	1970	1971
January	4,592	4,737	5,325	5,746	4,367
February	4,394	3,470	3,862	4,772	3,760
March	4,887	3,566	7,733	5,111	3,931
April	4,352	5,727	8,335	7,818	6,495
May	4,658	5,719	3,612	8,307	3,908
June	4,973	5,260	7,324	5,160	5,524
July	4,047	6,132	4,782	4,325	3,983
August	10,710	9,244	8,442	9,025	7,733
September	11,403	14,198	9,394	11,771	7,531
October	14,776	17,903	15,794	10,024	12,174
November	15,096	19,132	23,697	17,385	10,356
December	7,318	14,539	10,959	12,258	3,497
Year	91,206	109,627	109,259	101,702	73,259

Source: Michigan Agricultural Statistics, MCRS, July, 1972.

MATERIALS AND METHODS

The CNS cases for this study were submitted to the Department of Pathology, Michigan State University, over a 7-year period from January 1, 1966, to January 1, 1973. These cases were submitted by the Department of Large Animal Surgery and Medicine and by practicing veterinarians.

The tissues were routinely fixed in 10% buffered formalin, trimmed and processed in an Autotechnicon* and embedded in Paraplast.** Sections were cut at 6 microns and stained with hematoxylin and eosin in the manner described in the *Manual of Histologic and Special Staining Technics* (1968).

Tissues from suspected cases of poisoning were submitted to the Michigan Department of Agriculture, Laboratory Division, for toxicologic analysis. Brain tissue from suspected cases of rabies was submitted to the Michigan Department of Public Health, Division of Communicable Disease, for fluorescent antibody and mouse inoculation tests.

The data utilized were retrieved by the Veterinary Report Generating System from pathology case reports maintained on the CDC 6500 computer system at Michigan State University. The computer was asked

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*Technicon Company, Chauncey, N.Y.

**Aloe Scientific Division of Brunswick, St. Louis, Mo.

to retrieve all cases of central nervous disease in the bovine. Conditions in which CNS lesions may not have been the outstanding feature were asked for individually.

Utilizing information on the computer print-out, the stained microscope slides of each case were found in the slide library maintained by the Department of Pathology, Michigan State University. Each case was examined microscopically and the diagnosis was compared to that in the original pathologist's report.

RESULTS

Listeriosis

A majority of the cases of listeriosis occurred during the winter months (Table 4). Listeriosis was most common in female cattle over one year of age (Tables 5 and 6). Breed incidence is given in Table 7.

Gross lesions were rarely observed in listeriosis. Occasionally there was evidence of meningitis in the region of the brain stem. In one chronic case grayish foci of malacia were found in cross sections of the medulla and cervical spinal cord.

Microscopic lesions were consistent in their anatomical distribution being limited to the brain stem and anterior cervical spinal cord. These lesions consisted of microabscessation, glial proliferation, perivascular accumulation of lymphocytes and a lymphocytic leptomeningitis (Figures 1 and 2).

The microabscesses varied from small foci of malacia and gliosis, containing a few neutrophils, to larger areas of liquefaction and suppuration. The small microabscess was the consistent finding; in fact several foci of gliosis could be searched before finding neutrophils.

The perivascular cellular accumulations typically were composed of small hyperchromatic lymphocytes and a few neutrophils. Chronic accumulations had larger cell populations and contained small numbers of histiocytes and plasma cells.

Lymphocytes had usually infiltrated the pia mater especially in the area of the brain stem.

Table 4. Listeriosis: Seasonal incidence (1966-1972)

	Cases	Percent
Jan., Feb., March	36	51.4
April, May, June	21	30.0
July, Aug., Sept.	5	7.2
Oct., Nov., Dec.	8	11.4
Total	70	100.0

Table 5. Listeriosis: Sex incidence (1966-1972)

	Cases	Percent
Female	38	54.3
Male	9	12.9
Steer	15	21.4
Undetermined	8	11.4
Total	70	100.0

Table 6. Listeriosis: Age incidence (1966-1972)

	Cases	Percent
0-6 months	1	1.4
6-12 months	7	10.0
1-2 years	18	25.7
2-4 years	5	7.2
over 4 years	20	28.6
Undetermined	19	27.1
Total	70	100.0

Table 7. Listeriosis: Breed incidence (1966-1972)

	Cases	Percent
Holstein	29	41.4
Hereford	15	21.4
Angus	9	12.9
Others	8	11.4
Undetermined	9	12.9
Total	70	100.0

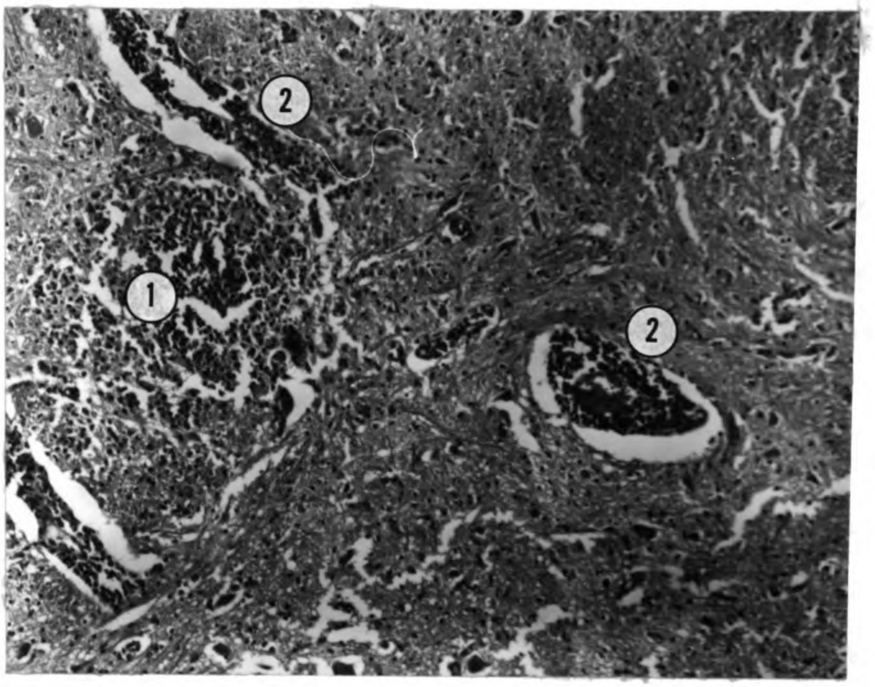

Figure 1. Listeriosis. Medulla oblongata with microabscessation (1) and perivascular accumulation of lymphocytes (2). Hematoxylin and eosin. x 125.

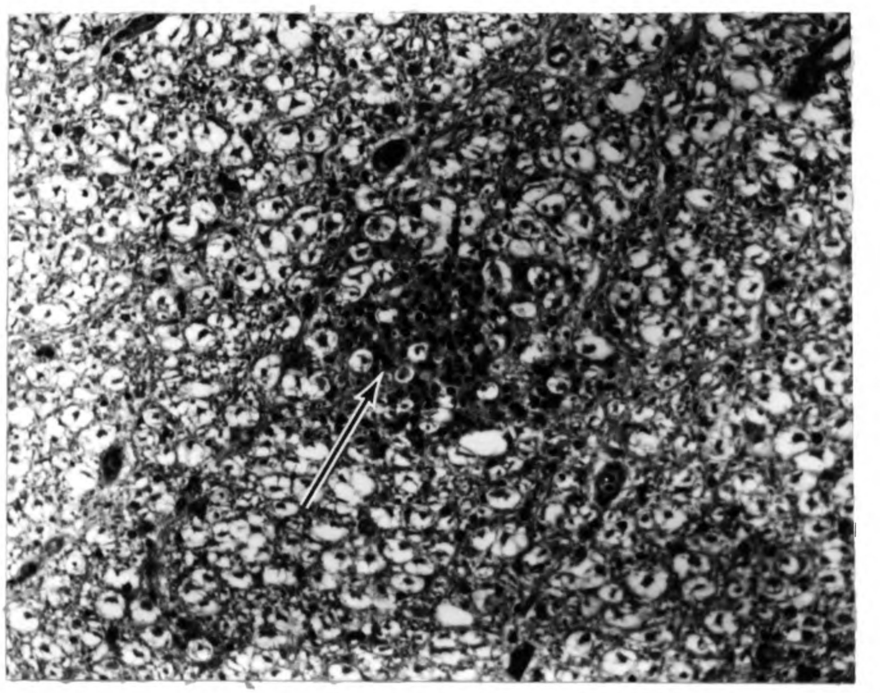

Figure 2. Listeriosis. A microabscess in the anterior cervical spinal cord (arrow). Hematoxylin and eosin. x 250.

Application of appropriate stains would usually demonstrate the presence of gram-positive organisms in the areas of abscessation.

Polioencephalomalacia

The majority of the cases of PEM occurred during the months of January, February and March (Table 8). The highest incidence was in the 0- to 6-month age group of Holstein calves (Tables 9 and 10). Sex incidence is given in Table 11.

The gross lesions observed consisted of a bilateral, somewhat symmetrical, yellowish discoloration of the cerebral cortex limited to the dorsolateral gyri and sulci. In chronic cases the gyri were atrophic (Figure 4). The cut surface often revealed an area of separation of the affected gray matter from the underlying white matter.

Microscopic lesions in acute cases consisted of necrosis and edema of the deeper cerebral laminae (Figures 5 and 6). Neurons in the affected areas were undergoing pyknosis. Satellitosis was increasingly evident with duration of the lesion. Neuronophagia, capillary prominence, macrophage accumulation and gliosis were evident in chronic cases. A distinct demarcation between normal and affected tissue was easily visualized.

There was no observable difference in the severity of involvement between the gyri and sulci, nor was there any observable predilection of the lesions for either of these anatomical locations.

Infectious Thromboembolic Meningoencephalitis

The incidence of TEME was higher in the fall and winter months (Table 12). Six- to twelve-month-old steer calves of beef breeds were most frequently infected (Tables 13 and 14). Breed incidence is given in Table 15.

Table 8. Polioencephalomalacia: Seasonal incidence (1966-1972)

	Cases	Percent
Jan., Feb., March	28	47.5
April, May, June	11	18.6
July, Aug., Sept.	4	6.8
Oct., Nov., Dec.	16	27.1
Total	59	100.0

Table 9. Polioencephalomalacia: Age incidence (1966-1972)

	Cases	Percent
0-6 months	26	44.1
6-12 months	11	18.6
1-2 years	8	13.6
Undetermined	14	23.7
Total	59	100.0

Table 10. Polioencephalomalacia: Breed incidence (1966-1972)

	Cases	Percent
Holstein	33	55.9
Hereford	9	15.2
Angus	7	11.9
Mixed	4	6.8
Other	3	5.1
Undetermined	3	5.1
Total	59	100.0

Table 11. Polioencephalomalacia: Sex incidence (1966-1972)

	Cases	Percent
Female	18	30.5
Male	10	17.0
Steer	17	28.8
Undetermined	14	23.7
Total	59	100.0

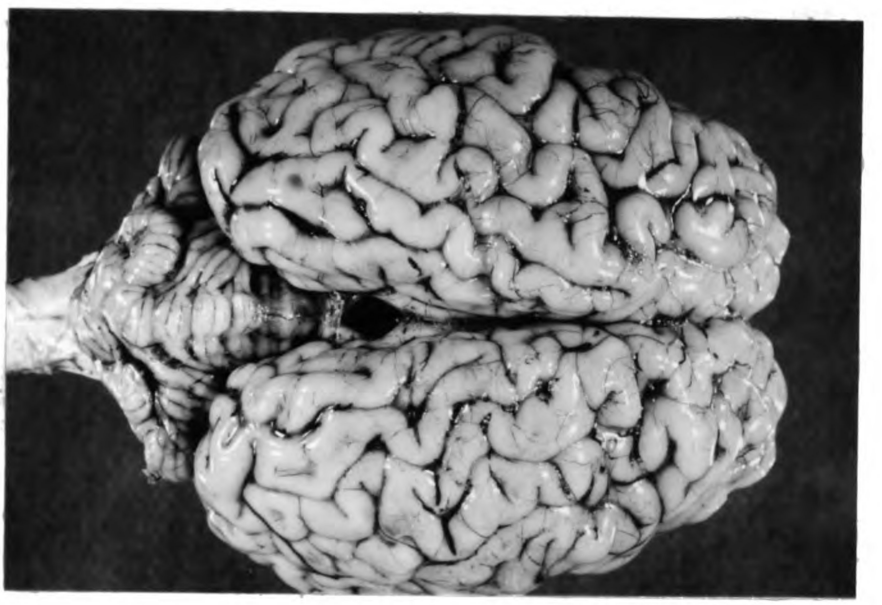

Figure 3. Normal bovine brain.

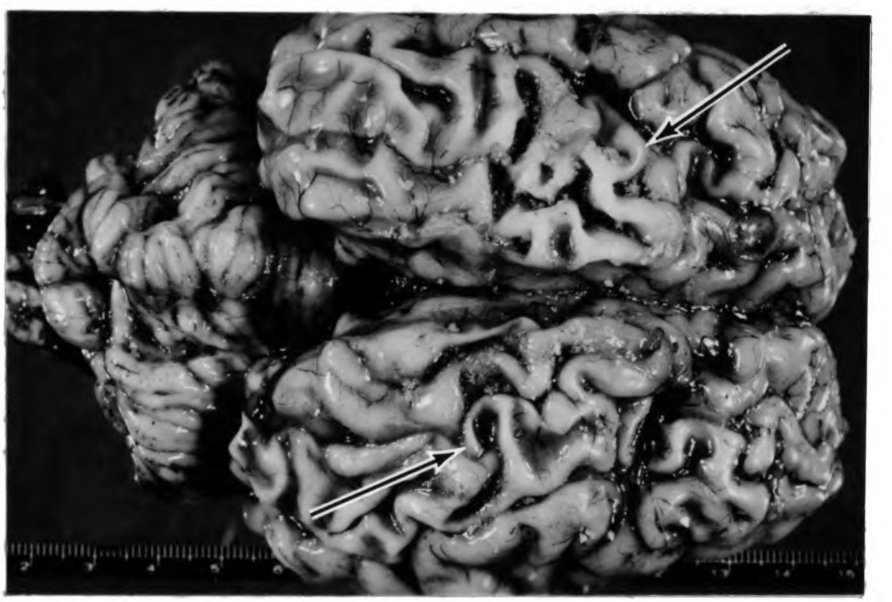

Figure 4. Polioencephalomalacia. Brain from a chronically affected bovine showing atrophy of the cerebral gyri (arrows).

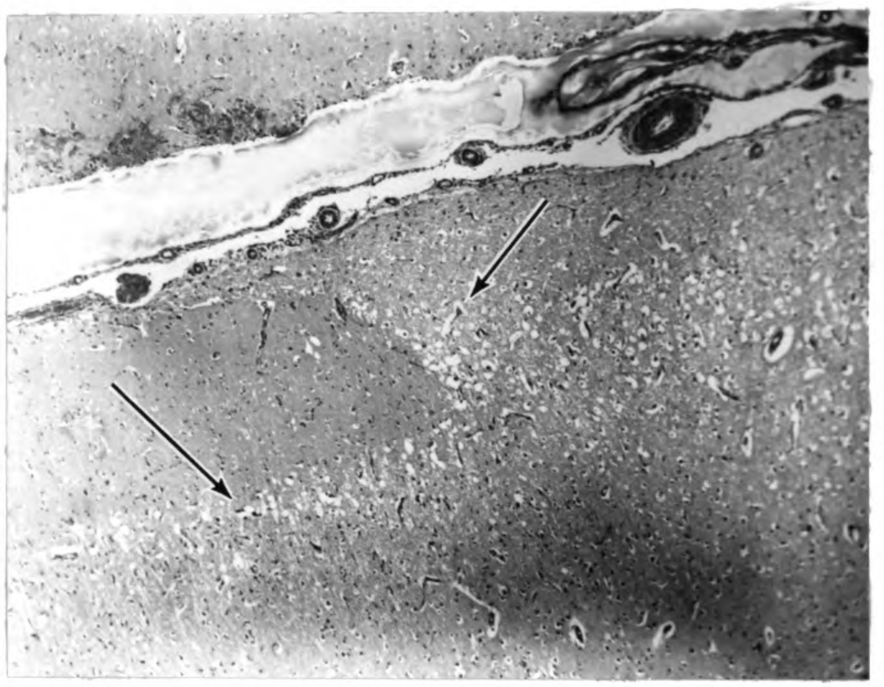

Figure 5. Polioencephalomalacia. Laminar necrosis of the cortical gray matter (arrows). Hematoxylin and eosin. x 50.

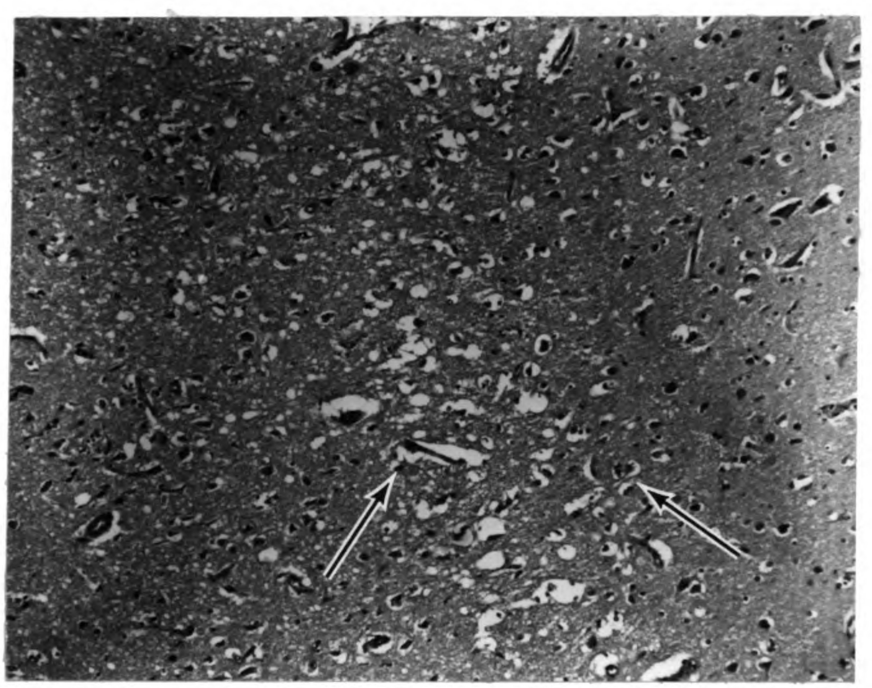

Figure 6. Polioencephalomalacia. Laminar necrosis of the cortical gray matter (arrows). Hematoxylin and eosin. x 125.

Table 12. Infectious thromboembolic meningoencephalitis: Seasonal incidence (1966-1972)

	Cases	Percent
Jan., Feb., March	13	31.7
April, May, June	2	4.9
July, Aug., Sept.	4	9.7
Oct., Nov., Dec.	22	53.7
Total	41	100.0

Table 13. Infectious thromboembolic meningoencephalitis: Age incidence (1966-1972)

	Cases	Percent
0-6 months	3	7.3
6-12 months	22	53.7
1-2 years	5	12.2
2-4 years	1	2.4
Undetermined	10	24.4
Total	41	100.0

Table 14. Infectious thromboembolic meningoencephalitis: Sex incidence (1966-1972)

	Cases	Percent
Female	5	12.2
Male	8	19.5
Steer	24	58.3
Undetermined	4	9.8
Total	41	100.0

Table 15. Infectious thromboembolic meningoencephalitis: Breed incidence (1966-1972)

	Cases	Percent
Holstein	2	4.9
Hereford	11	26.8
Angus	12	29.2
Charolais	7	17.1
Mixed	4	9.8
Undetermined	5	12.2
Total	41	100.0

The gross lesions consisted of multiple necrotic and hemorrhagic foci found in any part of the brain, cervical spinal cord or meninges (Figure 7). Frequently these lesions were seen on the surface of the brain. In transverse sections the brain had similar well defined lesions scattered throughout with variations in size and shape (Figure 8). Some lesions had central liquefaction.

Microscopic lesions were found in various areas of the cerebrum, cerebellum, brain stem and meninges. Thrombosis and vasculitis were consistently associated with areas of suppuration and necrosis (Figure 9). Both capillaries and larger blood vessels were involved. The inflammatory exudate was voluminous and was composed almost entirely of neutrophils. The meninges were often infiltrated with many neutrophils.

Lead Encephalopathy

The majority of cases of lead poisoning occurred from April through September (Table 16). There were 2 high risk age groups, the very young and those over 4 years of age (Table 17). Female Holstein cattle were most commonly involved (Tables 18 and 19).

Specific gross and microscopic lesions of lead encephalopathy were not observed.

Correlation of the 4 Conditions Which were Most Frequently Encountered

Table 20 and Figure 10 give the incidence of listeriosis, PEM, TEME and lead poisoning observed during the survey period. Figures 11, 12, 13 and 14 illustrate the seasonal, age, breed and sex incidences of these 4 conditions.

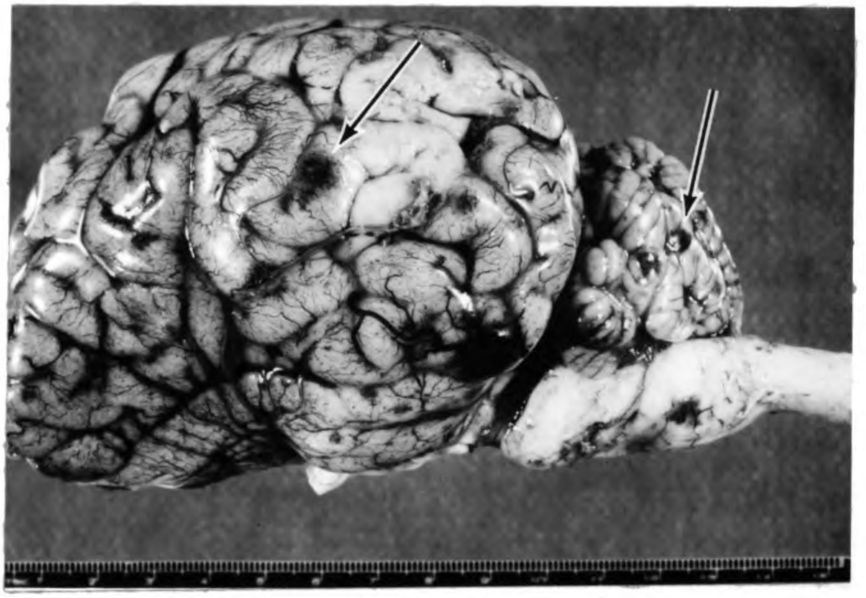

Figure 7. Infectious thromboembolic meningoencephalitis. Brain with multiple hemorrhagic foci (arrows).

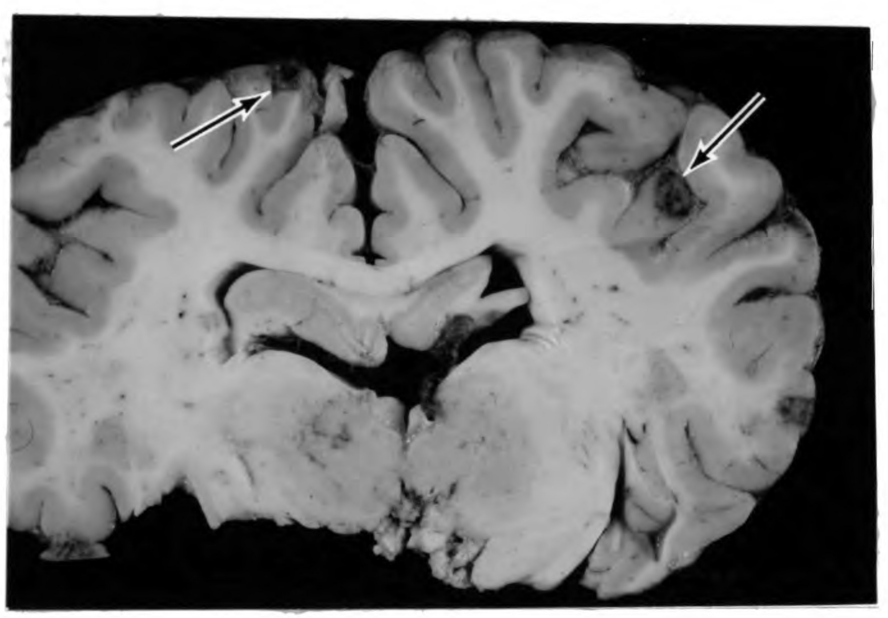

Figure 8. Infectious thromboembolic meningoencephalitis. Cross section of a brain with multiple foci of necrosis and hemorrhage (arrows).

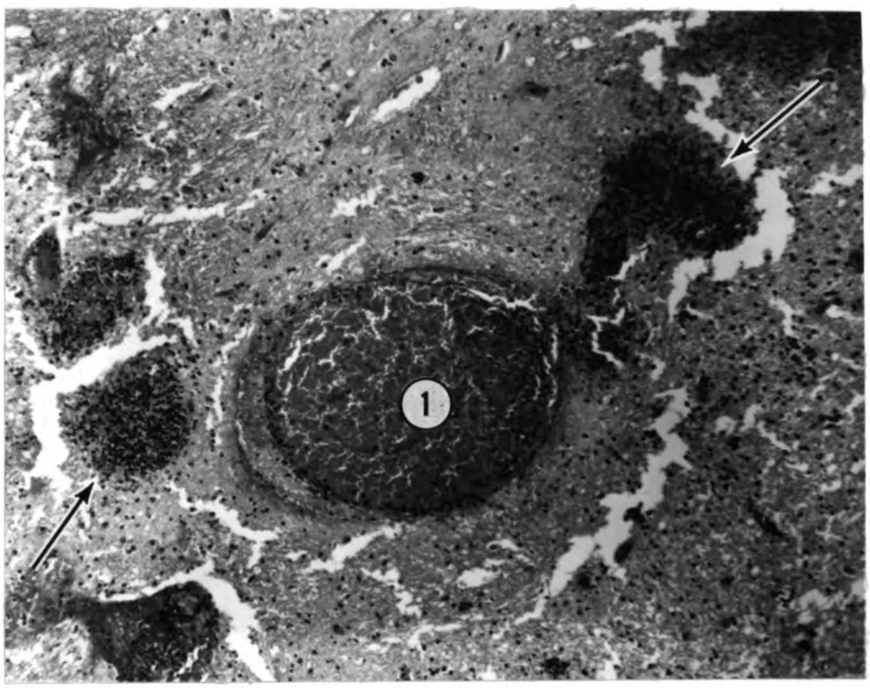

Figure 9. Infectious thromboembolic meningo-encephalitis. Thrombosis of a vein (1) and foci of suppurative encephalitis (arrows). Hematoxylin and eosin. x 50.

Table 16. Lead poisoning: Seasonal incidence (1966-1972)

	Cases	Percent
Jan., Feb., March	5	14.0
April, May, June	12	33.3
July, Aug., Sept.	12	33.3
Oct., Nov., Dec.	7	19.4
Total	36	100.0

Table 17. Lead poisoning: Age incidence (1966-1972)

	Cases	Percent
0-6 months	11	30.6
6-12 months	2	5.6
1-2 years	5	13.9
2-4 years	3	8.3
Over 4 years	10	27.7
Undetermined	5	13.9
Total	36	100.0

Table 18. Lead poisoning: Sex incidence (1966-1972)

	Cases	Percent
Female	23	63.9
Male	4	11.1
Steer	5	13.9
Undetermined	4	11.1
Total	36	100.0

Table 19. Lead poisoning: Breed incidence (1966-1972)

	Cases	Percent
Holstein	23	63.9
Hereford	4	11.1
Angus	3	8.3
Mixed	3	8.3
Other	1	2.8
Undetermined	2	5.6
Total	36	100.0

Table 20. Disease distribution by year (1966-1972)

CNS disease	Year 1966	1967	1968	1969	1970	1971	1972	Total
Listeriosis	16	9	15	9	7	8	6	70
PEM	10	4	8	12	11	6	8	59
TEME	2	2	1	4	12	11	9	41
Lead poisoning	2	4	1	0	5	11	13	36

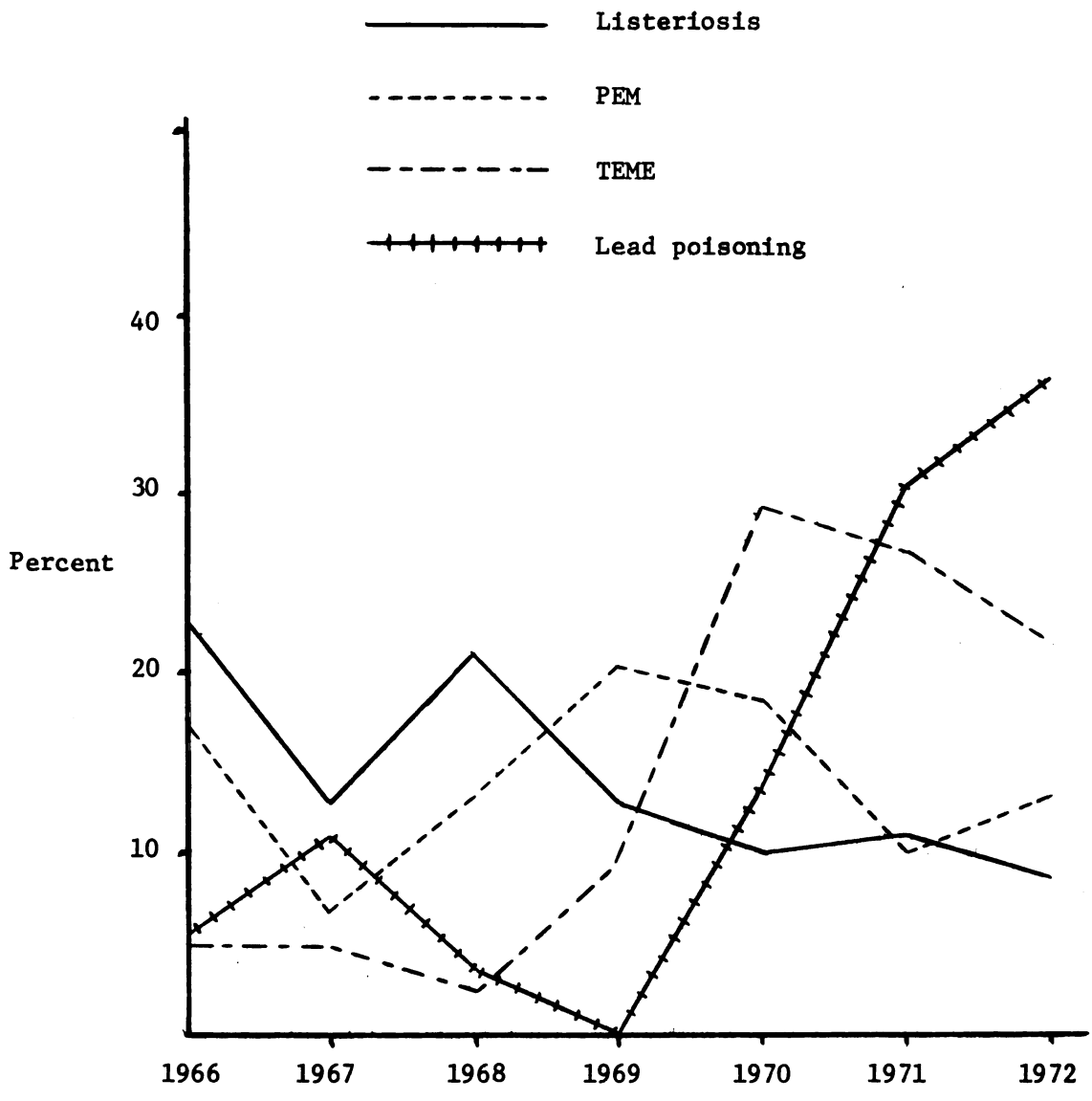

Figure 10. Yearly incidence of central nervous system diseases (1966-1972).

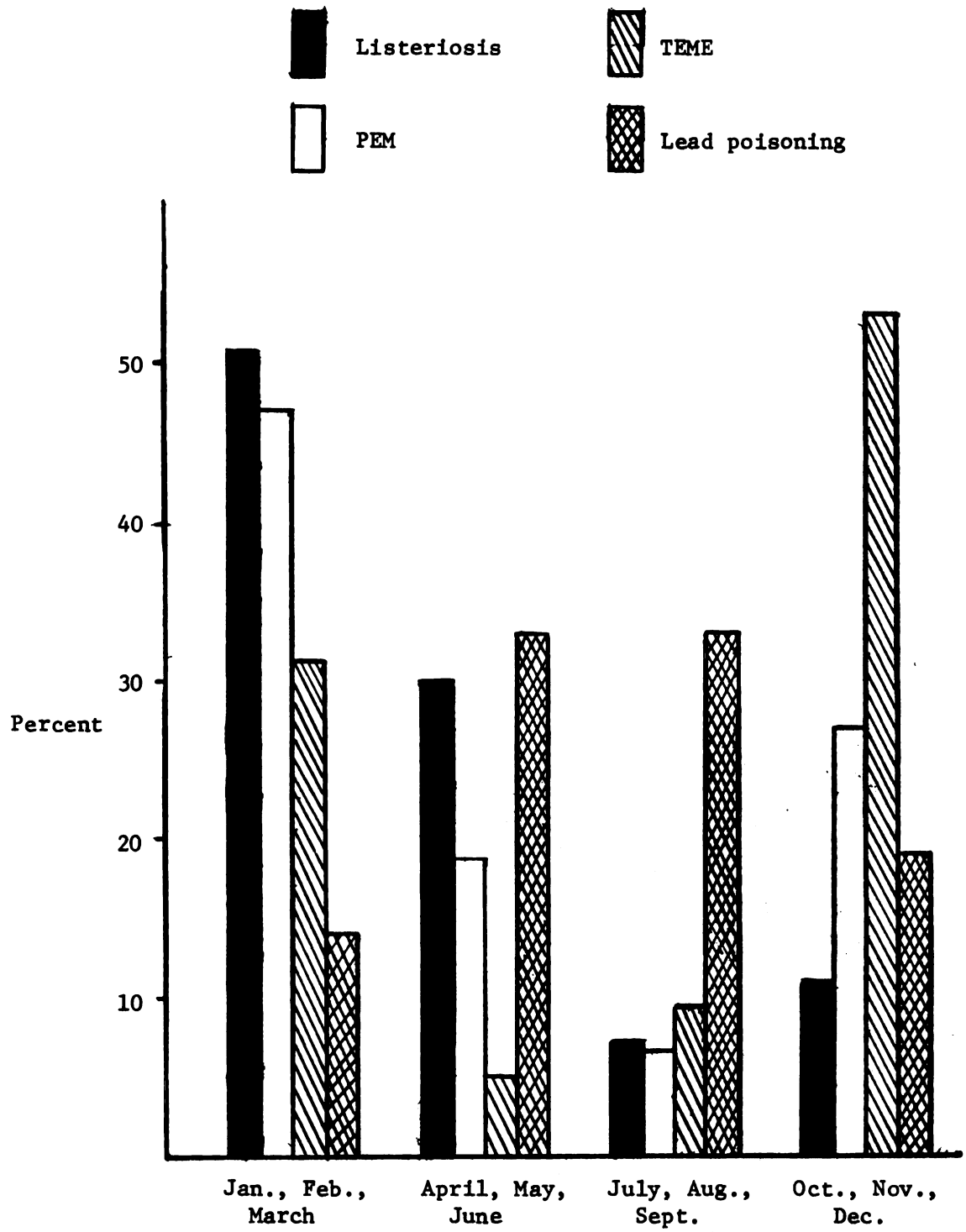

Figure 11. Seasonal incidence of central nervous system diseases (1966-1972).

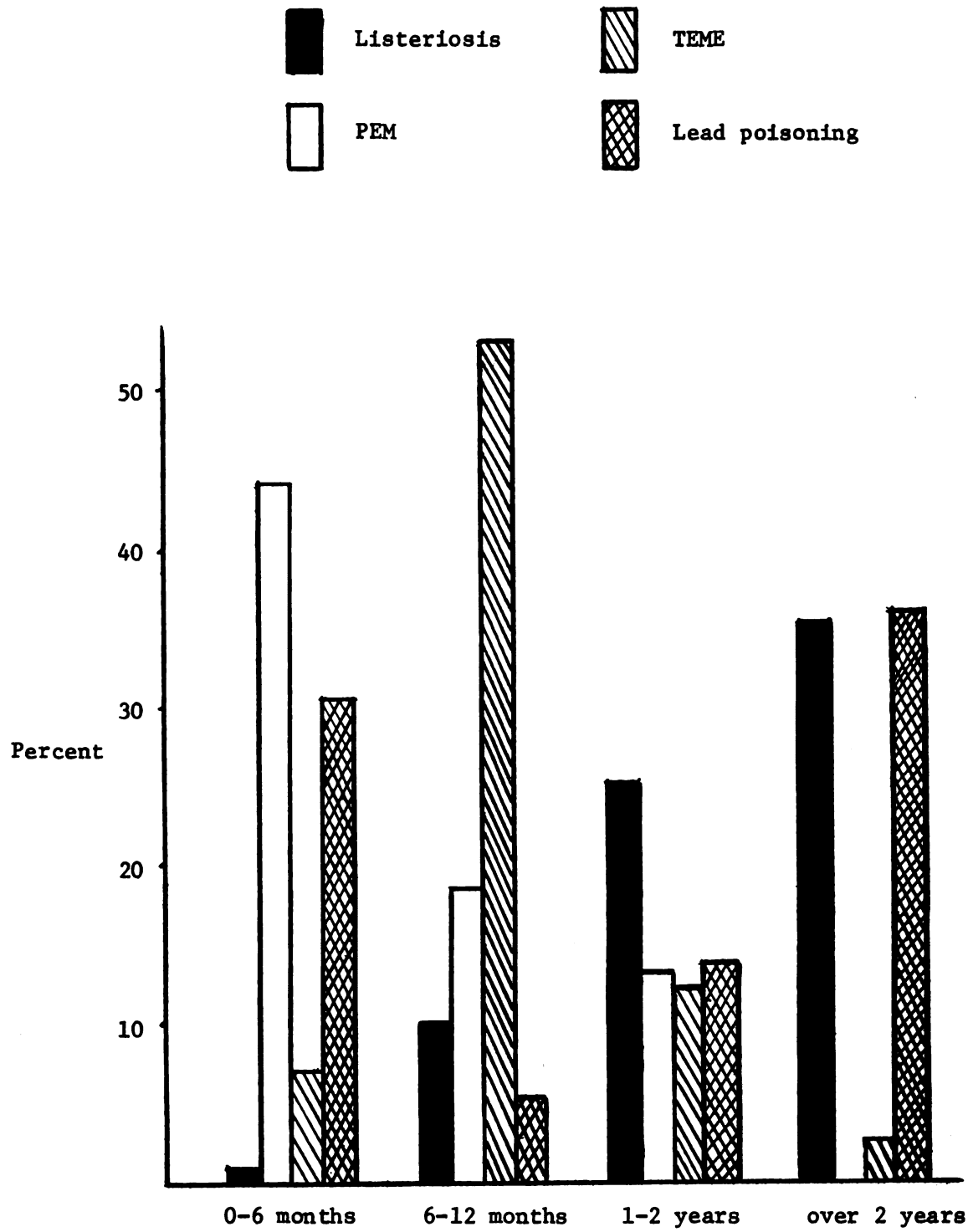

Figure 12. Age incidence of central nervous system diseases (1966-1972).

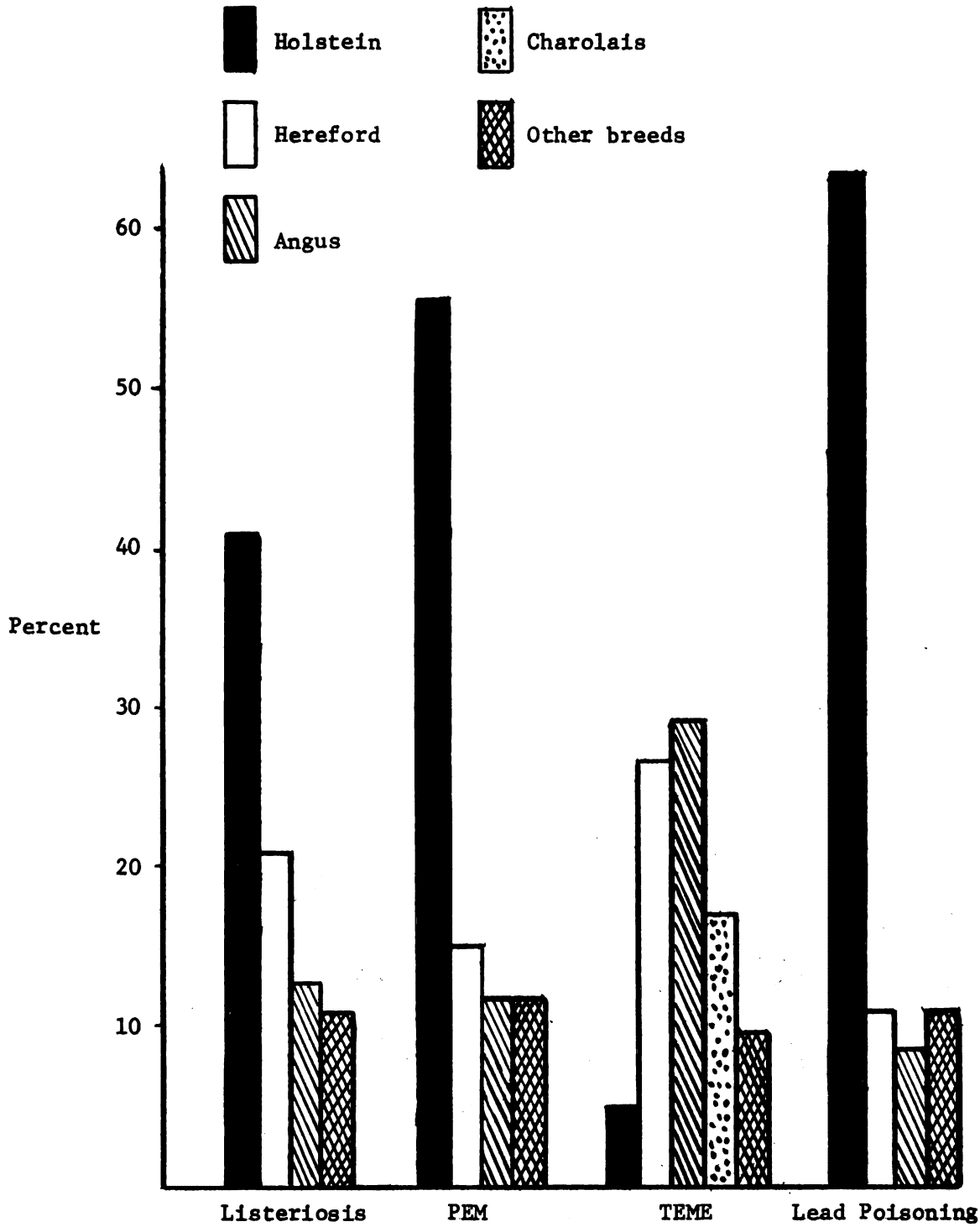

Figure 13. Breed incidence of central nervous system diseases (1966-1972).

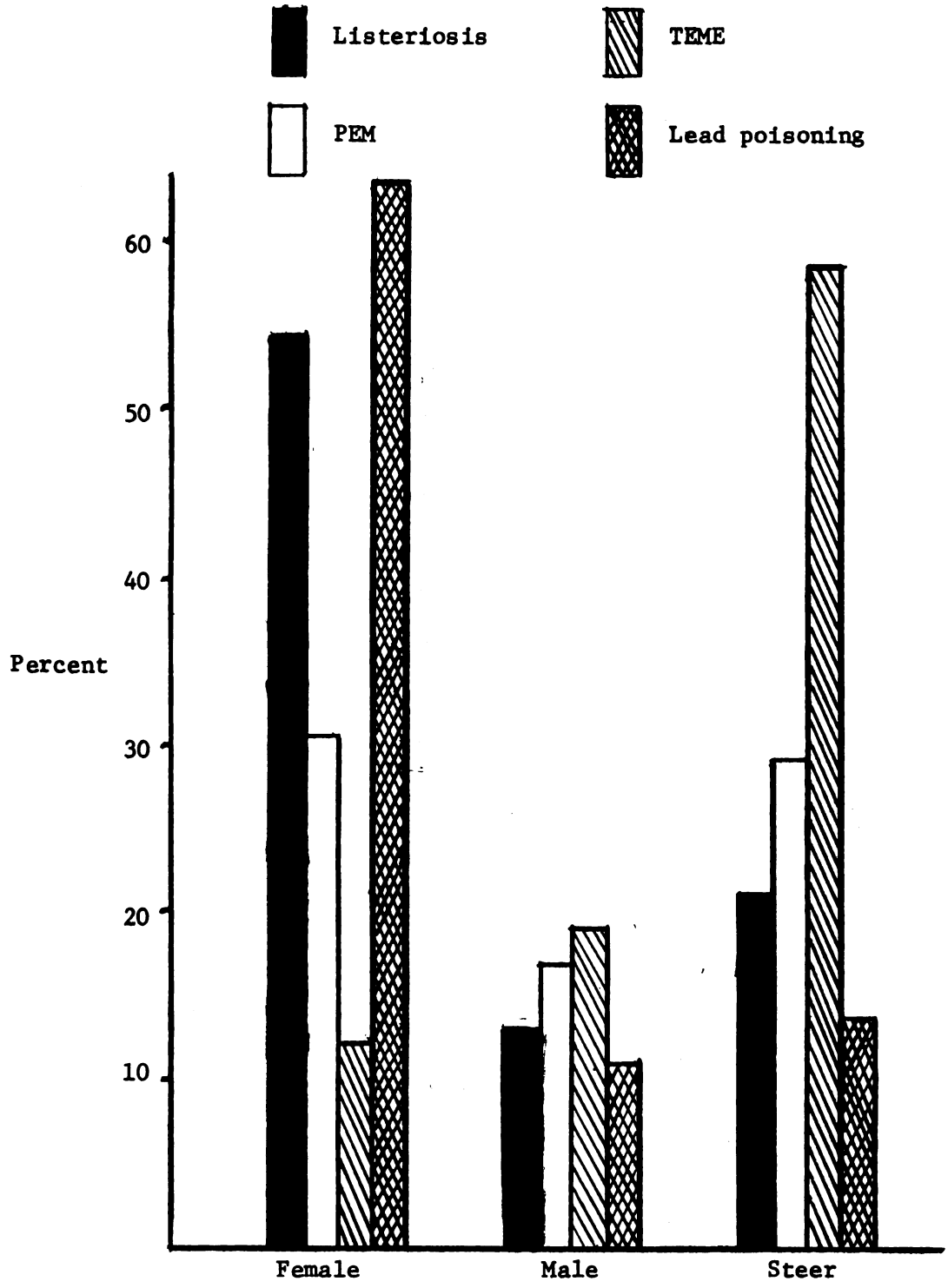

Figure 14. Sex incidence of central nervous system diseases (1966-1972).

Encephalitis Due to *E. coli* Septicemia

The majority of the cases of infectious calf scours occurred during the months of January, February and March (Table 21). The highest incidence was in the 0- to 6-month age group of Holstein calves (Tables 22 and 23). There was no sex bias of the disease (Table 24). In all of these cases *E. coli* was cultured from one or more of the major body organs.

Gross lesions were occasionally observed in the CNS and consisted of meningeal congestion, petechiation and edema. Microscopically a generalized purulent meningoencephalitis was the common finding. Sometimes bacterial emboli were found.

Rabies

Fourteen cases of rabies in Michigan cattle were diagnosed during the preceding 7-year period (Table 25). These data were obtained from Coohon (1973) and were determined by the Michigan Department of Public Health. Two of these cases were initially diagnosed at Michigan State University, both in 1966. The seasonal, age, breed and sex data of cattle infected with rabies were not available for evaluation.

Microscopic examination of the 2 cases available revealed the presence of intracytoplasmic inclusions, Negri bodies, in the neurons of the hippocampus, brain stem, cerebellum, and gasserian ganglion. There were no observable differences in the numbers of inclusions found in the hippocampus and Purkinje cells of the cerebellum. The inclusions in the gasserian ganglion were larger than those found in other areas. Lymphocytic perivascular accumulations were common throughout the brain (Figure 15). There was a lymphocytic meningitis in the area of the cerebellum. Glial proliferations or Babes nodules were

Table 21. Encephalitis due to *E. coli* septicemia: Seasonal incidence (1966-1972)

	Cases	Percent
Jan., Feb., March	8	38.1
April, May, June	4	19.0
July, Aug., Sept.	6	28.6
Oct., Nov., Dec.	3	14.3
Total	21	100.0

Table 22. Encephalitis due to *E. coli* septicemia: Age incidence (1966-1972)

	Cases	Percent
0-6 months	19	90.4
6-12 months	1	4.8
1-2 years	0	0
2-4 years	1	4.8
Total	21	100.0

Table 23. Encephalitis due to *E. coli* septicemia: Breed incidence (1966-1972)

	Cases	Percent
Holstein	16	76.1
Hereford	3	14.3
Angus	1	4.8
Mixed	1	4.8
Total	21	100.0

Table 24. Encephalitis due to *E. coli* septicemia: Sex incidence (1966-1972)

	Cases	Percent
Female	7	33.3
Male	6	28.6
Steer	0	0
Undetermined	8	38.1
Total	21	100.0

Table 25. Rabies incidence

Year	Cases
1966	3
1967	0
1968	0
1969	2
1970	3
1971	5
1972	1
Total	14

Source: Michigan Department of Public Health.

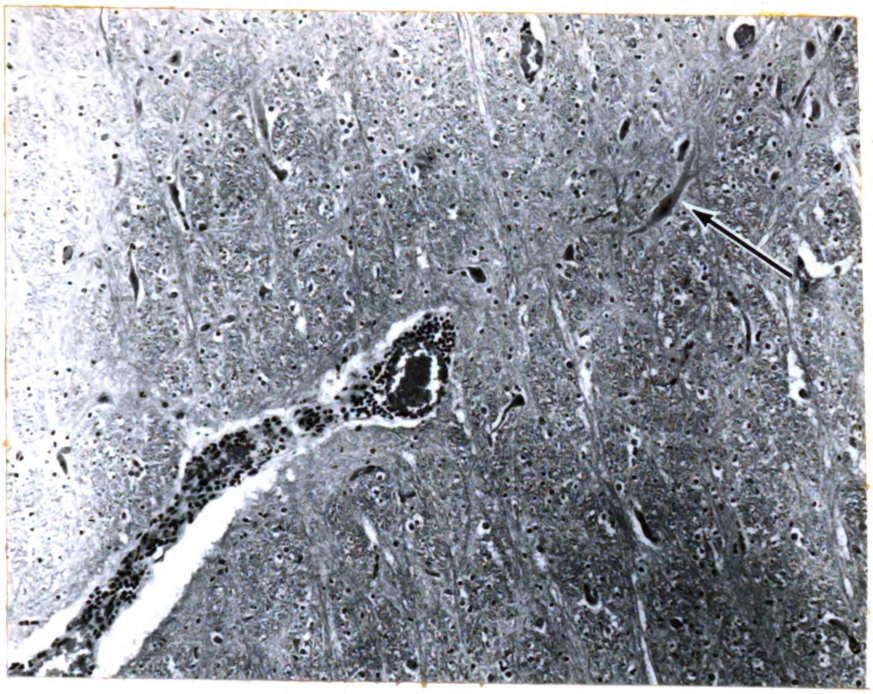

Figure 15. Rabies. Cerebrum with perivascular accumulation of lymphocytes and a neuron with an intracytoplasmic Negri body (arrow). Hematoxylin and eosin. x 125.

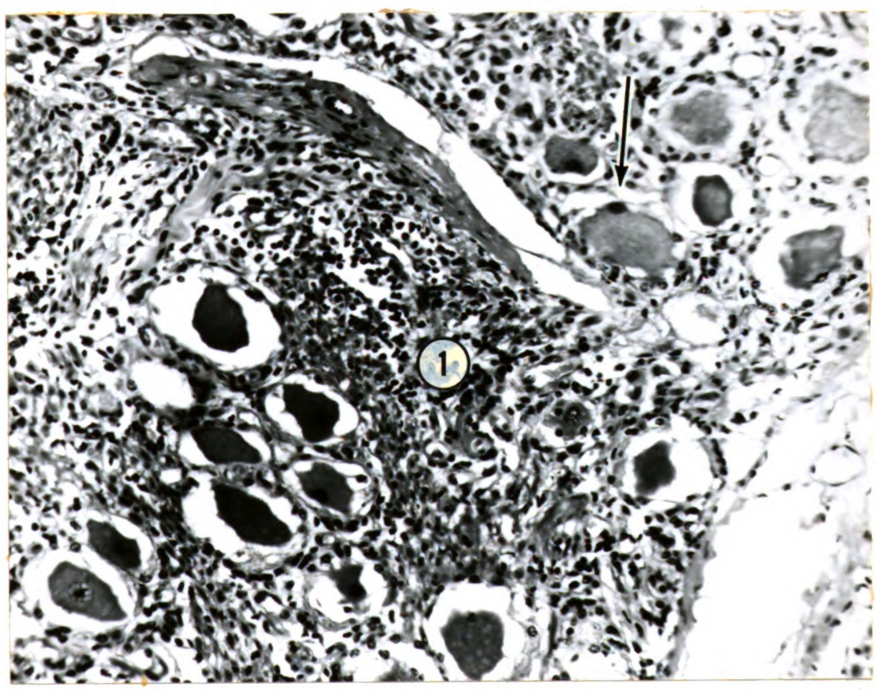

Figure 16. Rabies. Gasserian ganglion containing proliferating capsule cells (1) and an intracytoplasmic Negri body (arrow). Hematoxylin and eosin. x 250.

difficult to find except in the gasserian ganglion, where changes similar to those reported by Lapi *et al.* (1952) were found (Figure 16).

Malignant Catarrhal Fever

The majority of the cases of MCF occurred during the summer months from April through September (Table 26). Holstein female cattle were most commonly infected (Tables 27 and 28). There was no age bias of this disease (Table 29).

Gross CNS lesions were not seen. Microscopic lesions observed within the CNS consisted of a lymphocytic meningitis and a vasculitis. Occasional vessels within the cerebrum exhibited vasculitis with pyknosis of endothelial nuclei and small perivascular accumulations of lymphocytes (Figures 17 and 18). Meningeal plasma exudations were observed in one case.

Miscellaneous Encephalitides

Table 30 gives the miscellaneous encephalitides diagnosed during this period.

Of the cases of suppurative encephalitis which were cultured, the following organisms were isolated: *Corynebacterium pyogenes* (4 cases), *Pseudomonas aeruginosa* (2 cases), *Staphylococcus aureus* (1 case), *Salmonella sp.* (1 case), and *Pasteurella multocida* (1 case). *Clostridium perfringens* was cultured from 2 cases of focal symmetrical leukomalacia.

Table 26. Malignant catarrhal fever: Seasonal incidence (1966-1972)

	Cases	Percent
Jan., Feb., March	2	15.4
April, May, June	5	38.4
July, Aug., Sept.	4	30.8
Oct., Nov., Dec.	2	15.4
Total	13	100.0

Table 27. Malignant catarrhal fever: Breed incidence (1966-1972)

	Cases	Percent
Holstein	6	46.1
Brown Swiss	4	30.8
Mixed	2	15.4
Undetermined	1	7.7
Total	13	100.0

Table 28. Malignant catarrhal fever: Sex incidence (1966-1972)

	Cases	Percent
Female	9	69.2
Male	2	15.4
Steer	0	0
Undetermined	2	15.4
Total	13	100.0

Table 29. Malignant catarrhal fever: Age incidence (1966-1972)

	Cases	Percent
0-6 months	2	15.4
6-12 months	2	15.4
1-2 years	3	23.1
2-4 years	3	23.1
Over 4 years	1	7.7
Undetermined	2	15.3
Total	13	100.0

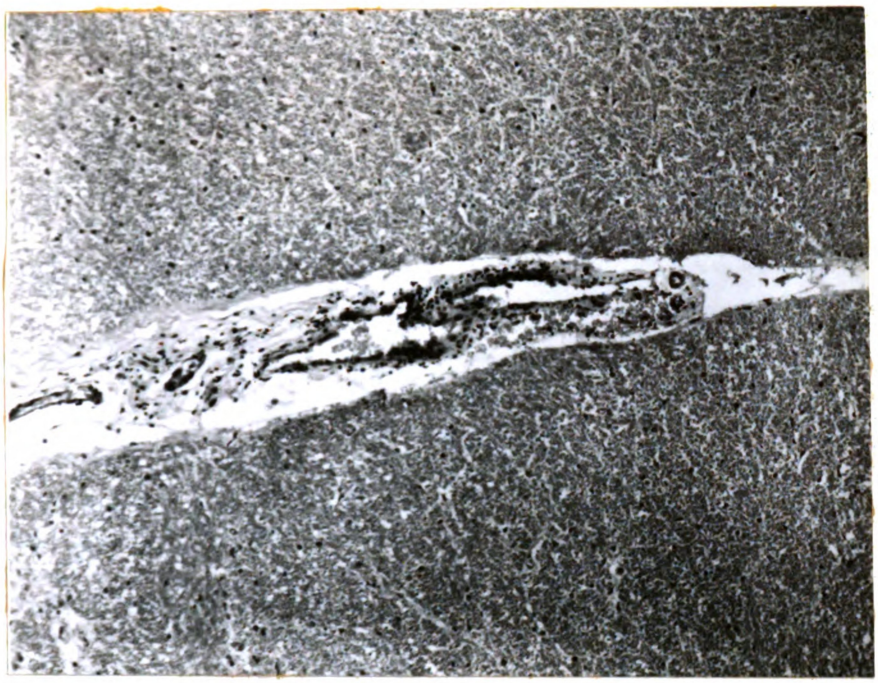

Figure 17. Malignant catarrhal fever. Necrotizing vasculitis with cellular accumulation in the adventitia. Hematoxylin and eosin. x 125.

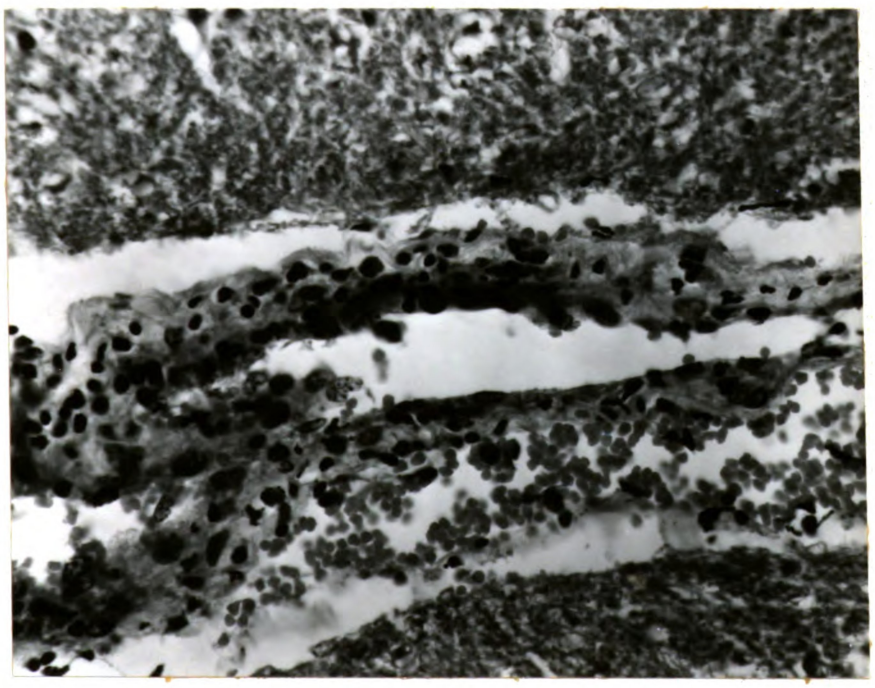

Figure 18. Malignant catarrhal fever. Necrotizing vasculitis with cellular accumulation in the adventitia. Hematoxylin and eosin. x 500.

Table 30. Miscellaneous encephalitides (1966-1972)

Disease	Cases
Suppurative encephalitis	45
Hemorrhagic encephalitis	8
Trauma	7
Cerebellar hypoplasia	6
Congenital hydrocephalus	5
Nonsuppurative encephalitis	4
Focal symmetrical leukomalacia	4
Congenital anomalies	3
Parasitic encephalitis	1
Arteriosclerosis	1
Mercury poisoning	1
Arsenic poisoning	1
BVD	1
Necrosis of granular layer of cerebellum	1
Neurofibrosarcoma	1
Astrocytoma	1

DISCUSSION

Listeriosis

The seasonal incidence of listeriosis was similar to other areas within the Northern Hemisphere and generally occurred from late November to early May and was most prevalent during January, February, March and April. Animals of both sexes and all ages and breeds may be affected, but it was most common in animals over 1 year of age and rare in animals less than 6 months of age. The one case of listeriosis within the 0 to 6 month group was in an animal 6 months of age.

There was no breed or sex bias in Michigan as these incidences closely followed the breed and sex distribution of the indigenous cattle population. It is doubtful if any of these factors, season, age, breed and sex, were of major importance in feedlot listeriosis. Consumption of silage and/or exposure to environment contaminated with *Listeria monocytogenes* as suggested by Olafson (1940), Grey (1960), Palsson (1963) and Gibbons (1963) seemed to be important in the epidemiology. The longer an animal lived, the greater was the probability of its contracting listeriosis, especially during the silage feeding seasons.

Microscopic lesions of listeriosis were typical of those described in the literature. Lesions were located principally in the brain stem and consisted of extensive perivascular cuffing and numerous glial nodules. These glial proliferations probably represented chronic microabscesses. Microabscesses were found but diffuse infiltration of neutrophils was not observed. The meninges also were usually

infiltrated with inflammatory cells, particularly over the posterior areas of the brain. Microglial cells and lymphocytes were the chief cellular inflammatory elements with neutrophils inconsistently present.

Polioencephalomalacia

Michigan's data differ from those reported by Jensen *et al.* (1956) in Colorado and Wyoming but agree with those reported by Terlecki and Markson (1961) in Britain and those reported by Little and Sorenson (1969) in Minnesota. The main differences were: 1) the age of the animals most commonly affected, and 2) the incidence of PEM in pastured cattle in the western states. These differences are explained by local management practices in the respective locations. The age of calves most commonly affected fell between 2 age parameters established for this survey. Calves 4 to 8 months of age were most frequently affected. In Michigan dairy calves born in the fall are fed a ration consisting entirely of stored feeds and they will consume these feeds during the change from the monogastric stomach to the ruminant stomach. Michigan feedlot operators raise feed crops in the summer. In the fall following harvest they purchase feeder calves weighing approximately 550 pounds or 6 to 8 months of age and place them directly into their feedlots. Western states are predominantly beef cow areas having a spring calving season. Feeder calves are pastured until they weigh approximately 750 pounds and are 10 to 12 months of age before they enter commercial feedlots for fattening.

Feeder calves, beef cows and dairy cows are pastured in the summer in Michigan but the incidence of PEM was lowest during this period of the year. The high incidence of PEM during the winter months might be attributed to greater environmental stress predisposing young calves to

PEM. Also this was a period when the diet is composed entirely of stored feeds. Such feeds by this time have had time to become contaminated with some substance which might interfere with the utilization of thiamine. These factors support the subclinical PEM theory of Davies *et al.* (1968) and Loew *et al.* (1970).

In speculation of the cause of PEM in these young bovine animals in the transitory state from monogastric to ruminant digestion and the role thiamine may play in the development of the condition, the following factors should be considered: Was there inadequate dietary intake and/or insufficient synthesis of thiamine to meet the needs of a rapidly growing body? Was there decreased absorption or chronic destruction of thiamine in the digestive tract? It seems logical to conclude that the younger the animal the greater the risk of its becoming affected with PEM, especially during periods of environmental stress and high dietary intake of stored feeds.

Infectious Thromboembolic Meningoencephalitis

Michigan's data agree with reports from other areas in that TEME has been basically a disease which infected feedlot cattle. Minor discrepancies between Michigan and other areas are explained by differences in local management practices. The greater incidence of TEME in the fall and winter months corresponded to the time of increased inshipment of feeder calves and to the time when Michigan feedlot operators assembled mixed lots of feeder calves into their feedlots. In addition to the adverse environmental conditions during this period, the stress of shipping and assemblage may have contributed to the incidence as suggested by Panciera *et al.* (1968). Outbreaks in some feedlots following the addition of new animals supported the possibility of an inapparent carrier state as suggested by Olander *et al.* (1970).

On several occasions when 2 animals, originating from the same feedlot and having similar clinical signs, were submitted to the diagnostic laboratory, one diagnosis of TEME would be made and one diagnosis named for the pathologic lesions observed would be made, the microscopic lesions being identical except for the presence of thrombi in the former and the absence of thrombi in the latter. The histopathologic lesions of many of the CNS cases in which no specific diagnosis was made closely resembled those of TEME but lacked the presence of thrombi. Sectioning lesions in other areas of the brain in many of these cases may have revealed the presence of thrombi. Within the CNS, the presence of a vasculitis and thrombi with an accompanying intense purulent inflammatory response should be strongly suggestive of and perhaps pathognomonic for TEME. The same lesions with the absence of demonstrable thrombi could be considered as suggestive of the infection especially with a compatible history.

Two cases diagnosed as listeriosis in 1966 were found to be TEME. One case each in 1967 and 1969 diagnosed as a suppurative meningoencephalitis were found to be TEME. Michigan probably has a greater incidence of TEME than is indicated by the data presented in this thesis.

In 1969 there was a sharp increase in cases of TEME. The initial diagnostic confusion between TEME and other suppurative encephalitides was not sufficient to have caused the observed increase. It may have taken *H. somnus* until 1969 to have sufficiently established itself in the cattle population to have resulted in an increased incidence of the organism in Michigan cattle.

Brain and meningeal lesions are probably not necessary for a diagnosis of *H. somnus* infection. The respiratory and joint involvement

as stated by Panciera *et al.* (1968) and Brown *et al.* (1970) are adequate clinical signs and postmortem lesions to justify a tentative diagnosis. The naming of the condition TEME was probably a misnomer. The suggestion by Brown *et al.* (1970) to refer to this condition as the *Hemophilus somnus* complex has considerable merit in light of our present information.

Lead Encephalopathy

The seasonal incidence of lead poisoning in Michigan corresponded to the time when the ground was not snow covered and when cattle were allowed the freedom of pasture conditions. Cattle are inquisitive and readily explore and taste foreign materials which may have inadvertently contaminated their surroundings. Since the lethal dose is based on grams per kilogram body weight, it would be expected that younger animals would be more susceptible. Lead poisoning in calves has usually been due to carelessness on the part of the owner, allowing pens and paddocks to become or remain contaminated with lead compounds. Allcroft (1950) demonstrated that metallic lead was not important as a primary cause of lead poisoning. Metallic lead when well weathered may contain ample surface quantities of lead salts which could result in toxicosis. The tendency for particulate material to accumulate in the reticulum where lead salts could be converted to soluble lead acetate by the action of the acid medium of the forestomachs may have been a factor in the increased incidence in older cattle. A more probable explanation of this incidence would be a direct relationship between time spent in the contaminated environment and risk of poisoning. Hence, the longer an animal lived, the greater the probability of its finding and ingesting lead compounds.

In 1969 there was a sharp increase in cases of lead encephalopathy. It is not possible to ascertain if the increased frequency of diagnosis was due to increased recognition or to an actual increase in incidence; it is suspected that both factors were involved. Many sources of lead have long been available for farm use. Recent interest in environmental pollution has resulted in an increased awareness of heavy metal toxicosis. The adage that you can't find something unless you look for it may be especially true of lead poisoning.

Gross lesions of lead toxicosis have not been observed. Liver, kidney and rumen contents from suspect cases were submitted to the Michigan Department of Agriculture Laboratory Division for toxicologic analysis. If the history, clinical signs and postmortem changes were compatible with lead poisoning, levels above 10 and 20 p.p.m. lead in the liver and kidneys, respectively, were sufficient to confirm a diagnosis.

Microscopic lesions in cases of lead poisoning confirmed by chemical analysis have been vague and nonspecific. Occasionally edema and small foci of spongiosis were found in the superficial cerebral laminae but were difficult to distinguish from artifacts. Eosinophilic meningeal infiltration as reported by Kradel *et al.* (1965) and cerebral laminar cortical necrosis localizing on the tips of the gyri as reported by Christian and Tryphonas (1971) were not seen in any of the confirmed cases. According to data presented by Courville (1958) and Christian and Tryphonas (1971), a laminar necrosis of the cerebral cortex would be expected to be observed in cases of lead poisoning. Such lesions would impair the microscopic differentiation between lead poisoning and PEM. Michigan findings in cases of lead poisoning have been more like those reported by Little and Sorensen(1969).

Encephalitis Due to *E. coli* Septicemia

The seasonal incidence of infectious calf scours was similar to that in other areas. The greater incidence of this disease in the winter supported the possibility of a stress mechanism initiating the disease as suggested by Moll (1965). During the winter months in addition to the adverse weather conditions, the quality of husbandry and nutritional value of the diet usually diminished. The high incidence in the 0 to 6 month age group of Holstein calves was consistent with local management practices. The majority of cases within this group occurred in calves less than 1 month of age.

There was no attempt to correlate lesions in visceral organs with those found in the CNS. The meninges were more severely affected; the inflammatory reaction was purulent in nature and extended into the parenchyma of the brain from foci of infective emboli.

Rabies

Microscopic lesions in the cases reviewed closely resembled those seen in listeriosis. Perivascular accumulations of lymphocytes were 3 to 4 cells in thickness. Neutrophils were not found in the "cuffs" and were often difficult to find in cases of listeriosis. There were no distinguishing characteristics of the glial proliferation of either condition. In rabies these lesions, unlike listeriosis, were not limited in anatomical distribution but could be found in the brain stem. When Asahi *et al.* (1957) and Borman *et al.* (1960) demonstrated that *L. monocytogenes* could ascend the trigeminal nerve, they in effect refuted the conclusion by Lapi *et al.* (1952) that changes in the gasserian ganglion were characteristic enough to make a presumptive diagnosis of rabies without demonstrating Negri bodies. The presence

of Negri bodies, while being pathognomonic for rabies, is also necessary to make the histopathologic diagnosis. Many laboratories consider the fluorescent antibody technique the only valid diagnostic procedure.

Malignant Catarrhal Fever

Outbreaks of MCF have been too few to permit valid conclusions in regard to the seasonal, age, breed and sex incidences. There does seem to be a predilection of the disease for summer months which agreed with data presented by Jubb and Kennedy (1970). The breed and sex incidences followed the normal cattle population distribution of Michigan. It is interesting to note that 30.8% of the cases of MCF were in the Brown Swiss breed. This was a reflection of a high incidence within one herd where both sheep and cattle occupied the same pastures.

The microscopic lesions observed in various non-neural tissues were consistent with those described by Jubb and Kennedy (1970) and Smith *et al.* (1972). Microscopic lesions in the brain and meninges were difficult to demonstrate. A few cases had isolated cerebral arteries with a vasculitis and endothelial cell necrosis. Moderate mononuclear meningeal infiltrations especially over the cerebellum were observed in some cases. Plasma exudations were not a characteristic finding, in contrast to data presented by Jubb and Kennedy (1970).

These data supported the statement by Jubb and Kennedy (1970) that MCF has 2 pathognomonic lesions. The vascular lesions, however, were not as common in the brain as in other tissues and there was no marked proliferation of lymphoreticular cells within the CNS. In Michigan the majority of cases of MCF have been diagnosed by histopathologic changes in non-neural tissues.

Miscellaneous Encephalitides

It was not intended to discuss each of the miscellaneous conditions encountered. Suppurative encephalitis was a common diagnosis and was based on the histopathologic lesions observed. When bacteriologic findings were nonconclusive or were not done, the pathologist reported cases out in this manner to suggest to the submitting veterinarian that the condition was bacterial in nature. Similarly a diagnosis of non-suppurative encephalitis suggested a nonbacterial condition.

Of the 6 cases of cerebellar hypoplasia all were in calves 0 to 2 weeks of age. Three of these cases were due to vaccination of the dam with the modified live BVD-MD virus.

SUMMARY

Diseases which affected the central nervous system of Michigan cattle during a 7-year period were surveyed. Seasonal, age, breed and sex relationships, the pathologic changes and the pathogenic mechanisms of the conditions encountered were discussed. Those diseases occurring most frequently were listeriosis, polioencephalomalacia (PEM), infectious thromboembolic meningoencephalitis (TEME) and lead poisoning.

Listeriosis occurred most frequently in Holstein female cattle over 2 years of age. It was most prevalent in the first quarter of the year and was associated with feeding silage. *Listeria monocytogenes* has been proven the causative agent. Microscopically there were lesions of limited distribution consisting of microabscessation, glial nodules and perivascular cuffing with lymphocytes.

Polioencephalomalacia occurred most frequently in Holstein calves 0 to 6 months of age. It was most prevalent in the first quarter of the year. The occurrence of this disease was associated with stress and a high dietary intake of stored foods. Treatment of affected animals has implicated thiamine in the pathogenic mechanism. Microscopically there was a laminar necrosis of the cortical gray matter.

Infectious thromboembolic meningoencephalitis occurred most frequently in steer calves of beef breeds 6 to 12 months of age. It was most prevalent in the fourth quarter of the year and was associated with stress and possible carrier animals. *Hemophilus somnus* has been proven the

causative agent. Microscopically foci of thrombosis and vasculitis with an accompanying purulent response were found randomly throughout the brain and meninges.

Lead poisoning occurred most frequently in Holstein female cattle. There were 2 high risk age groups, the very young and older animals. It was more prevalent in the second and third quarters of the year and was associated with pasture conditions where access to lead compounds was enhanced. Microscopic lesions were nonspecific.

Encephalitis due to *E. coli* septicemia, rabies, malignant catarrhal fever and a group of miscellaneous encephalitides were observed but were of low frequency.

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VITA

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In 1965 he graduated from Michigan State University as a Doctor of Veterinary Medicine. He then practiced in a dairy and swine area of Wisconsin for 1-1/2 years. Returning to Michigan he practiced in a dairy area of the Thumb for 1 year. He then moved to a mixed practice in his own home town.

In July of 1969 he became associated with the Laboratory Division of the Michigan Department of Agriculture, and entered the graduate school of Michigan State University majoring in veterinary pathology.

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