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# Nervous Coccidiosis

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# Nervous Coccidiosis

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Diseases involving the central nervous system of the bovine species present a bewildering diagnostic challenge to the veterinary profession, the large animal practitioner in particular. This bewilderment can be attributed to the fact that the clinical manifestations of many of these diseases are very similar and their etiologies are not completely understood. In recent years, however, research and study into the etiology, diagnosis, and treatment of many of these diseases have made their differentiation and treatment considerably easier for the practitioner. There still remain several significant diseases of the central nervous system on which little study or research has been conducted. "Nervous coccidiosis" of the bovine species is one such disease.

Since there is little in the literature about nervous coccidiosis, most of the information in this paper was gleaned from practitioners and clinicians who have diagnosed and treated cases of this condition. This clearly indicates the speculative nature of much of this paper.

The diagnosis of nervous coccidiosis is dependent upon the concurrent diagnosis of the enteric form of coccidiosis. For this reason we feel that a basic review of enteric coccidiosis is important before describing the nerovus syndrome.

It has been reported that there are at least ten species of coccidia of the genus *Eimeria* which infect cattle.[7] The species most commonly responsible for clinical coccidiosis are *Eimeria zurnii* and *Eimeria bovis*. An infestation occurs with the ingestion of sporulated oocysts. The action of the digestive enzymes and muscular activity of the gut free the sporozoites from the oocysts. The free sporozoites then invade the epithelial cells of the intestine, usually only one sporozoite per host cell. In some species, invading sporozoites may come to rest in macrophages of the *lamina propria*, or even *submucosa*, where they continue growth. The sporozoites grow and enlarge at the expense of the host cell and become trophozoites. The trophozoites become schizonts when a number of banana-shaped merozoites are produced by asexual multiple division known as schizogony. The schizonts rupture and release into the intestinal lumen the smaller merozoites which, in turn, invade other epithelial cells. After a limited number of asexual generations (dependent upon the species of coccidia) sexual reproduction begins with the trophozoites developing into gametocytes. The microgametocytes (male) form a large number of small motile microgametes. The macrogametes (female) remain much larger than the microgametes. The microgametes then leave the host cell, encounter the macrogamete in another host cell, fertilization occurs, and an oocyst is formed. The oocyst is released by rupture of the host cell and is eliminated in the feces. It takes approximately 17–24 days for completion of this cycle.

All endogenous stages of the bovine coccidial life cycle occur only in the alimentary canal and do not invade other organs.[3] The gametocytes seem to be the most pathogenic stages in bovine coccidiosis but all stages cause rupture of cells they invade.

In the case of *Eimeria zurnii* and *Eimeria bovis* the most severe lesions oc-

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cur in the lower ileum, cecum and colon.

Before the oocysts can be infective, they must sporulate. This process takes place most readily in moist, temperate or cool environmental conditions. For this reason, the disease is most common in the southern states; however, it is common throughout most of the United States. In the Midwest it is most common in late fall and early spring. Clinical severity is dependent upon the susceptibility of the host and the number of sporulated oocysts ingested. Infection is most common in calves between 3 weeks and 6 months of age. This age susceptibility is probably related to lack of immunity due to previous exposure; although physiologic differences, less fully developed immunologic competence, and lowered resistance due to environmental or nutritional stress may also play a part.

Because the oocysts are passed in the feces, infection usually occurs by ingestion of contaminated feed and water. For this reason the disease is most common in confined, crowded animals where fecal contamination can easily occur; e.g., feedlots and winter housing.

The first sign of illness usually occurs 2 weeks to 17 days post-infection with the sudden onset of diarrhea with foul smelling fluid feces containing mucus and blood. The feces may be dark and tarry or fresh blood may be present either mixed with the feces or alone. Tenesmus is a frequent finding and the tail and perineal region are usually blood stained. Anemia is variable, dependent upon the amount of blood lost, but in severe cases dehydration, emaciation, pallor of mucous membranes, staggering and dyspnea occur. The course of the disease is usually 5–6 days with death the outcome of severe infections. More commonly, recovery with some loss of gain, or actual weight loss and subsequent poor efficiency is the final outcome.

A tentative diagnosis can be made by the observation of the above signs. The diagnosis can usually be confirmed by examining the feces for oocysts via direct smear or more commonly, flotation. According to Blood and Henderson,[3] a count of over 5,000 oocysts/gram of feces is considered significant. The absence of a significant number of oocysts in the feces does not rule out the infection because the oocysts are not produced in significant numbers until after the merozoite and gamete stages —the two most pathogenic phases of the cycle.[3] In these instances, numerous free merozoites can often be observed in saline smears of feces or intestinal contents at necropsy.

Nervous signs in conjunction with clinical cases of coccidiosis are not a new finding. Marsh reported noting cerebral disturbances in certain cases of coccidiosis as far back as 1923.[14] The condition is seen in a very small percentage of those cattle manifesting clinical cases of enteric coccidiosis—possibly 4 percent or lower. There does not appear to be any breed or sex predilection. The syndrome is most commonly seen in feedlot calves under 500 lbs. but has been reported in calves up to 700 lbs.[11]

As stated previously, those animals which exhibit the nervous symptoms are experiencing a concurrent enteric infection. Usually the enteric infection is not difficult to diagnose—observation of characteristic diarrhea and perineal staining or oocyst count in the feces. It should be kept in mind that in certain cases the oocyst count may not be significant and there have been cases where diarrhea has not preceded initial central nervous system involvement. This is not to say that the enteric involvement is absent—only that it is probably either prepatent or subclinical.

Aside from the enteric infection these calves appear normal between seizures. This includes a normal temperature unless taken right after a seizure.

The seizures occur spontaneously or when the animal is excited or overactive. The animal suddenly becomes apprehensive and exhibits a stilted gate or stance. The muscles around the face and neck begin to twitch, the animal falls on its side and a grand mal type of epileptiform seizure ensues. There is tonic, clonic involvement of the entire body, opisthotonus is often seen and, quite frequently, nystagmus. The seizures terminate with a final motion in which the hind legs are drawn up against the abdomen and the eyes

rolled back. The duration of the seizures varies but usually lasts about five minutes. Following a brief period of rest (5–15 minutes) the animal is usually back on his feet and appears normal. As the condition proceeds, the duration of the seizures increases and the interval between seizures decreases.

The mortality rate of the nervous form is quite variable—if not treated, some say over 50 percent will die and if treated, less than 20 percent will die.[13] Others say that over 75 percent will die regardless of treatment.[9] Until the mechanism is discovered, the cause of death cannot be stated.

Gross lesions on post-mortem examination of the CNS are essentially non-existent. The only lesion which has been reported is cerebral edema detected on histopathologic examination and this is not a constant finding.[1,5] This is further complicated by the frequent occurrence of artifacts resembling edema in histologic preparations of CNS tissue.

Intestinal lesions are always present and are significant in confirming the diagnosis. Typically the mucosa of the cecum, colon and distal portion of the ileum is hemorrhagic to mildly necrotic with yellow patches of coagulated fibrin covering the luminal surface. Edema is often found in the mesentery, especially between the coils of the spiral colon.[6] If scrapings are taken from the involved mucosal surfaces, large numbers of oocysts are usually found. One interesting finding has been the presence of *Eimeria zurnii* in over 90 percent of the cases. Some individuals feel that the finding of *Eimeria zurnii* is important in confirming the diagnosis of nervous coccidiosis.[5,10,13]

Although the effect of the condition on the CSF, blood values and electrolyte balance has not been determined, it has been reported that these animals frequently exhibit glycosuria. It is possible that this is merely an "emotional glycosuria," which occurs when an animal is experiencing extreme fear or excitement. The stress causes increased secretion of epinephrine, which leads to sudden mobilization of glucose, hyperglycemia and spill over in the urine.[2]

Several theories have been reported regarding the etiology of this syndrome but these are purely speculative, and I am presenting them only as an aid in understanding the use of certain regimes of therapy.

It is thought by some that the nervous signs are the result of a toxin liberated by the coccidia.[4,8,12] These individuals feel treatment of the enteric infection and supportive measures are the best therapy. Systemic and oral sulfonamides are used—these are given for four to five days. Sulfa enemas (2–4 oz./treatment) *bid* are also thought to be of value. Supportive therapy includes Vitamin ADE injections IM and fluids when necessary.

Other individuals feel that the nervous signs are the result of an electrolyte imbalance—hypomagnesemia in particular.[5] Certain practitioners have reported good response to the administration of ½ to 1 oz. magnesium sulfate (½ oz. < 350 lb.; ¾ oz. 350–600 lb.; 1 oz. > 600 lb.) in ½ to 1 gallon of normal electrolyte solution (volume dependent on the degree of dehydration). In addition, the enteric infection is treated with oral and/or parenteral sulfonamides. If seizures can still be induced after twenty-four hours, the magnesium sulfate treatment is repeated. Usually no more than two treatments are required.[11]

A third theory holds that the condition is due to a prolonged inadequate intake of Vitamin A in conjunction with impaired Vitamin A absorption due to chronic subclinical coccidiosis.[6,9] It has been proven that growing calves deficient in Vitamin A may have an elevated CSF pressure and clinically may exhibit muscular incoordination, convulsive seizures and staggering gait.[7] This deficiency has not, however, been proven in calves exhibiting nervous coccidiosis. The treatment advocated includes:

Coccidiostat
Supportive therapy
  Vit. A, balanced electrolytes IV if dehydrated, B-complex, amino acid solutions.

Regardless of the differing ideas as to etiology, all people contacted felt that treatment of the enteric involvement was essential.

From this discussion it becomes appar-

ent that the nervous form of coccidiosis is not a well-understood syndrome. In spite of this fact, diagnosis of the syndrome and differentiation from other similar nervous conditions can be made if the following features are kept in mind:

1) Between seizures the animal appears normal with the exception of signs indicative of enteric coccidiosis;
2) Seizures may occur spontaneously but they can usually be induced by exciting the animal;
3) The seizures are epileptiform in type;
4) Animals exhibiting the nervous signs have a concurrent enteric infection;
5) Post-mortem findings are negative except for the lesions due to the enteric infection.

### *FOOTNOTES*


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# Diabetes Insipidus— An Overview and a Case Report


**by R. L. Peiffer, Jr., D.V.M.**


Antidiuretic hormone (ADH, vasopressin) is an octapeptide produced in the nuclei of the anterior hypothalamus. The major source of this hormone is the supraoptic nuclei with minor production taking place in the paraventricular and filiform nuclei. From these centers ADH is secreted down the supraopticohypophysial tract to the posterior pituitary where it is stored until released in response to the appropriate stimuli.

The hormone is released in response to increased solute concentration—primarily sodium and its salts—in the plasma or extracellular fluid. ADH is carried by the vascular system to the kidney where it renders the collecting duct epithelium more permeable to water, allowing osmotic equilibrium between the tubular fluids and the hyperosmotic interstitum. Dilution of plasma and/or extracellular fluids inhibits ADH release, completing the negative feedback system.

The thirst center is functionally and anatomically closely related to the antidiuretic mechanism. Osmotic stimulation of the supraoptic nuclei creates a sensation of thirst; dilution of plasma inhibits thirst.


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