Polioencephalomalacia

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The general topic of this meeting is "General Metabolic Disorders, their Diagnosis and Treatment." I will talk briefly on the subject of polioencephalomalacia and its relationship to thiamine metabolism, but my emphasis will probably be in the field of diagnosis.

It is now approximately 20 years since Doctors Jenson, Greiner and Adams, at Colorado State University, were the first to recognize cases of what they then were calling "forage poisoning"—the disease which we now recognize as polioencephalomalacia. It was also recognized in cattle in Wyoming, where it was known as either "blind staggers" or "alkali disease." This may seem an appropriate time for us to review our knowledge of this disease, or put another way, to review and assess our ignorance of it!

Since those days in the early 1950's, polioencephalomalacia, whether known by that name or some other, has been recognized or identified in cattle in other areas of the world, particularly in the mid-western and eastern states of the United States, as well as in Canada, in Britain, on the European continent, in New Zealand and in Australia. The disease, although first recognized in beef cattle, has also been recognized in dairy cattle, and although first recognized in animals older than six months of age, it has since been recognized in much younger calves, particularly in Britain.

In some of these other areas of the world, the term "cerebral cortical necrosis" is used to define this disease. We, however, prefer to continue to call it polioencephalomalacia since we believe that there is more to the process than necrosis of the cortex. As you will be aware, a similar disease, possibly an identical disease, exists also in sheep, and it has also been recognized in goats.

So, this appears to be a disease confined, more or less, to the ruminant species. How may we define this condition? Well, polioencephalomalacia is an acute non-infectious, afebrile, metabolic disorder occurring in young ruminants from about four months up to about two years of age. Generally, it is seen in early winter and it is characterized by sudden onset, in individual or several animals in a

herd, of signs of disorientation, early excitation followed by depression, muscle tremors, impaired vision or blindness, leading to recumbency with the grinding of the teeth, opisthotonus and ultimately to coma.

The course of the disease in affected individuals may extend only over a period of a few hours, terminating either in death or in recovery, which may be complete when the course is so short. Alternately, the disease may extend over a much longer period, which may extend from days to even weeks, and may lead to partial recovery, though there usually is a permanent residual visual defect, or it may lead to the production of a typical "dummy" animal.

The morbidity in any particular group of cattle is usually fairly low, usually in the region of 5% or 10%, but it may occasionally occur in some herds under certain circumstances with a much higher morbidity, and the disease has been seen in Wyoming with a 60% morbidity in quite large herds. About one-half of the affected animals will die, and most of the remainder will recover only inadequately; that is, with some residual impairment of vision or with the "dummy" syndrome.

Now I said that I would like to emphasize particularly the diagnosis of this condition, and particularly the differentiation of this disease from other diseases of the brain that commonly occur in cattle in this country. For clinical diagnosis we depend heavily upon the recognition of some of the signs I have mentioned in defining the disease, and it is then necessary to differentiate this condition from these other relatively common disorders of the brain of this class of livestock. Therefore, the problem resolves itself into recognizing polioencephalomalacia from thromboembolic meningo-encephalitis, the *hemophilus somnus* infection, which in our experience occurs as commonly or more commonly than polioencephalomalacia. Secondly, other infectious meningitis and encephalitis of cattle, particularly those due to streptococci, in yearlings or two-year-olds, and those due to *E. coli* in much younger calves. Thirdly, to differentiate from listeric encephalitis, which in our experience in recent years, is a very much less common condition in cattle than is either polioencephalomalacia or thromboembolic, and, finally, to differentiate polio from brain abscess and other sporadic incidental occurrences of individual animals.

I would emphasize, particularly, in clinical diagnosis and differentiation then, the following features of polioencephalomalacia. First, this disease has a normal temperature in the individual, from the beginning of clinical signs. Secondly, this disease does not commonly give rise to localizing neurologic features; that is, clinical features that one relates to the involvement of individual cranial nerves, such as, for example, dropped ear, head tilt, or deflection of the tongue, etc. These are localizing signs indicating involvement of individual cranial nerves, and these signs are characteristically not seen in polioencephalomalacia; whereas, as you will note, they characteristically are seen in the listeric encephalitis. Thirdly, a feature of this disease is the domination of the features of dullness and depression over features of excitation. What I

mean by that is that in this disease, particularly in its later stages, or shall we say in the stages after the first few hours or the first day, these animals tend to be much more dull and depressed than they tend to be excited. Excitation, contrary-wise, is a feature perhaps more of thromboembolic meningo-encephalitis, and other infectious meningitides. And, finally, amongst the points I would emphasize in clinical diagnosis, there is in this disease, polioencephalomalacia, an absence of stiffness or swelling of the joints which may be commonly seen in animals affected with thromboembolic menino-encephalitis. So, once again then, normal temperature, from the beginning of the course of the disease, the absence of localizing signs, the domination of features of dullness or depression, rather than features of excitation, and the absence of features of stiffness or swelling of the joints. Since a high proportion of these animals die, often the problem presents itself to you as one of postmortem diagnosis.

The postmortem diagnosis of this disease depends upon a reasonably careful examination of the brain. There are essentially no lesions in other organs that are related to this disease, and the first point about the examination of the brain that I would emphasize is that polioencephalomacia is essentially the only disease of cattle that gives rise to a uniform swelling of the brain. It is essentially the only common disease of cattle that fairly consistently gives rise to a uniform swelling of the brain. This is in my view an extremely valuable tip, but it requires that you be able to recognize the features of brain swelling in cattle.

First, of course, in this disease, there is a tendency for the cerebral cortex, particularly, to have a pale yellow color, and the texture of this cortex is soft and rather sloppy. It is characteristically bloodless; that is, the small capillaries of the leptomeninges overlying the cerebral hemispheres are usually bloodless, so there is an appearance of *ischemia* that goes along with the palor of the brain. The top of the gyri may be seen to be somewhat flattened in most instances, so the texture of the brain is rather soft and fluctuent and sloppy, but where the tops of the gyri have been pushed against the bony cranium, they tend to be flattened. These are features then that are seen in the affected areas of brain tissue, particularly the cerebral hemispheres. The other features that I would recommend you to look for as evidences of brain swelling include the following. If you will carefully examine the cerebellum in animals affected with polioencephalomalacia, you will often be able to remark that the cerebellum has been compressed caudally into the foramen magnum, so that the edges, the surfaces and edges of the cerebellum, reflect the effects of this compression. Now, what I mean by that is that the lateral edges of the cerebellum will often in these cases be sharp because the whole cerebellum is jammed backwards into the foramen magnum, and this then becomes a concave shape, as well as the occipital hemispheres which press into the tentorium. This is a valuable sign of brain swelling. It means that the cerebral hemispheres have increased in volume; they have pushed

backwards on the tentorium cerebelli, which is the sheet of duri extending across this region, and with this pressure they have compressed the cerebellum into the cranium magnum. A further useful sign is the presence of necrosis in the hippocampus. This feature is a reflection of the brain swelling. I think you are all aware of the fact that this disease is characterized by cerebral cortical necrosis and softening.

You will see brownish yellow discoloration of the cortical tissue. All of that cortex has undergone necrosis.

In animals that have survived for a longer period, the cortex undergoes complete malacia and as a result the meninges then come to lie on the white matter of the brain itself. This is a so-called decorticated brain. A cross section of such a brain shows the complete stripping of cortical tissues away from the underlying white, so that the meninges come to lie directly upon the white matter.

Histologically, this is a disease characterized by acute necrosis of neurons which result ultimately in a malacic response with cystic cavitation, and the production of this feature where the meninges are then lying over the cystic space.

This disease, from a postmortem diagnosis point of view, can be differentiated from other common encephalitities of cattle, particularly tromboembolic meningo-encephalitis where the surface of the brain is frequently marked by rusty brown spots. The disease can be recognized from those common meningitis infections of cattle, particularly *Strept* and *E. coli*, where there is visible exudate in the leptomeninges. Exudate is never present in polio. The disease may also be differentiated from a case of brain tumor since the latter gives rise to asymmetric swelling of the brain, whereas polio gives rise to symmetric swelling. We know very little in spite of twenty years about the cause of this disease, and I will not attempt to review the various suggestions that have been made. These cover selenium poisoning, cobalt deficiency, enterotoxemia, hydrogen sulfide inhalation from rumen, etc.

In 1965, in Britain, the suggestion was made that animals with cerebral cortical necrosis respond to thiamine treatment. Support has been given to the concept of this disease being a thiamine deficiency syndrome or one related to abnormalities of thiamine metabolism by the following evidence: First, in Britain, calves affected with polioencephalomalacia have a high blood pyruvate level. They have a low blood transketolase level—this enzyme being thiamine dependent. They have low thiamine levels in the brain and the liver, and they have, in Britain, been able to demonstrate, in some instances, the presence of a thiamine-destroying enzyme, a so-called thiamine-ase in the rumen contents of affected animals. In Britain, the present feeling is that this disease may be related to certain feeds that contain molds able to produce thiamine-ase enzymes, and the presence of these molds in the rumen reduces the effectiveness of rumen microorganisms to synthesize thiamine.

Experience in North America generally centers around the effects of treatment with thiamine, and in general, I think it should be said that the effects of treatment have been variously reported by different authors. Some claim that the thiamine treatment is miraculously effective; others claim that it is essentially no better than the administration of fluids and corticosteroids. There is, therefore, a great difficulty in the interpretation of results with regard to this disease, and I think personally that this difficulty may arise out of the problem that polioencephalomalacia in different parts of the world, and in different classes of livestock, may not be one disease but several diseases—and that these several diseases arising out of different circumstances may all affect different individuals in the same way they produce brain swelling, and the consequences of brain swelling, I then believe to be cerebral ischemia cortical necrosis, and malacia of the cerebral cortex.

CHAIRMAN FOX: Thank you very much, Dr. Young. You will note that I didn't bug him quite as much as the other speakers because I have an inclination to have an inherent fear of pathologists. (Laughter)