EQUINE INCOORDINATION (WOBBLER SYNDROME) OF HORSES By

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Equine incoordination ("wobbler syndrome") is a disease of horses characterized by disturbances of gait with acute episodes and chronic manifestations. The incoordination of locomotion most commonly involves the pelvic limbs but may also involve all four limbs. The term "wobbler" has been applied to this disease because of the characteristic gait of the affected animals, that is, they wobble from side to side as they walk. The disease has been recognized in all age groups from weanlings to 20 year olds.

While equine incoordination has been definitely recognized as a clinical entity, there has been question as to whether the pathological manifestations were constant enough to warrant its acceptance as a pathological entity. The etiology in most cases has not been definitely established. This report describes the lesions found in animals sacrificed after varying durations of illness in an attempt to determine the etiology and to develop a concept of the pathogenesis of the disease.

Note

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Materials and Mothods:

Clinical observations of 140 horses*, 37 of which were wobblers, at the Royal Thai Army Horse Breeding Farm, Kanchanaburi, were made for gait coordination ability, A film record was made of 8 cases with gait incoordination and inability to back up without falling.

Blood samples were collected from the 140 horses for hematological, parasitological, and serological examination. Routine hematolosical examinations, hematocrit, Hemoglobin, erythrocyte counts, white blood cell counts and differential counts, were done on samples from 22 wobbler and 10 control horses of this group. From the 140 samples, wet mount blood films were examined for microfilaria and l ml. volumes were examined following Knott's concentration technique (Knott, 1939). The 140 samples were examined to determine if the hemagglution inhibition titers for Japanese B encephalitis and Chikungunya were more frequent or of a greater magnitude in affected horses.

To obtain cerebrospinal fluid (CSF) from 20 horses, the horses were tranquilized with promazine hydrochloride, 20 minutes later a casting harness was applied, the horses cast, and then tied in lateral recumbency. The skin site over the atlanto-occipital area was surgically prepared and the skin infiltrated with a local anesthetic. An 18 gauge, short bevel, 80 mm. needle was introduced into the cysterna magna for collection of CSF. The samples were frozen with dry ice until examined for sugar, total protein, and chlorides. Total cell counts and differential cell counts were obtained at the time of CSF collection.

Eleven horses varying in age from 8 months to 22 years, which were sacrificed or died, were necropsied. Four of these were control material

Note *In conducting the research described in this report, the investigators adhered to the "Guide for Laboratory Animal Facilities and Care", as promulgated by the Committee on the Guide for Laboratory Animal Resources, National Academy of Sciences, National Research Council.

and 7 were horses sacrificed 3 months to several years following the onset of illness. The necropsy included removal of the spinal cord and brain, examination of the viscera and examination of the joints by open incisions. During necropsy the entire cerebrospinal fluid was collected, then filtered, and the sediment examined microscopically. The tissues were fixed in 10% buffered formalin. Staining methods used were hematoxylin and eosin (HGE), periodic acid-Schiff method, and Luxol fast blue-PAS.

Results:

Typically, the "wobbler" horse showed incoordination of the hind legs, but occasionally the forelimbs were involved also. The result of this incoordination was apparent at the walk as well as at other speeds of movement. Backing and turning in a tight circle helped in the analysis of the gait deficit. When backing the affected horses would sit, fall, or do a complete back flip. In turning they would get their legs crossed and often fall. At the walk the affected horse was seen to sway from side to side and to flex or extend a limb either excessively or inadequately. A loss of cutaneous sensation was not observed.

The hematologic findings from 22 wobbler and 10 control horses were essentially the same, The average results from the wobbler group were as follows: hematocrit 37%, hemoglobin 13.4 gm/100 ml, erythrocytes 8,989,000/cu. mm, white blood cells 12,280/cu mm. and differential counts, neutrophils 46%, lymphocytes 49%, monocytes 1% and eosinophils 4%,

Microfilaria were found in wet mount blood films of two affected and one normal horse of the 140 horses. An adult Setaria equina was found free in the peritoneal cavity of one wobbler horse and Setaria spp. were found in the eyes of two wobbler and two normal horses.

The hemagglutination inhibiton titers for Japanese B encephalitis and Chikungunya were essentially the same frequency and magnitude in affected animals as in control animals (Table I). The frequency and magnitude of titers increased with age in all groups.

The cerebrospinal fluid findings are shown in Table II. The results are within normal limits. Even in the case where a nematode was found migrating in the spinal cord there was no pleocytosis or biochemical changes.

Necropsy of seven characteristic cases revealed grossly visible focal areas of yellowish gelatinous softening in the brain. The areas ranged from microscopic sizes up to 1.5 cm in size. They were found in and around the internal capsule, putamen, thalamus, and in the central white matter of the cerebral cortex. Microscopically, the areas were focal cavitations surrounded by malacia, demyelination, and loss of nervous tissue components in the area. Blood vessels and accompanying connective tissue formed a lacy spider web pattern throughout the area of malacia. Pigment laden "Gitter Cells" were numerous in the area. In one instance, cavitation surrounded by malacia and demyelination was found at Cl level of the spinal cord.

In the microscopic examination of the spinal cord from an eight month old horse a nematode parasite was found in the cervical cord at G8 level. It was located in the dorsal white matter and involved the funiculus cuneatus and funiculus gracilis. The parrsite, a larval stage, was surrounded by an inflammatory reaction consisting of macrophages, small mononuclear cells, eosinophils, and malacia of the nervous tissue.

Osteoarthritis and arthropathy of the intervertebral diarthrodial and amphiarthrodial articulations as described by Fraser and Palmer, 1967, were not seen in affected animals, nor were the spinasl column constrictions of the spinal canal as described by Olafson 1942, found in affected animals. However, the major articulations of the legs, hip, stifle, hock, shouldesr, and elbow did have loss of surface cartilage and corrugation or grooving of opposing joint surfaces,

Nodules, from 4 to 10 cm. in diameter were found in the stomach walls of all horses necropsied. Each horse had one to three of the nodules which contained Habronema spp. Microscopic examination of sections from all of the viscera failed to disclose any constant microscopic lesion that could not be explained on the basis of some intercurrent disease.

Discussion:

Japanese B encephalitis has been suggested as a cause of "wobbler syndrome" as well as an accompanying disease to cerebrospinal nematodiasis (Innes and Saunders, 1962). In chronic Japanese B encephalitis of horses malacia has been found in the thalamus, cerebral cortex, substantia nigra, and basal ganglia. We found similar lesions of malacia in "wobbler" horses; however, there was no associated Japanese B encephalitis titer

Cervical cord lesions as the result of intervertebral arthropathy or osteoarthritis as reported by Dimock and Errington, 1939; Jones, Doll, and Brown, 1954; and Steel, Whittem and Hutchins, 1959, were not found in our cases. However, arthropathy was found in the major joints of the legs, particularly in the rear legs. It is a matter of conjecture to postulate that the arthropathy developed after the CNS injury, although probable. Hyalinization of blood vessels in the lesions was reported by Fraser and Palmer, 1967. They found the malacia of the spinal cord to be particularly associated with hyalinization of blood vessels in all cases. We found fibrosis around blood vessels in areas of malacia but not the hyalinization. Innes and Saunders, 1962, have shown that clinically and pathologically the findings in proven cases of cerebrospinal nematodiasis have no essential points of pathologic difference from "wobbler syndrome". They assert that it is due to the immense difficulties in finding lesions and then worms in a brain and very long spinal cord that more wobbler cases are not shown to be due to nematode migration. Our finding of only one migrating nematode in the spinal cord confirms the difficulty. The same type of encephalomalacia seen in our cases has been shown by others to be the aftermath of nematode migration, Innes, Shoho, and Pillai, 1952.

Summary:

Equine incoordination ("wobbler syndrome") in Royal Thai Army horses was studied by clinical and pathologic examinations. Thirty-seven of

140 horses at Kanchanaburi were affected. The pathologic lesions were encephalomalacia of the central white matter of the cerebral cortex and basal ganglia. In one case a nematode parasite was found in the cervical spinal cord. The lesions were not associated with an increase in Japanese B encephalits titer or with cerebrospinal fluid or hematological changes.

HEMAGGLUTINATION INHIBITION TEST

TABLE I

	TITERS					
SAMPLES	Japanese B	Encephalitis	Chikungunya			
10.29 年3年3年4月1日	Range	Average	Range	Average		
37 Horses with wobbler syndrome	0-320	66	0-80	50		
20 Horses with ophthalmic pathology	0-160	55	0-80	32		
83 Essentially normal control horses	0-160	51	0-160	52		

CEREBROSPINAL FLUID FINDINGS

TABLE II

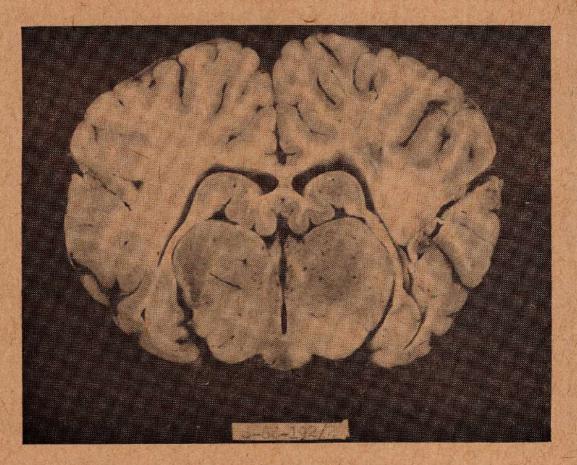
Name	Gross	Cell count	Sugar mg %	Chlo-	Total	Differential Count	
	Appe- arance	per cu. mm.		rides mEg/L	Protein gm %	Lymph- ocytes	Undifferen- tiated
Kamjoon	Clear	<1	70	130	15.4	4	5
Kaitpichit	Clear	<1	71	129	44.0	(0)	.2
Keptpoo	Clcar	<1	52	129	41.0	1	2
Ngoom-Ngum	Clear	<1	60	127	32.7	0	2
Gradoung	Clear	<1	41	108	32.0	1	2
Karnjanart	Clear	<1	60	131	30.0	2	1
Kapong	Clear	<1	64	129	30.6	0	2
Pugsai	Clear	<1	56	135	25.0	0	2
Keptong	Clear	<1	84	134	14.6	0	2
Kordai	Clear	<1 <1	60	130	11.1	0	3
Karngeow	Clear	<1	54	130	24.3	0	2
Abpob	Clear	<1	50	116	21.9		1
Gounar	Clear	<1	53	125	24.3	1	4
Gromarech	Clear	<1	56	122	38 2	3	4
Hataporn	Clear	<1	62	137	35.1	0	2
Sandug	Clear	<1	45	131	31.3	0	2
Ronio	Clear	<i< td=""><td>77</td><td>122</td><td>40.3</td><td>1</td><td>2</td></i<>	77	122	40.3	1	2
Goo-Lee	Clear	<1	57	123	44.5	0	1
Goumgum	Clear	<1	55	123	40.0	2	1
Kumjun	Clear	<1	57	124	35.0	1	2

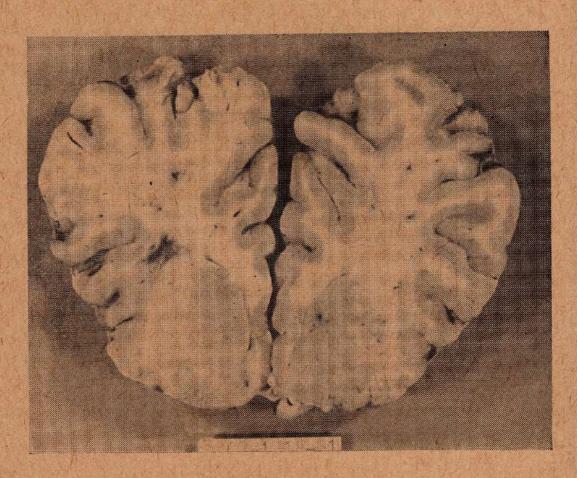
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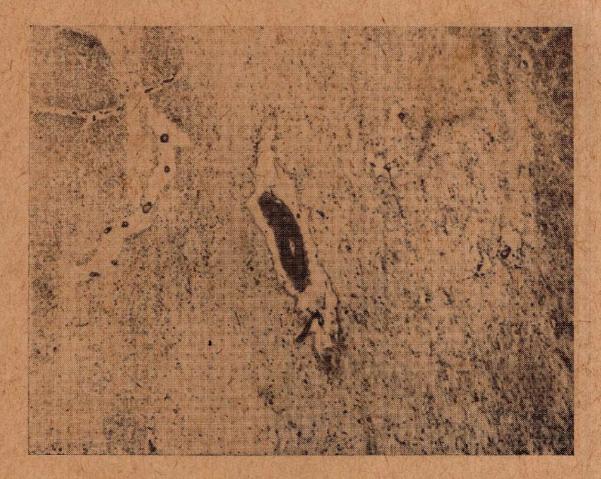
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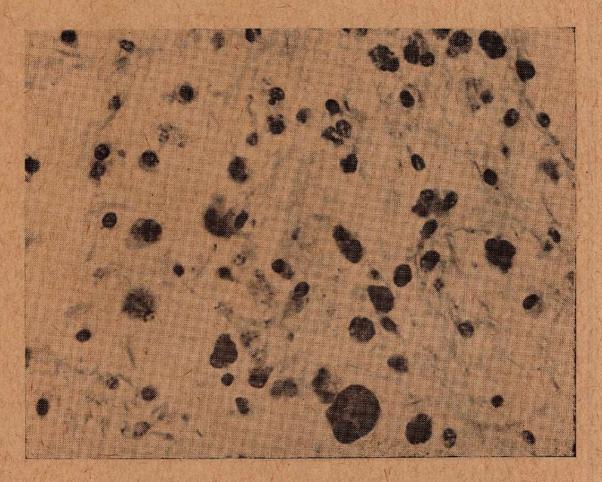
FIGURES

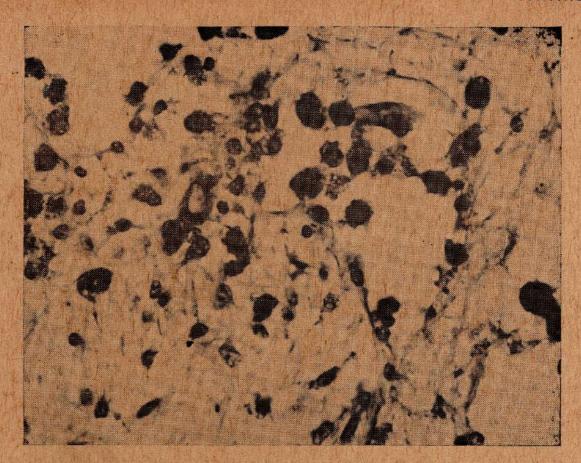
- Figure 1. Coronal section of cerebral cortex with yellowish gelatinous softening in the central white mater.
- Figure 2. Cerebral cortex with dark area of malacia in central white matter.
- Figure 3. Malacia of central white matter in cerebral cortex.
- Figure 4&5 Pigment laden "Gitter Cells" in the areas of malacia.
- Figure 6. Cl level of the spinal cord with malacia and demyelination.
- Figure 7. Inflammatory reaction surrounding nematode in the spinal cord at C-8 level.
- Figure 8. Two sections of nematode at C-8 level of the spinal cord.
- Figure 9. Swollen axis cylinders and disappearance of nerve fibers in the spinal cord.

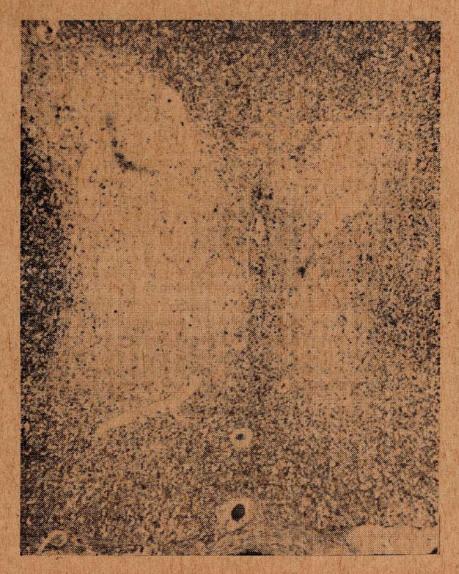


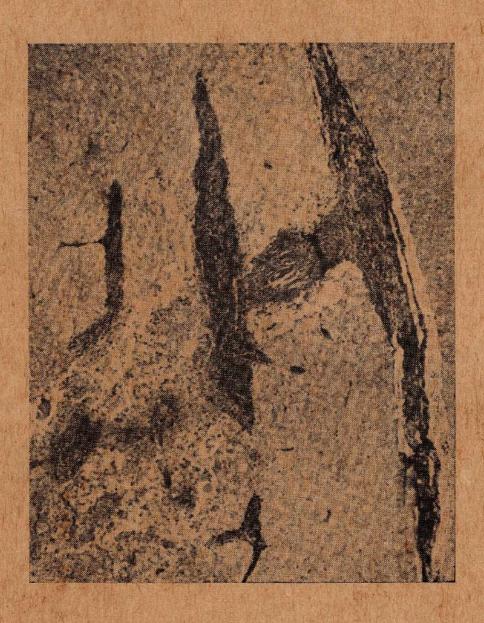


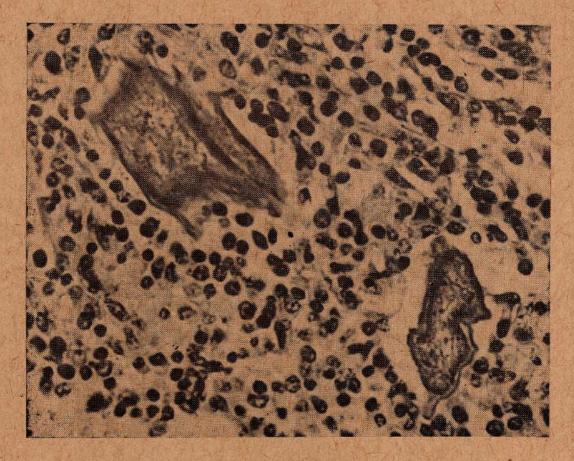


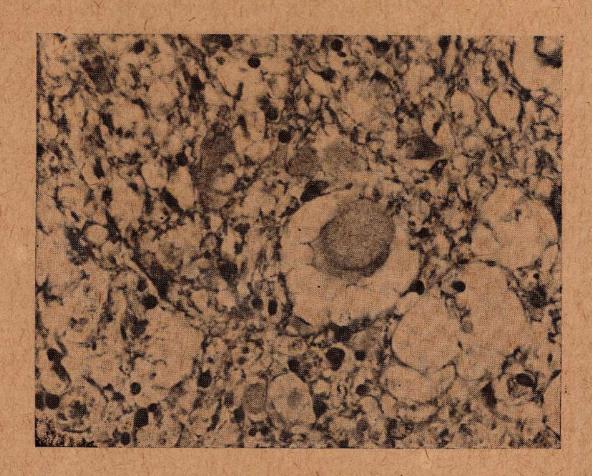












โรคการขาดการสมดุลย์ในม้า

(วอบเบลอร์ ซินโครม)

โดย

เจร์. ดี. พุลเลียม, ดุสิต จันทยานี และ ชาร์ล. อี. เกิลสตัน แผนกทดลองพยาชิวิทยา องค์การ ส.ป.อ.

ม้าของกรมการสัตว์ทหารบกที่จังหวัดกาญจนบุรี จำนวน 140 ตัว ได้รับ การศึกษาถึงเรื่องโรคการขาดการสมกุลย์ในม้า (Incoordination or Wobbler Syndrome) ปรากฏว่ามีม้าเป็นโรคนี้จำนวน 37 ตัว

การศึกษาของทางพยาธิวิทยาพบว่าเป็นเพราะ encephalomalacia ของ Central White matter ของส่วนที่เป็น Cerebral Cortex และ basal ganglia จาก ม้าที่เป็นโรคได้ตรวจพบว่าม้าป่วยตัวหนึ่งมีพยาธิ nematode อยู่ที่ Cervical Spinal Cord

ลักษณะทางวิการของม้าที่เป็นโรก ได้พบว่ามีความสัมพันธ์เกี่ยวกับการ เพิ่มขึ้นของ Japanese B. encephalitis titer และไม่มีการเปลี่ยนแปลงของ cerebrospinal fluid หรือการเปลี่ยนแปลงของโลหิตไปจากปกติ.