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What is This?
Spinal Ataxia in Zebras
Comparison with the Wobbler Syndrome of Horses


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Abstract. Eight of 17 zebra foals (Equus burchelli) born at the National Zoological Park, Washington, D. C., developed an ataxic condition that clinically resembled the wobbler syndrome of horses. Four were males and four were females. All were the progeny of one stallion and two mares. The parent animals were not ataxic. In three of the affected foals raised at the zoo, signs progressed to severe ataxia, and the animals were killed. In contrast to the findings in wobbler horses there was no radiographic or pathologic evidence of narrowing of the vertebral canals, nor were malacic foci found in the cords to suggest that focal compression had occurred. There was degeneration of ascending and descending tracts in the same segments throughout the spinal cords. There were no lesions in two brains studied nor were other possible causes for the spinal cord degeneration evident. These findings and the high incidence of ataxia in this herd suggest a familial degenerative myelopathy.

Approximately half of the zebra foals born over a 12-year period at the National Zoological Park developed a disorder of the central nervous system that clinically resembled the wobbler syndrome (wobbles, equine incoordination, ataxia of foals [5, 7, 8, 14]). This is an ataxic condition of predominantly young male horses that is characterized by a swaying or wobbling of the hindquarters. It can be progressive and may lead to muscular weakness and severe locomotor disfunction of both pelvic and thoracic limbs. A similar clinical picture is described in eight zebras. The pathologic changes in the spinal columns of three animals, however, indicate that the pathogenesis may not be the same as for the wobbler syndrome. These differences are discussed, and the zebra disorder is compared with ataxic conditions of other species and man. This is believed to be the first report of a possible primary degenerative myelopathy in an equine species.
**Clinical Study**

The original herd of Grant's zebras exhibited at the National Zoological Park was derived from one stallion (Tom) and two mares (Janet and Zebbie) (fig. 1). These animals were unrelated and showed no abnormalities of gait or signs of incoordination. From 1960 to 1971, there were 17 offspring (10 from Janet and 7 from Zebbie) of which eight were ataxic in the hind quarters. As noted in the pedigree chart (fig. 1) a precise account of sex ratios and frequency cannot be made since complete records of all foals were not available. Four of the affected animals were males and four females.

Signs of ataxia began between 4 and 6 months of age. They were first detected as subtle incoordinated movements of the hindquarters that were most noticeable when the animals were trotting slowly or turning. The ataxia could be accentuated by forcing the animals to move in a small circle. Detailed neurological examinations were not possible because of the difficulties of manipulating these animals, but vision, temperament, and other motor functions appeared to be normal. Appetite and hematological studies were within normal limits. The animals were fed standard pelleted horse feed and hay and had access to salt bricks containing trace minerals. There were few endoparasites in the herd because of routine examination and treatment.

Four of the mildly ataxic zebras were sent elsewhere before they were yearlings, and further records were unavailable; one other animal died of causes unrelated to the ataxia. Three affected animals, A1, A2, and A3 (table I) remained at the zoo, and their condition worsened. Between 1 and 2 years of age they developed severe ataxia and weakness of the rear quarters, with milder weakness of the thoracic limbs. Hypermetria was not seen, nor was the ability to step backwards appreciably altered. They often fell and made strenuous efforts to right themselves. Because of the trauma from these episodes, the animals were killed and necropsied. The original herd sire, Tom (N1) was also killed and necropsied because of his advanced age and an arthritic condition of his front limbs.
Table I. Identification, clinical condition and tissues available at necropsy

<table>
<thead>
<tr>
<th>Zebra</th>
<th>Sex</th>
<th>Ataxia</th>
<th>Age at necropsy</th>
<th>Tissues studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>male</td>
<td>advanced</td>
<td>3 years, 1 month</td>
<td>spinal cord only</td>
</tr>
<tr>
<td>A2</td>
<td>female</td>
<td>advanced</td>
<td>1 year, 4 months</td>
<td>cervical vertebrae, brain, cervical and thoracic cord</td>
</tr>
<tr>
<td>A3</td>
<td>female</td>
<td>advanced</td>
<td>1 year, 9 months</td>
<td>cervical vertebrae, entire central nervous system</td>
</tr>
<tr>
<td>N1</td>
<td>male</td>
<td>none</td>
<td>20 years</td>
<td>cervical vertebrae, entire central nervous system</td>
</tr>
</tbody>
</table>

Materials and Methods

At various intervals zebras A2, A3, and N1 were each given 3 mg of M99 (etorphine, Cyanamid®) plus 30 mg of acepromazine (Provet®) while restrained in a chute (Ranger). After the animals were immobilized, an endotracheal tube was inserted and fluothane (halothane, Ayerst Laboratories, New York, N.Y.) was administered to produce surgical anesthesia. Radiographs of the cervical vertebrae were taken both while the zebras were in normal posture and during extreme flexion of the head and neck. Lateral and ventrodorsal views were obtained. The animals, including A1, were killed and immediately necropsied.

The tissues available (table I) from the three affected zebras (A1, A2, and A3) and from the one unaffected animal (N1, control) were processed for study in the following manner. The cervical vertebrae were disarticulated, trimmed of excess muscle tissue, dried, and exposed to a colony of Dermestidae beetles. The brains and spinal cords were removed intact, examined for gross abnormalities, and fixed in 10% formalin. After fixation, the spinal cords were sliced transversely at 2- to 5-mm intervals, and the serial sections were examined with a stereomicroscope. Samples from each segment, together with brain tissue, were embedded in paraffin, cut at 6 µm, and stained with hematoxylin and eosin (HE).

Special staining techniques performed on selected samples from all cases included, luxol blue-cresyl violet, Weigert (myelin), Holzer, Bodian, periodic acid-Schiff-Alcian blue, Mallory’s trichrome, and oil-red-O (frozen tissues).

Results

Radiographic Findings

The cervical vertebrae appeared normal. Vertebral subluxation or narrowing of the cervical vertebral canals was not evident.
Fig. 2. a Transverse section of cervical spinal cord (C4) from ataxic zebra, A3. Pale areas are loss of myelin in lateral and ventral funiculi. Compare with figure 1b. Weigert’s myelin stain. b Transverse section of cervical spinal cord (C4) from nonataxic zebra, N1. The myelin stains fairly consistently throughout the cord. Section prepared and stained at the same time as section in figure 1a. Weigert’s myelin stain.
Fig. 3. Higher magnification from an area of myelin pallor in the lateral funiculus shown in figure 1a. Myelin sheaths are irregular sizes. Many are distended and empty. HE.

Fig. 4. Similar area as in figure 2. Longitudinal section. Axons are depleted. A few axonal fragments remain. Bodian stain.
Fig. 5. Longitudinal section of spinal cord from zebra A3. Lipid-laden macrophages accumulate in a dilated myelin sheath. HE.

Fig. 6. Longitudinal section of thoracic spinal cord (middle) from ataxic zebra A2. Astrocytic nuclei (arrows) and glial fibers near degenerated tracts. HE.
Pathological Findings

There were no gross lesions in any of the spinal cords from the three ataxic zebras (A1, A2, A3). In Weigert-stained sections, each cord showed bilaterally symmetrical demyelination in the lateral and ventral funiculi (fig. 2a). Both ascending and descending tracts appeared to be affected in the lateral funiculi, and tracts (descending) adjacent to the ventral median fissure were involved in ventral funiculi. Myelin sheaths varied in width, and some were dilated and empty (fig. 3). Bodian-stained sections showed a deficit of axons and many were disrupted or fragmented (fig. 4). Occasionally, there were lipid-laden macrophages within the dilated sheaths (fig. 5), and numerous astrocytic nuclei and glial fibers occurred in the degenerated areas (fig. 6).

There was adventitial fibrosis of small blood vessels throughout the ventral and lateral funiculi of white matter (fig. 7). This was most prominent in zebra A2, but occurred in A1 and A3 to a lesser degree.

The pattern and intensity of the degeneration of neuronal fibers were similar in each of the ataxic zebras. In the zebras in which the entire spinal cords were examined (A1 and A3) lesions were present throughout the cervical, thoracic, lumbar, and sacral regions. Dorsal funiculi and gray
columns, however, appeared normal in all of the cords. In zebras whose brains were studied (A2, A3), the degenerated tracts could be followed into the medulla oblongata; lesions rostral to this area were not evident.

There were abnormalities in the cervical vertebrae of one (A2) of two affected zebras. In zebra A2 the articular surfaces of the left caudal articular process of the third vertebra and the left cranial articular process of the fourth were larger than those on the right (fig. 8). There was also thickening of the articular process of the fourth vertebra with proliferation of bone around the lateral and cranial margins of the joint surface. Narrowing of the vertebral canals did not occur in either zebra.

No significant gross or microscopic changes were seen in comparable sections of the spinal cord (fig. 2b) or cervical vertebrae of the unaffected zebra (N1).

Fig. 8. Fourth cervical vertebra (C4) from ataxic zebra A2. Left cranial articular process (upper right) has proliferation of bone around articular margins and is larger than the right process (lower right).
Discussion

Because the clinical features of this ataxia in zebras so closely resembled the wobbler syndrome of horses, and since zebras are 'equidae', the changes in both conditions were compared. The lesions in the wobbler syndrome are malformations of the articular processes and Wallerian degeneration in the cervical spinal cord [3, 7, 8, 11]. A malacic focus (primary lesion) occurs in the cervical cord apparently as a result of subluxation of the abnormal vertebrae. Secondary degeneration of ascending tracts cranial to the primary lesion results and may be followed for several segments. Likewise, descending tracts degenerate caudal to the primary lesion, and these may extend to the lumbar segment. When the primary lesion is not readily apparent, it may be located by 'mapping' the cranial limit of the descending damage and the caudal limit of the ascending damage.

In the zebras, the spinal cord lesions did not have a Wallerian pattern. Vertebral arthropathy (fig. 7), reminiscent of the articular lesions of wobbler horses [14], was found in one zebra (A2), but detailed microscopic studies of its cervical cord and of cords of the others, did not show malacic foci. The degenerate ascending and descending tracts were not anatomically segregated but could be consistently followed together throughout the lengths of spinal cords available for study (table I). Correlations between the gait abnormalities and the lesions of specific tracts have been described in the wobbler syndrome [12]. The fact that functionally similar tracts were involved in the zebra spinal cords may explain the similar clinical signs of both conditions.

Further points of comparison include possible factors such as sex, breed, and heredity. The wobbler syndrome has its highest prevalence in the longer-necked breeds such as thoroughbreds and standardbreds, and there is a male predominance. Accordingly, Rooney [13] relates the vertebral arthropathies to abnormal stresses in the cervical columns of these long-necked animals and ascribes the predominance in males to their added muscle bulk and consequent additional strength of the head and neck region. Heredity has been suggested as a factor in the wobbler syndrome but never proved [6]. Although we cannot draw conclusions on sex ratios, there was no apparent trend for a male predominance in the zebra. Zebras are relatively short-necked 'equines', and our pathologic findings differ significantly from those of the wobbler syndrome, indicating that the pathogenesis of both conditions is not the same. Therefore, this ataxic condition of zebras should not be designated as the wobbler syndrome.
The specific cause of this diffuse myelopathy in zebras was not evident. There was no basis for an ischemic pattern [2], hence the adventitial fibrosis of vessels noted in all of the zebras was not considered to contribute to the spinal cord degeneration. Similar vascular changes caused by factors such as vertebral fractures and space-occupying lesions have been seen in wobblers and in other equine myelopathies [7]. Several causes were considered. Nutritional factors might be implicated. Copper deficiency has been reported to cause a demyelinating disease in lambs. This condition is called enzootic ataxia and has been compared with the wobbler syndrome of horses [10]. A deficiency of copper would not be expected in these zebras since all received ample supplements in their pelleted rations and had free access to copper in mineralized salt blocks. Proof of such a deficiency would depend on assays to determine serum and tissue copper levels.

Parasitic migration in the central nervous system was also considered. Parasites migrating aberrantly in the spinal cords have been reported to create wobbler-like signs in horses [9]. This was not so in the zebra condition as there were no parasites or parasitic remnants nor were there inflammatory changes that suggested infectious agents in the central nervous system.

The high incidence of ataxic sibling foals in this small zebra herd and our finding of a diffuse myelopathy with no apparent cause suggest that this condition is a familial disorder with primary degeneration of spinal cord white matter. Familial degenerations of the spinal cord do occur. Friedreich’s ataxia of children is described as a familial form of progressive ataxia. It is characterized by a degeneration of long, ascending and descending tracts of the spinal cord [2]. A degenerative myelopathy, possibly familial, has recently been described in aged German Shepherd dogs [1]. In several horses that clinically were wobblers DE LA HUNT A [4] has seen diffuse myelopathy similar to that in our zebras. He did not consider his animals true wobblers.

Proof of a familial ataxia in zebras must await future breedings between related animals that are possibly carrying genetic traits; complete clinical and pathological studies must be performed on any that are affected. Subsequent progeny from dams of our ataxic animals and from their normal daughters bred to an unrelated herd sire, Fred (moo-180), are clinically normal to date.

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References


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