I: The Healthy Spinal Cord

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1. Introduction

This first paper aims to review our current understanding of the structure and function of the normal equine spinal cord and its associated vertebral column, beginning with the latter. Because of their intimate relationship, an understanding of these two structures is vital for localising signs of spinal cord disease particularly as many spinal cord disorders involve changes in the vertebral column. Clinically relevant aspects of vertebral and spinal structure and function that are important to the neurological examination will be highlighted.

A plea is made here to use correct anatomical terminology as detailed in Nomina Anatomica Veterinaria. Importantly, “backbone” and “spine” are human terms and are not used; “vertebrae” and “vertebral column” are appropriate animal terms and are used. Also, the terms “dorsiflexion” and “ventroflexion” are hackneyed and incorrect. The correct terms to be used are “extension” (or “lordosis”) and “flexion” (or “kyphosis”) of vertebrae.

Finally, little will be said of ancillary aids, because these will be covered within the specific disorders presented in part II.

2. The Vertebral Column

The generally accepted vertebral formula for the domesticated horse is

\[ C = 7, \; T = 18, \; L = 6, \; S = 5, \; Ca = 15-21, \]

and that for the donkey is

\[ C = 7, \; T = 18, \; L = 5, \; S = 5, \; Ca = 15-17, \]

where C is cervical, T is thoracic, L is lumbar, S is sacral, and Ca is caudal (coccygeal).

However, many variations exist for all equids. In studies involving more than 250 equids, 7 cervical vertebrae was the rule. Of almost 100 domestic horses in these studies, 17 T vertebrae were found in 10%, 19 in 6%, and 18½ (the ½ indicating a transitional vertebra) in 1%. In 3% of horses there were only 5 L vertebrae and in 1% there were 5½. The sacrum varied more, with 5% having 4 vertebrae, 1% having 4½, 1% having 5½, and 7% having 1 or 2 fused Ca vertebral bodies forming the sacral plate. Variations in the remaining vertebrae need to be interpreted somewhat cautiously, depending on
one’s definition of “thoracic,” “lumbar,” and “sacral” vertebrae.\textsuperscript{5,6}

In a recent study of 36 Thoroughbred racehorses,\textsuperscript{6} the proportions of L and S vertebrae in various combinations were as follows: 5 L, 5 S: 3%; 5 L, 6 S: 28%; 6 L, 5 S: 61%; and 6 L, 6 S: 8%.

The T-L vertebral column is a relatively rigid structure\textsuperscript{7} with little movement in all 3 axes at most intervertebral complexes. The greatest degree of flexion and extension appears to occur at L6–S1, with some at T1–2 and most axial rotation and lateral bending at T2–T16. Townsend and Leach\textsuperscript{8} related differences in the intervertebral articulations (size, angulation, etc.) with differences in mobility of the 4 regions of the back, viz. T1–2, T2–16, T16–L6 and L6–S1. Recently, an in vivo study of mobility of the back was reported for 10 horses (average 16 hands high at withers). Average total dorsoventral movement at the middle of the back (T16) was 13 cm and average total left-right bending movement was 15 cm.

In concordance with the greater rigidity of the T16–L6 vertebral column, symmetrical or asymmetrical intertransverse articulations were found for at least one site in all of 36 racing Thoroughbreds and ankylosis of transverse processes in 28\% of horses.\textsuperscript{6} Physeal closure in these T-L vertebral bodies occurred between 5 and 7 years of age. Also, a “ventral crest” was referred to without further explanation as spanning 4–8 vertebral bodies from T16–L4.\textsuperscript{6} These may well represent ventral projections of the cranial and caudal physeal plates seen on T-L vertebrae in several breeds of equids that can appear to fuse at gross post mortem examination, but radiographically show incomplete fusion (Figs. 1 and 2). Other degenerative and probably traumatic pathologic articular changes were found in these 36 active racehorses.\textsuperscript{10,11} Half the horses had stress fractures of the laminae of the arches of the vertebral canal. The severity of these fractures was positively related to the severity of osteoarthritis in the articular processes in the same horses.\textsuperscript{10} Impingement of dorsal spines and transverse lumbar processes were present in more than 90\% of horses and degenerative changes (osteoarthritis) was present in almost all T, L, and L-S articular processes; some of these changes were severe.\textsuperscript{11}

The intervertebral disks at T1–2, T2–3, and L6–S1 were found to be thicker by 215\%, 124\%, and 131\%, respectively, compared with the average thickness of the other T-L disks.\textsuperscript{8} In the 96 T-L intervertebral disks examined there were only 3 (T8–11) in one horse that had a gelatinous center, possibly a nucleus pulposus.

There are consistently 7 cervical vertebrae in mammals, and very occasionally rudimentary cervical ribs a few centimeters in length are present. I have seen 2 horses with 6 neck vertebrae cranial to a complete set of first ribs (Figure 3). Because the entire spinal cord and vertebral column were not fully examined, it must remain likely that these structures represent ‘cervical ribs’ with thoracicalization of the seventh cervical vertebra in both cases.

Considerable morphologic variation exists in cervical vertebrae even within breeds,\textsuperscript{12} and 3 of these variations are notable. Asymmetrical variations in the bony borders for the second cervical nerve root in its neurovascular bundle at the cranial region of the
arch of the axis should not be confused with bone disease. There can be simply a notch, or partial separation into a foramen or a complete foramen on one or both sides; this being also recognised at the T-L intervertebral sites. Likewise, the radiographically variable, serpentine, vascular channel in the dorsal spine of the axis and the separate centers of ossification at the caudal end of the ventral laminae of C6 should not be mistaken for fractures. The most common major variation in cervical vertebrae is transposition of one or both ventral laminae of the transverse processes of C6 onto C5 or, most often, onto C7. This was found in 2 of the last 20 sets of cervical radiographs taken at the University of Edinburgh, Easter Bush Veterinary Centre. These reasonably frequent anomalies are very important to identify individual cervical vertebrae correctly on radiographs, at surgery and at necropsy examination.

Finally, quantitative morphological variations have been identified in C3 to C7 in 4–23-mo-old Thoroughbreds. Regarding the kinematics of the adult equine head and neck, at least in cadaver specimens, most dorsoventral and some axial movement occurs at the atlantooccipital joint but most of the axial movement occurs at the atlantoaxial joint. Excluding atlantooccipital (head) movement, more dorsoventral and lateral movement occurs in the caudal than in the cranial neck, especially at C6–7 (Figs. 4–6). Neonatal foals have greater range of motion of joints so it perhaps is not surprising that cadaver necks from foals (<12 months) have approximately 20% more motion around all 3 axes than horses older than 2 years. Current work using image analysis will help provide in vivo data for cervical kinematics in adult horses (Licka T, 1999, pers. comm.).

Morphologically, physeal closure in cervical vertebrae is very delayed compared with limb longbones; the cranial physes of C3–7 are closed by 3 years, whereas the caudal physes of C2–7 are present and discontinuous by 6–9 years but absent by 12 years of age. Cervical intervertebral disks are composed of fibrocartilage but do undergo ageing changes. In addition, fibrocartilage can be seen to bulge into the vertebral canal in normal horses. A more recent extensive study of 103 cervical vertebral columns from animals 42 weeks gestational age to 23 years of age revealed considerable, age dependant, degenerative changes in cervical disks in the absence of clinical signs. In some cases there was necrosis of fibrocartilage that could be detected radiographically (Fig. 7).

3. Spinal Cord Structure

A. Gross

The spinal cord in a 500-kg horse is almost 2 m long. This can be divided into 5 functional sections as shown in Table 1.

The lengths of these spinal (and corresponding vertebral) sections are important in explaining why focal compressive spinal cord lesions in horses less often result in evidence of gray matter damage such as profound weakness, reflex loss and muscle atrophy, and sensory loss compared with evidence in small animals. For example, a C7–T1 compressive lesion 2 cm long would potentially damage 6% of the

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**Fig. 4.** Mean ranges of maximal cervical dorsoventral movement at each intervertebral site and at the atlantooccipital site (AO). Data are given as percentages of total head and neck movement (lighter bars) and as a percentage of total neck movement alone, excluding AO movement (full bars, except the AO bar). (Modified from Clayton and Townsend.)

**Fig. 5.** Mean ranges of maximal cervical axial movement at each intervertebral site and at the atlantooccipital site (AO). Data are given as percentages of total head and neck movement (lighter bars) and as a percentage of total neck movement alone, excluding AO movement (full bars, except the AO bar). (Modified from Clayton and Townsend.)

**Fig. 6.** Mean ranges of maximal cervical lateral movement at each intervertebral site and at the atlantooccipital site (AO). Data are given as percentages of total head and neck movement (lighter bars) and as a percentage of total neck movement alone, excluding AO movement (full bars, except the AO bar). (Modified from Clayton and Townsend.)
gray matter in the brachial intumescence of a horse and would be most unlikely to cause localizing signs noted above. In a standard-sized dog with a 40-cm-long spinal cord, a compressive lesion of, say, 1 cm would potentially damage 15% of the gray matter in the brachial intumescence and most likely cause such localizing signs.

Because the spinal cord ends at S2 in the horse, there is the well utilised opportunity to sample cerebrospinal fluid (CSF) from the reasonably voluminous L-S cistern. However, the S3–4 segments potentially can be damaged during this procedure. Rarely is this the case however and a complication of bladder paresis due to sacral parenchymal hemorrhage must have occurred at no more than 1 in 1000 in the author’s experience. It is almost impossible to penetrate free-floating S1–4 nerve roots at this site unless there is violent movement of horse or needle with the spinal needle bevel acting as a knife; the author has not known for this to occur.

B. Subgross

The sub-gross anatomy of the spinal cord has been well-studied and a summary of the relationship of cross-sectional areas of spinal cord gray matter, white matter and sub-dural (CSF) space is given in Fig. 8. This gives an indication of how large the vertebral canal must be to contain the spinal cord at each variably-sized section. Although the absolute values for cross-sectional areas given by Braun (Fig. 8) are smaller than measurements taken from a spinal cord from a 500-kg horse measured by the author, the relative proportions do appear to be useful (Fig. 8). It is probably axiomatic that the vertebral canal diameter will depend upon the size of the dural compartment or sac (Fig. 8). Also, the amount of sub-arachnoid/dural space (CSF) will depend on the amount of dorsoventral and lateral movement at each site (see above).

4. Spinal Cord Physiology and Biochemistry

Much of what is understood about the function of tracts and regions of the equine spinal cord has been assumed from work in other species and derived from observed syndromes caused by naturally occurring equine neurological diseases. For the purpose of this review, three findings will be mentioned here as having clinical relevance.

Firstly, the pyramidal (cerebrospinal) tract in mammals is the direct motor pathway from the motor cortex in the forebrain to motor nuclei of cranial nerves in the brainstem and lower motor neurons in the ventral gray columns of the spinal cord. In the horse this tract certainly ends cranial to the brachial intumescence and probably in the cranial cervical segments. Major lesions in this pathway alone (e.g. a motor cortex lesion) result in little or no permanent gait abnormality.

An interesting involvement of spinal afferent fibers in respiratory control has been shown in ponies. One month after dorsolateral spinal lesions at L2 were created (mainly sectioning the dorsal spinocerebellar tracts) there was attenuation of exercise hypocapnea (PaO2 not measured) recorded at the onset of treadmill exercise compared with pre-lesion measurements. It is thus possible that minor spinal lesions may influence performance through effects on respiration.

An important role for proprioceptive afferent input from the neck via the first 3 cervical dorsal nerve roots appears to have been overlooked in equine neurology. Local anesthetic block of these nerve roots in 10 macaque monkeys and baboons consistently caused severe defects in balance, orientation and coordination, mimicking the vestibular signs recorded in monkeys with bilateral inner ear ablation (labyrinthectomy). Post mortem confirmation

**Table 1. Functional Sections of the Equine Spinal Cord**

<table>
<thead>
<tr>
<th>Section</th>
<th>Length (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1–5</td>
<td>55</td>
</tr>
<tr>
<td>C6–T2</td>
<td>33</td>
</tr>
<tr>
<td>T3–L3</td>
<td>88</td>
</tr>
<tr>
<td>L4–S4</td>
<td>18</td>
</tr>
<tr>
<td>S5–Ca+</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>200</td>
</tr>
</tbody>
</table>
of the site of deposition of the anesthetic solution was confirmed in 4 animals. Bilateral neurectomies of the C1–dorsal (sensory) roots in 2 monkeys produced the same syndrome that improved over 2 weeks. The same temporary clinical syndromes of symmetrical vestibular signs has been produced in more than 20 ponies by local anesthesia of the cranial dorsal nerve roots (C. Frigast, Denmark, 1999, pers. comm.). Correct deposition of the anesthetic solution around the dorsal nerve roots (and not around the spinal cord) was confirmed with radiography and at post mortem dissection.

Finally, it is probably of some clinical importance that, both biochemically and morphologically, foals’ spinal cords appear to mature before birth; these findings are consistent with the strong gait demonstrated by foals a few hours old compared with other domestic species.

5. Neurological Examination for Spinal Cord Disease

A. Neurological Testing in the Physical Examination

The procedure for neurological examination of the adult horse is based on information given in references 24–30. Frequently it will not be clear whether a horse suspected of having a spinal cord disorder indeed does have one until neurological evaluation has been made. This is particularly so for mild gait abnormalities and following some injuries when orthopedic and neurological disease are both possible and suspected.

The physical examination should incorporate several basic neurological tests to assist the practitioner decide whether or not a neurological disorder exists. Table 2 indicates some of the observations that can easily be included within a routine physical examination that will help indicate if a neurological abnormality is present in the great majority of cases in which the signs are not intermittent.

B. Neurological Examination Procedure

After evaluation of the head for evidence of brain and cranial nerve disorders, an evaluation of the neck, forelimbs, trunk, hindlimbs, tail and anus is then undertaken. Evidence of bony and muscular asymmetry, localized sweating, focal muscle atrophy, decreased pain perception and localized painful responses should be searched for and documented. Areas of sweating and decreased sensation (hypalgesia), and depths and diameters of muscle masses suspected as being atrophied should be measured accurately. When marked abnormalities of gait are evident it is relatively easy to determine which limbs are affected. The degree of limb involvement and the characteristic of any abnormality is recorded in order to determine the site and extent of any neurological lesion (Table 3).

For the most part, neurological gait abnormalities involve degrees of weakness and ataxia. Weakness may predominantly involve flexor or extensor muscle groups and ataxia can be characterized as having components of decreased range of joint movement (hypometria or spasticity), and increased range of joint movement (hypermetria or high striding). Extensor weakness in a limb is best evaluated by observing for muscle trembling, buckling on a limb when turning and the ease in which the patient can be pulled to the side by the tail, both while standing still and while moving (Fig. 9). Flexor weakness may be more evident as dragging of a toe and a low foot flight, particularly while turning. Subtle degrees of weakness in the thoracic limbs may be accentuated by performing a hopping test wherein one forelimb is held up and the horse made to hop laterally away from the examiner on the other forelimb (Fig. 10). A horse with extensor weakness often will buckle on an affected limb. Pelvic limb and/or thoracic limb weakness can be detected by attempting to pull on the halter and tail at the same time. This is particularly useful if there is asymmetry in the degree of weakness. Normal alert horses resist such pulling whereas a weak animal is easy to pull to the side.

Mild degrees of ataxia can be detected by performing additional postural maneuvers. Considerable time usually is spent in performing serpentine maneuvers, circling wide and tight (Fig. 11), elevating the head while walking the patient on a flat (Fig. 12) and on a sloped (Fig. 13) surface, turning tightly upon stopping abruptly from a trot and backing. These maneuvers alter visual, gravitational, vestibular

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Postural Deficits</th>
<th>Paresis</th>
<th>Ataxia</th>
<th>Hypometria</th>
<th>Hypermetria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal cord—UMN</td>
<td>++ ++ ++ ++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vestibulospinal</td>
<td>++ ++ ++ ++ ++</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Spinocerebellar</td>
<td>++ ++ ++ ++ ++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinal cord—LMN</td>
<td>++ ++ ++ ++ ++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>++ ++ ++ ++ ++</td>
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</table>

NOTE. Characteristics of many musculoskeletal disorders are included for comparison.
UMN, upper motor neuron; LMN, lower motor neuron; ○, not usually expected; +, mild if present; ++, usually present; ++++, quite characteristically present.
*Due to weakness.
†Mechanical.

Table 3. Prominent Gait and Postural Abnormalities Present with Neurologic Lesions at Different Locations

Table 2. Basic Tests That Can Be Included in a Physical Examination to Assist in Detection of a Spinal Cord Disorder

<table>
<thead>
<tr>
<th>Test Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symmetry of neck, trunk, and limbs</td>
</tr>
<tr>
<td>External thoracolaryngeal (slap) reflex</td>
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<tr>
<td>Tail and anal tone</td>
</tr>
<tr>
<td>Anal reflex</td>
</tr>
<tr>
<td>Examination of rectum and bladder</td>
</tr>
<tr>
<td>Postures adopted at rest</td>
</tr>
<tr>
<td>Gait at walk and trot</td>
</tr>
<tr>
<td>Gait while turning</td>
</tr>
<tr>
<td>Faster gaits</td>
</tr>
</tbody>
</table>

Table 4. Muscular Dystrophy and Atrophy

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Muscle Groups Involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal muscle</td>
<td>Swayback</td>
</tr>
<tr>
<td>Lumbar muscle</td>
<td>Hindlimb weakness</td>
</tr>
<tr>
<td>Pelvic muscle</td>
<td>Pelvic limb weakness</td>
</tr>
</tbody>
</table>

Table 5. Clinical Signs of Spinal Cord Disease

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical spinal cord</td>
<td>Head and neck weakness</td>
</tr>
<tr>
<td>Thoracic spinal cord</td>
<td>Hindlimb weakness</td>
</tr>
<tr>
<td>Lumbar spinal cord</td>
<td>Pelvic limb weakness</td>
</tr>
</tbody>
</table>

Table 6. Spinal Cord Lesions

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Lesion Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical cord</td>
<td>Compression</td>
</tr>
<tr>
<td>Thoracic cord</td>
<td>Compression</td>
</tr>
<tr>
<td>Lumbar cord</td>
<td>Compression</td>
</tr>
</tbody>
</table>

Table 7. Spinal Cord Lesions and Clinical Signs

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Lesion Type</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Compression</td>
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</tr>
<tr>
<td>Thoracic cord</td>
<td>Compression</td>
<td>Hindlimb weakness</td>
</tr>
<tr>
<td>Lumbar cord</td>
<td>Compression</td>
<td>Pelvic limb weakness</td>
</tr>
</tbody>
</table>

Table 8. Spinal Cord Lesions and Muscular Dystrophy

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Muscle Groups Involved</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Lumbar spinal cord</td>
<td>Pelvic limb weakness</td>
</tr>
</tbody>
</table>
lar and proprioceptive input to the nervous system such that any subtle sensory or motor deficit can become more clearly expressed. The overall severity of any gait abnormality in each of the four limbs can be graded 1 through 4, as subtle, mild, moderate or severe.

With the neurological examination completed the examiner may be able to decide if and where any possible lesions exists. If this is not clear then it is often worthwhile returning to the patient and performing an even more critical evaluation. With a very fractious or a very excited horse suspected of...
having a neurological abnormality involving the limbs, exercise such as lunging or running on very soft going for 20 minutes can be undertaken and then a re-evaluation made.

When lameness is present or is suspected, possibly interfering with interpretation of a horse's gait and posture, then appropriate regional analgesia or short acting systemic analgesia (e.g. using synthetic opioids), may be used. In more chronic cases, nonsteroidal anti-inflammatory drugs may be given at a relatively high dose for several days (or weeks) and then the horse's gait can be re-evaluated, when lameness often will be reduced.

Equine practitioners do find cases for which there is some indication of spinal cord involvement but no definitive proof. These cases usually are suspected of suffering from a painful musculoskeletal disorder, a peripheral neuromuscular spastic disorder, a behavioral problem (belligerency), laziness or back disease. Such patients may show one or more of the signs listed in Table 4. Other forms of frantic behavior have been associated with a strong suspicion of exposure to nettles or poison ants, but in these situations the signs usually abate with time.

With horses that demonstrate a mild or unusual gait or postural abnormality, emphasis often will be on detecting evidence of spinal cord, peripheral nerve and muscle disease and distinguishing such evidence from signs resulting from primary orthopedic disorders.

C. Points to Emphasize in Cases of Spinal Cord Disease

i. The “Slap Test”

The thoracolaryngeal response (“slap test”) is a useful part of the complete neurological examination of horses suspected to have involvement of the vagal or recurrent laryngeal nerves or cervicothoracic spinal cord.31 The test can be performed in cooperative horses by palpating the dorsal and lateral laryngeal musculature while simultaneously slapping the contralateral dorsolateral thoracic (saddle) region, from the cranial withers to near the last rib (Fig. 14). The hand slap should be performed during expiration and examiners may find it easier to perform a double slap, palpating for a double movement of the laryngeal musculature and/or cartilages. If there is difficulty in interpretation of this test, observing the larynx via an endoscope while performing the test may be necessary.

This response is not consistently absent in horses with cervical vertebral malformation or other forms of cervical spinal cord disease (wobblers) and may inexplicably be absent in some apparently normal horses. Depression or absence of the reflex on the left side must be taken as strong evidence for the presence of recurrent laryngeal neuropathy or prior laryngeal surgery.32,33 Exercising the horse will be necessary to confirm any clinical problem arising from laryngeal paralysis. Bilateral absence of the palpable response in the absence of other signs of laryngeal or cervicomedullary disease must be interpreted cautiously particularly in excitable horses.

![Fig. 13. Walking this ataxic horse up an incline with the head extended exaggerates the hypermetria in the thoracic limbs.](image)

![Fig. 14. The thoracolaryngeal response (slap test) can be performed by slapping the cranial dorsolateral thorax while palpating the dorsolateral laryngeal musculature on the opposite side for movement.](image)

**Table 4. Syndromes Wherein an Organic Spinal Cord or Vertebral Column Lesion May Be Suspected but Usually Not Proved**

<table>
<thead>
<tr>
<th>Prominent toe dragging</th>
<th>Shivering</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent, unusual lameness</td>
<td>Stringhalt-like movements</td>
</tr>
<tr>
<td>Prominent sinking with dorsal lumbar pressure</td>
<td>Other spastic movements</td>
</tr>
<tr>
<td>Throwing to the ground when a saddle is applied</td>
<td>Extreme difficulty in getting up</td>
</tr>
<tr>
<td>Rearing violently when first ridden</td>
<td>Lying down a lot</td>
</tr>
</tbody>
</table>
ii. Cervical Reflexes
The local cervical and cervicofacial reflexes can be useful to help confirm the presence and sometimes the location of a cervical lesion. The former is the contraction of the cutaneous coli (neck) muscle in response to tapping the neck with a blunt probe. The sensory input for this reflex is segmental but the motor output from the CNS is not completely clear but includes ventral spinal nerve roots, especially C6. The second of these reflexes is twitching of all facial muscles including the cutaneous faciei (facial sub-cuticular) muscle in response to the same cervical stimulus as for the first reflex. The same segmental cervical sensory input likely applies to this reflex. However, both the sensory and motor pathways may be more complex as there is an anastomotic connection from the C2 nerve (and subsequently the first 6 cervical nerves via the transverse cervical nerve) and a cervical branch of the facial (cranial nerve VII) nerve. The fact that this anastomosis could contain sensory or motor or both types of fibers makes accurate interpretation of this reflex enigmatic.

Finding depressed and particularly asymmetrical local cervical and cervicofacial reflexes can be useful in localizing a cervical spinal cord lesion. Because of the incomplete understanding of these reflexes interpretation may need to be as imprecise as “consistent with a caudal cervical lesion” or “consistent with a cranial cervical lesion.”

iii. Cutaneous Trunci Reflex and Cutaneous Sensation
The cutaneous trunci reflex (incorrectly referred to as the panniculus reflex) is a robust reflex in horses. It consists of contraction of the cutaneous trunci muscle in response to tapping the lateral trunk with a blunt probe. The sensory input is segmental through dorsal thoracic nerve roots passing cranially through the spinal cord to reach the motor neurons in the cranial thoracic gray matter to reach the cutaneous trunci muscle via the lateral thoracic nerve. This reflex can be useful in delineating the cranial extent of a thoracic spinal cord lesion particularly when such a lesion is asymmetrical.

Rarely, a degree of hypalgesia can be detected caudal to the cranial extent of a region of cutaneous trunci hyporeflexia. This is seen only with severe thoracic spinal cord disease.

Evaluating for hypalgesia over the trunk, as elsewhere, should be performed with a two-pinching test. This is performed by pinching the skin into a fold, inserting the fold into the jaws of a strong hemostat or needle holder and after the patient has settled to this, a brief, sharp squeeze is applied to elicit a behavioral response.

iv. Neck pain
It is difficult to interpret an apparent reluctance of a patient to move the neck passively or actively in any direction as indicating neck pain or stiffness. On the other hand, a horse that will not lower its head to eat usually is quite evidently compromised, usually because of mechanical or painful disruption to flexion of the caudal cervical vertebrae (Fig. 15) or extension of the atlanto-occipital and/or the atlantoaxial joints.

v. Sweating
Identifying the presence of well-delineated regions of cervical and thoracic sweating can be useful in localizing a spinal cord lesion. Such areas can represent focal sympathetic denervation (decentralization) of the vasculature in the skin, resulting in increased circulating epinephrin stimulating sweat glands. However, care must be taken in interpreting patchy sweating that is not well delineated. Very asymmetrical patchy sweating can occur in horses that are excited or distressed, particularly when in a draughty trailer or stall, without a sympathetic lesion being present.

vi. Gait and Posture
As noted above, when spinal cord disease is suspected, considerable time should be spent in evaluating gait and posture of the patient (Table 3). Rather than manually placing limbs in abnormal positions to evaluate conscious proprioception, it appears more reliable to maneuver the horse rapidly (say in a circle) and stop the maneuver abruptly. This often results in an initial awkward placement of a limb and then the examiner can determine how long the horse leaves the limb in such an abnormal posture to determine the presence or not of a conscious proprioceptive deficit.

To determine the presence of weakness in the limbs of a horse suffering from spinal cord disease the three most useful tests are the tail pull (Fig. 9), the tail and halter pull and thoracic limb hopping (Fig. 10). Pulling the tail while the patient remains static initiates an extensor (patellar or quadriceps) reflex. This reflex is poor with lower motor neuron disease at the level of L3–4 and the patient will demonstrate weakness while standing still (hypotonia) as well as voluntary extensor weakness while moving. In contrast, a horse with an upper motor neuron lesion (wobbler) will have good resting muscle tone and be difficult to pull to the side in a singular movement while standing still. However, such a patient will be easily pulled to the side while walking (Fig. 9). This demonstrates voluntary extensor weakness but the presence of intact or even hyperactive extensor reflexes in the pelvic limb.

Pulling on a lead rope and the tail simultaneously while circling the horse around the examiner is a postural reaction that also evaluates voluntary extensor strength and in addition can exaggerate a patient’s tendency to pivot on a hindlimb and to maneuver limbs in an ataxic fashion. Flexor weakness leads to the patient not flexing the affected limb well and thus dragging the toe on the ground. A worn toe will result. Some neurologically normal...
horses will “toe-drag”; many of these will have orthopedic disease.

A horse that has extensor weakness in a thoracic limb often will tend to tremble on the limb while the opposite thoracic limb is held up on initiation of the hopping test. It also will have difficulty in hopping to the side when pushed with the examiner’s shoulder (Fig. 10).

Evaluation of horses while being walked across kerbs has not proven to be a useful test of proprioceptive dysfunction. Normal horses, particularly if distracted, often will stumble and those that are quite weak and ataxic but moving cautiously often can maneuver such obstacles. In the author’s experience blindfolding a horse suspected of suffering from spinal cord disease usually has not added anything substantial to the neurological evaluation. Normal horses react in different ways, from extremes of excitement to calmness and the subsequent movements they make depends on this behavioral response. In contrast, signs of dysmetric ataxia and loss of balance will be markedly exacerbated when a blindfold is applied to a horse suffering from vestibular or occasionally from spinocerebellar disease.

After the precise type of gait abnormality present has been determined (Table 3), the most likely site of an acute spinal cord lesion and the pathways involved frequently can be accurately defined, with some exceptions. With peracute lesions, particularly those of an inflammatory nature and those with soft tissues compressing the spinal cord (such as with caudal cervical synovial cyst formation), resulting signs can wax and wane quite dramatically over periods of hours to days. Such signs usually stabilize with subacute to chronic lesions.

On the other hand, a horse with chronic spinal cord disease may show quite different neurological signs. For example a horse that has suffered a single insult of cervical spinal cord compression a year before examination may have an unusual, perhaps hypermetric, mild ataxia in the pelvic limbs with no evidence of weakness. There may be no signs in the thoracic limbs save for a questionable response to hopping.

D. Neurological Examination of Foals

The neurological evaluation of foals is very similar to that of adults but several developmental landmarks should be borne in mind.23,26,35,36

The spinal reflexes are hyperactive in newborn foals and the patellar reflex, cranial tibial and gastrocnemius tendon reflexes are performed easily. Up to one month of age there are normal, strong, crossed extensor reflexes in the thoracic (Fig. 16) and pelvic limbs. In addition, an extensor thrust reflex is obtained, at least in very young foals, by rapidly overextending the animal’s toe while the limb is
already in extension. This results in forceful extension of the limb.

In addition to the evaluation of gait and reflexes in nonrecumbent adults, other postural reactions can be performed in foals and are of most benefit in detecting subtle proprioceptive and motor pathway lesions when the gait is normal. These include wheelbarrowing the patient to make it walk on just the thoracic limbs, hopping it laterally while supporting weight on just the left then the right thoracic limb in turn and hemistanding and hemiwalking the patient by making it stand and then move laterally on both left, then both right limbs. Spinal cord lesions cause postural reaction deficits on the same side as the lesions. Lesions involving the proprioceptive and motor pathways to the limbs result in an extremely slow or absent hopping response in that limb.

E. Objective Neurophysiologic Testing

Neurological examinations in horses have tended to be primarily subjective however recent publications addressing the use of objective electrodiagnostic measurements in the horse are available. These include the evaluation of the thoracolaryngeal reflex, recurrent laryngeal nerve conduction velocities, and sensory nerve conduction velocities. Needle electromyography can be a useful diagnostic adjunct in defining the extent of any denervation but probably should be delayed for 3–4 weeks when repeatable denervation potentials will be recordable.

6. Conclusion

Variations in number and shape of vertebrae as well as prominent performance-related and ageing changes in vertebrae need to be understood to interpret disease states more accurately. More objective measurement of vertebral canal and myelographic diameters in different sized horses will definitely improve identification of spinal cord compression. The roll of cranial cervical afferent input to the vestibular system and the clinical syndromes seen with lesions to cranial cervical nerves need to be better understood. Finally, critical documentation of localizing signs such as hypalgesia, hyporeflexia and sweating over the neck and trunk is often difficult but can be extremely useful in localizing spinal cord disease.

References


