Interpretation of Signs of Gait Abnormalities; Orthopedic or Neurologic?

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In a recent AAEP study (Keegan et al, 2008), experienced equine ppractitioners agreed on the existence of lameness in the forelimbs and hind limbs by only 25% and 15% above chance, respectively! The 95% confidence interval for a single AAEP lameness score [grades 1 to 5] was +/- 1.5 grades! Thus if a horse showed a mild lameness of 1 grade, experienced practitioners would grade the severity as anything from 0 to 2.5 out of 5! If we then add in degrees of neurologic gait abnormalities it is most likely that there will be even greater variability in grading the abnormalities. Indeed this is where some of the newer techniques of neuromuscular assessment such as magnetic motor evoked potentials [mMEP] (Nollet *et al*, 2008) and quantitative electromyography [Q-EMG] (Wijnberg *et al*, 2004) may improve our clinical acumen. Notwithstanding, distinguishing orthopaedic from neurologic gait abnormalities is thwart with errors of interpretation. This paper attempts to give a basis for the differences in gait seen with orthopaedic and neuromuscular disorders. This discussion attempts to more clearly define the characteristics of neurological gait abnormalities that helps distinguish them from gait changes resulting from orthopaedic disorders; of course both can occur together.

Gait and Postural Abnormalities

Musculoskeletal and painful disorders causing gait and postural disorders are far more commonly encountered in large animals than those involving just the nervous system [Figure 1]. Thus, two observations to be made when evaluating gait and posture are firstly, which limbs are abnormal, and secondly, is there evidence of lameness suggesting a musculoskeletal or painful cause for any gait abnormality?

Figure 1: Heavy patients with various neuromusculoskeletal disorders can have difficulty rising. Such patients, especially ruminants and pigs that adopt a dog-sitting posture for several seconds to minutes while getting up, usually have lesions caudal to the thoracic limbs and T3. However adult ruminants are seen to rest in the field in such a posture without having an overt neuromusculoskeletal explanatiom. Also, occasionally patients such as this horse suffering from mild caudal cervical spinal cord compression caused by CVM may also adupt and maintain such postures. This particular horse also was a tongue sucker .

The essential components of neurologic gait and postural abnormalities are **paresis** and **ataxia** and considerable effort needs to be made to define the presence, characteristics and severity of these findings when evaluating patients suspected of suffering neurologic disorders. In my experience, the most frequent mistake made in observing for, describing and teaching about characteristics of gait and postural abnormalities are failure to describe accurately what is seen before interpreting what any abnormalities might represent. Thus, we do not see ataxia, weakness, lameness and pain, but we do see irregular and unpredictable foot placement, toe dragging, head nodding and abnormal postures that can be interpreted as such.

Paresis or weakness can be defined as poor ability to initiate a gait, to maintain a posture, to support weight of the body or its parts, and to resist gravity. Often it can be determined that such weakness is predominantly involving extensor or flexor muscle groups, or both. As a generality, extensor, anti-gravity weakness is most indicative of **lower motor neuron paresis**. This is often seen as a bouncing

gait with short stride length, trembling, buckling and bunny-hopping, and a lowered neck carriage. This results in an apparent weight bearing lameness due to the exaggerated head nodding, particularly when it is asymmetric. For clarification, bunny-hopping is a non-specific gait characteristic, more often seen with musculoskeletal disorders, but when representing paresis reflects the added weight support offered when two limbs are used together.

In distinction, **upper motor neuron paresis** often is seen as a delay in initiating movement, therefore usually involving flexor muscles. Thus, a slow onset of the protraction or swing phase and a swinging, longer stride with decreased joint flexion [degrees of hypometria], are characteristics of upper motor neuron paresis. On this basis, with a prominent C_6-T_2 lesion involving gray and white matter in a smaller patient, one can see a very characteristic gait whereby there is a short-stride, bouncing gait in the thoracic limbs and a slower, long-stride with toe dragging in the pelvic limbs. This can be called a two-engine gait, with lower motor neuron, extensor paresis in thoracic limbs and upper motor neuron, flexor paresis in pelvic limbs.

In addition for observing for signs of abnormal gait and posture indicative of weakness, three most useful postural reaction tests for determining the presence of weakness in the limbs of a horse suffering from spinal cord disease are the tail pull, the tail and halter pull and thoracic limb hopping. Pulling the tail while the patient remains standing initiates an extensor, quadriceps contraction, mimicking performance of a patellar reflex. This reflex is poor when there is a lower motor neuron lesion at the level of L_3-$_5$ and therefore the patient will demonstrate weakness in resisting a tail pull while standing still as well as voluntary weakness while moving. In contrast, a wobbler horse with an upper motor neuron cervical lesion will have good resting muscle tone and be difficult to pull to the side in a singular movement while standing still, but is easily pulled to the side while walking. The first example demonstrates depressed extensor reflexes in the pelvic limbs while the wobbler demonstrates intact or even hyperactive extensor reflexes in the pelvic limbs in the face of voluntary, upper motor neuron weakness. The tail pull test is very useful in detecting extensor weakness in the pelvic limbs but often it is performed far too vigorously; it is not a contest between examiner and patient! It is best to apply constant lateral tension to the tail and determine what voluntary pull the patient exerts against that tension while it is weight bearing on the nearest limb.

The tail and halter pull test is performed by pulling on a lead rope and on the tail while circling the horse around the examiner and is testing a postural reaction that also evaluates voluntary strength. In addition it can exaggerate a patient's tendency to pivot on a hindlimb, thus demonstrating either flexor weakness or hypometria, and to maneuver limbs in an ataxic fashion. Again, ease in pulling the patient to the side during circling occurs because of weakness resulting from descending upper motor pathway involvement or a lesion that involves ventral horn gray matter level with the limb, or the peripheral nerves or muscles constituting a lower motor neuron lesion. With the latter, extensor weakness is often profound and it is easy to pull such a patient to the side while it is standing still and while circling. In contrast, a weak animal with a lesion of the upper motor pathways usually can reflexly fix the limb in extension when pulled to one side by the halter and tail, whereas while circling, the patient does not have the voluntary motor effort necessary to resist the pull.

Hopping a patient laterally on one thoracic limb while the pelvic limbs are free to move may reveal that a horse is weak by a tendency for it to tremble on a thoracic limb when the opposite thoracic limb is held up to initiate the hopping test. Such a patient will also have difficulty in hopping to the side, and may stumble, when pushed away with the examiner's shoulder.

Flexor paresis often is evident when an animal drags its limbs, has worn hooves, and has a low arc and long swing phase to the stride. When an animal bears weight on a limb demonstrating extensor weakness, the limb often trembles and the animal may even collapse on that limb because of lack of support. While circling, walking on a slope, and walking with the head elevated, an animal frequently will stumble on a limb having extensor weakness and knuckle over at the fetlock.

With severe weakness in all four limbs, but no ataxia and hypometria, neuromuscular disease must be considered. Profound weakness in only one limb is suggestive of a peripheral nerve or muscle lesion in that limb. Weakness occurs with descending, upper motor neuron pathway lesions in the brainstem and spinal cord, and is present in the limb[s] on the same side and caudal to the lesion. A patient with peracute peripheral vestibular syndrome may appear weak in the limbs on the same side as the lesion because of the decreased extensor tone and tendency to fall in that direction, and the increased extensor tone in the contralateral limbs.

Ataxia is a term that, by its Greek derivative, means a lack of order or an inconsistency, and in this context is a proprioceptive dysfunction causing abnormal rate, range and force of movement and placement of the limbs and other body parts including head, neck, trunk and even at times, the eyes. What the examiner must see to interpret as ataxia is irregular and mostly unpredictable movement and placement of the limbs, head, neck, or trunk. To accomplish this, the patient is observed while standing still, walking, trotting, turning tightly and backing up, and while the patient moves in a serpentine path with the head held elevated and while moving on a slope. The best way of accomplishing the latter maneuvers is to walk backwards in a zigzag manner while holding the lead rope high to extend the patient's head and neck. The aim is to alter the intended direction of the patient's limbs while they are in protraction by turning the lead abruptly such that there must be a change in direction of each foot to be placed in the site the examiner intends for it to be placed. Some horses will not obligingly turn in tight or even large circles for examination. With practice, circling can be accompanied best by walking the horse forwards then start to turn in one direction slowly making the turn slightly tighter as the examiner moves from in front of the horse to level with the shoulder to level with the middle of the trunk, while coaxing the horse by flicking the rump with the free end of the lead rope. This way the patient turns around the examiner, not the examiner around the horse. Essentially, these maneuvers comprise the postural reaction tests for large animals. Thus, input to the upper motor centers is altered through changes in many modalities, including the visual horizon, vestibular stimulation and neck and limb proprioception that are synthesized into refined motor system signals. Subtle neurologic abnormalities, which may be compensated for under conditions of normal gait, are exaggerated during these maneuvers. It is important for the examiner to observe the patient performing these maneuvers from a distance and also to make the patient perform them oneself. Ataxic movements can be seen as irregular and mostly unpredictable foot flight and placement. To detect subtle asymmetry in limb protraction and the length of stride it can be useful to walk parallel to, or behind the animal, matching step for stride. An ataxic gait sometimes is most pronounced when an animal is moving freely in a paddock especially when attempting to stop abruptly from a trot or canter when the limbs may be wildly adducted or abducted.

Three descriptors are often used to identify the inconsistent movements that comprise ataxia. **Hypermetria** is used to describe a lack of direction and increased range of movement, and is seen as an overreaching of the limbs with excessive joint flexion. Hypermetria without paresis is characteristic of spinocerebellar and cerebellar disease. **Hypometria** is seen as stiff or spastic movement of the limbs with little flexion of the joints, particularly the carpal and tarsal joints. This generally is indicative of increased extensor tone, and of a lesion affecting the descending motor, or ascending spinocerebellar pathways to that limb. A hypometric gait, particularly in the thoracic limbs, is seen best when the animal is backed up or when it is maneuverd on a slope with the head held elevated. The thoracic limbs may move almost without flexing and resemble a marching tin soldier. The short-stride, staggering gait seen with vestibular disease may be considered hypometria. Also, movement of a limb with prominent flexor weakness can result in poor joint flexion and dragging of toes as with hypometria but the movement and placement of the limb is relatively repetitive and predictable. Finally, **dysmetria** is a term that incorporates both hypermetria and hypometria. Animals with severe cerebellar lesions may have a high stepping ataxic gait, but have limited movement of the distal limb joints, especially in thoracic limbs. This is best termed dysmetria. In all these various situations we do need to take other abnormalities into consideration in defining the presence and characteristics of ataxia.

Ataxic movements are thus seen as a swaying from side to side of the pelvis, trunk, neck and sometimes the whole body. It may also appear as a weaving of the affected limb during the swing phase. Such abnormalities can be seen whilst an assistant maneuvers the patient but also as one walks the horse with the head elevated and while pulling on the tail. The aim of the latter two maneuvers is to change the direction of limb flight during mid-stride to promote errors due to proprioceptive abnormalities. This often results in abnormal foot placement in abducted or adducted positions, crossing of the limbs, or stepping on the opposite foot especially while the animal is circling or turning tightly. Any animal that is substantially ataxic for any reason tends to pace when walking with both feet on the same side off the ground at the same time. Circumduction of the outside limbs when turning and circling is also considered a proprioceptive abnormality. Walking an animal on a slope with the head elevated often exaggerates ataxia, particularly in the pelvic limbs. This maneuver also frequently allows expression of a hypermetric

or hypometric component of ataxia in the thoracic limbs. When a weak and ataxic animal is turned sharply in circles, it leaves the affected limb in one place while pivoting around it. This may also occur when backing up.

Ataxia can also be classified in to three syndromes by the quality of the signs seen and the system or pathway involved in the nervous system. These are general proprioceptive ataxia, cerebellar ataxia and vestibular ataxia, and after observing characteristics of a gait abnormality in a patient it is reasonable to attempt to define which of one or more of these syndromes are present.

General proprioceptive ataxia results from involvement of afferent proprioceptive pathways in sensory nerves and more commonly in spinal cord and brainstem tracts. Proprioceptive deficits are caused by lesions affecting the general proprioception sensory pathways, which relay information on limb and body position to the cerebellum for subconscious proprioception, and to the thalamus and cerebral cortex for conscious proprioception. The gait is irregularly irregular and most particularly is unpredictable. There is a delay in onset and a swaying or floating swing phase and subsequent variable foot placement exaggerated by maneuvering the patient. This movement and placement may include adduction and abduction, and hyperflexion in hind limbs and hypoflexion or hypometria in forelimbs is common. General proprioceptive deficits likely contribute to scuffing toes and stumbling, especially on thoracic limbs. Obviously some of these signs are also associated with upper motor weakness, but because general proprioception and upper motor neuron tracts are adjacent in most parts of the central and peripheral nervous system, and involved in disease processes together, it is not necessary to distinguish which gait characteristics is due to dysfunction of one or the other.

Cerebellar ataxia can have characteristics of general proprioceptive ataxia but changes in limb placement and movement tend to be more abrupt in onset and excessive. The best definition of cerebellar ataxia being alterations in the rate, range and force of movement [13]. Thus jerky onsets of movement and hypermetria are often seen, becoming more pronounced with more complex maneuvers such as hurriedly regaining an upright posture from recumbency or abruptly turning to flee from being frightened. There is no upper [or lower] motor neuron paresis accompanying cerebellar disease but other signs of cerebellar involvement including head tremor and defective menace responses often are present. Signs of vestibular involvement also can be present with pan-cerebellar disease.

Concerning **vestibular ataxia**, although the gait limb movement and foot placement accompanying mild to moderate vestibular disease are irregular, and therefore can be called ataxic, they are somewhat less unpredictable. For example, if thoracic limb movement is forced to change in direction while the patient is lead with its head raised, the resulting correction will be predictably abducted. Also on turning a patient with mild vestibular disease, the wide movement and placement of an outside hind limb will not usually be accompanied by hypermetria and any hurried movements to maintain a balanced posture will be strong and multiple, thus again somewhat predictable.

Normal horses react in different ways to blindfolding from extremes of excitement or distress to acting very calm and subsequent movements they make while blindfolded then often depend on this behavioural response. Vestibular ataxia and loss of balance often will be markedly exacerbated when a blindfold is applied to a horse suffering from vestibular or occasionally diffuse spinocerebellar and cranial cervical spinal cord disease. On the other hand, observing the posture and gait in response to blindfolding a horse suspected of suffering from typical mid to caudal cervical spinal cord compression usually does not add anything substantial to the neurologic evaluation. Damage to the sensory, C_{1-3} dorsal nerve roots can produce vestibular ataxia and this may be expected to exacerbate with blindfolding the horse [20].

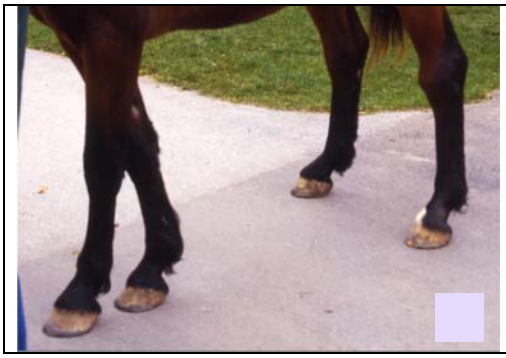

Figure 2: Stopping a patient abruptly after manoeuvring it may result in abnormal limb postures being adopted and maintained as shown here. This may be taken as evidence for abnormal conscious proprioceptive input from the limbs to the forebrain. On the other hand, an obtunded horse, one with prominent weakness or rarely one trained for unusual posturing may not correct such abnormal limb positioning without having any specific ascending conscious proprioceptive pathway lesion.

Regarding assessment of posture and postural abnormalities, flexing the foot to attempt to make the animal stand on the dorsum of the pastern and determine how long the animal leaves the foot in this state before returning it to a normal position, is said by some to be a test for conscious proprioception in dogs and cats. Almost certainly this involves somatic afferent pathways as well and a very weak patient may not be able to move the foot from many abnormal positions. This test can be attempted in large animals, but in my hands has not been helpful at all. Inactive and somnolent patients, especially calves, often allow the foot to rest on the dorsum for prolonged periods. Horses need to have almost total paralysis of the limb, or a nociceptive sensory deficit in the limb before they allow such postural anomalies to be accomplished. Other tests, such as manually crossing the limbs or placing one limb on a sack and slowly sliding the sack to the side, have been tried to test conscious proprioception but again in my hand have proven to by non-contributory to the examination process. Rather than manually placing limbs in abnormal positions, it appears more reliable to maneuver the horse rapidly, say in a circle, and stop the maneuver abruptly [Figure 2]. This often results in an initial awkward placement of the limbs and then the examiner can determine how long the horse leaves the limbs in such an abnormal posture to determine the presence or not of conscious proprioceptive deficits. This procedure probably does test for deficiencies in conscious proprioception. Examination of horses walking 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 moving cautiously, even if quite weak and ataxic, often can maneuver such obstacles.

Gait alterations can occur in all four limbs with lesions affecting the white matter in the caudal brainstem when head signs such as cranial nerve deficits are used to define the site of the lesion. Subacute to chronic lesions affecting the forebrain cause no substantial change in gait. However, postural reactions, such as hopping, are abnormal and sometimes the gait is slowly initiated on the thoracic limb contralateral to the side of a forebrain lesion.

In smaller patients, other postural reactions can be performed. These primarily help detect signs of subtle proprioceptive and motor pathway lesions when the gait is normal. Wheelbarrowing the patient to make it walk on just the thoracic limbs, hopping it laterally on each individual thoracic and pelvic limb and hemistanding and hemiwalking the animal by making it stand and then walk sideways on both left, then both right limbs, are three useful postural reactions to perform. Even in large, adult animals, particularly horses, it is possible to perform a modified hopping response test with the thoracic limbs. This is done by lifting each thoracic limb in turn while using the shoulder to make the horse hop laterally on the other thoracic limb. This test can help the clinician decide if there are subtle neurologic abnormalities in the horse's thoracic limb control. Brainstem and spinal cord lesions appear to result in postural reaction deficits on the same side as the lesion, whereas cerebral lesions produce contralateral abnormalities.

At the conclusion of the examination, a most likely site of any acute nervous system lesion frequently can be defined accurately by determining the precise characteristics and severity of any gait and posture abnormalities present and the degree of weakness, ataxia, hypometria, hypermetria and conscious postural deficits should be graded for each limb [Table 1].

With peracute lesions, particularly those of an inflammatory nature and those with soft tissue compression of the spinal cord such as with caudal cervical arthritis and 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. In contrast, a horse suffering from chronic spinal cord disease may show quite different neurologic signs. For example, a horse that has suffered a single insult of cervical spinal cord compression a year prior to examination may have an unusual, perhaps hypermetric, mild ataxia in the pelvic limbs with no evidence of pelvic limb weakness and no signs in the thoracic limbs other than a questionably poor response to hopping. The anatomic diagnosis in such cases may be a thoracolumbar, cervical, or diffuse spinal cord lesion. A moderate or severe abnormality in the pelvic limbs, and none in the thoracic limbs, is consistent with a thoracolumbar spinal cord lesion. With a mild, and a severe change in the thoracic and the pelvic limb gaits respectively, one must consider a severe thoracolumbar lesion plus a mild cervical lesion, or a diffuse spinal cord disease. Lesions involving the brachial intumescence at C_6-T_2, with involvement of the gray matter supplying the thoracic limbs, and diffuse spinal cord lesions may both result in a severe gait abnormality in the thoracic limbs and the pelvic limbs. A severely abnormal gait in the thoracic limbs, with normal pelvic limbs, indicates lower motor neuron involvement of the

thoracic limbs; a lesion is most likely present in the ventral gray columns at C_6-T_2, or thoracic limb peripheral nerves or muscle.

TABLE 1: Prominent gait and postural abnormalities present with neurologic lesion at different locations.

LESION LOCATION	GAIT AND POSTURAL ABNORMALITIES				
	POSTURAL DEFICITS	PARESIS	ATAXIA	HYPOMETRIA	HYPERMETRIA
Cerebrum	+++	O	O	O	O
Brain Stem	++	++	++	++	++
Vestibular	+++	O	++	++	O
Cerebellum	++	O	+++	+	+++
Spinal Cord / UMN	++	++	++	++	++
Peripheral Nerve / LMN	++	+++	+	(++)*	(+++)*
Musculo-Skeletal	+	++	O	+	O

O = not usually expected
\+ = mild if present
++ = usually present
+++ = quite characteristically present
* = usually only with selection sensory fiber involvement

Interpretation of Signs in Spinal Cord Disease

Neck and Forelimbs

If a gait alteration was detected in the thoracic limbs and there were no signs of brain involvement, then this part of the examination can confirm involvement of the C_1-T_2 cervicothoracic spinal cord or peripheral nerves or thoracic limb muscles; it should also help localize the lesion within these regions.

Results of the thoracolaryngeal adductor response or slap test can be a useful part of the complete neurologic evaluation of horses suspected to be suffering from lesions of the vagal or recurrent laryngeal nerves, caudal medulla oblongata or cervical and cranial thoracic spinal cord. As most emphasis is placed on its utility in diagnosing cervical spinal cord disease in wobbler horses, some aspects of testing will be reiterated here. The test can be performed in co-operative horses by palpating the dorsal and lateral laryngeal musculature while simultaneously slapping the contralateral dorsolateral thoracic region from the cranial withers to near the last rib during expiration. If there is difficulty in interpretation of this test, observing the larynx via an endoscope while performing the test may be necessary. It should be emphasized that the thoracolaryngeal response is not consistently absent in horses with cervical spinal cord disease or caudal brain stem disease and can be absent in horses with no evidence of CNS disease [22, 23]. However, a reduced or absent slap reflex on the left side of the larynx must be taken as strong evidence for the presence of idiopathic recurrent laryngeal neuropathy or prior laryngeal surgery, although treadmill exercise and endoscopic examination of the horse will be necessary to confirm any clinical problem of reduced laryngeal function and roaring. Bilateral absence of the response without other signs of severe laryngeal or cervicomedullary disease must be interpreted cautiously, particularly in an excitable horse. A normal response on the left side of the larynx and absent response on the right side most often indicates a neurologic disease other than classical idiopathic recurrent laryngeal neuropathy. The commonest cause of acute, acquired, severe bilateral laryngeal paralysis is hepatoencephalopathy.

Observation and palpation of the neck and forelimbs will detect gross skeletal defects, asymmetry in the neck and muscle atrophy. These signs may be associated with neurologic disease and thus be localizing findings. The neck should be manipulated to assess normal range of movement. Interpretation of what appears to be reluctance to move the neck passively or actively in any direction as indicating neck pain is fraught with difficulties. On the other hand, if a horse will not lower or bend its head to eat, drink or

reach for a treat this usually indicates a mechanical or painful disruption to movement of the cervical vertebrae, particularly in the caudal neck. Cervical vertebral arthrosis, involvement of cervical nerve roots, and marked cervical spinal cord disease can cause scoliosis and even torticollis.

Importantly, as musculoskeletal diseases are far more common than neurologic disease and as disuse atrophy can occur within at least weeks of onset of lameness, evidence of muscle atrophy, especially common over the scapula, should be taken as evidence of an underlying lameness until there is additional evidence that it is neurologic in origin.

Clearly delineated regions of cervical and thoracic sweating can be useful indicators of localized spinal cord or peripheral nerve disease in that they can represent sympathetic denervation or decentralization of the vasculature in the skin, resulting in increased circulating adrenalin stimulating sweat glands to secrete. Care must be taken in interpreting patchy sweating that is not well delineated. Very asymmetric patchy sweating can occur in horses that are excited or distressed, particularly when in a draughty box, without a specific sympathetic lesion being present. Involvement of the peripheral pre- and postganglionic sympathetic neurons in the horse result in localized sweating; this can be an extremely helpful localizing sign. Horner's syndrome will result if the cervical sympathetic trunk is damaged. In the horse, dermatomal patterns of sweating on the neck and cranial shoulder occur with involvement of the C_3-C_8 branches of the sympathetic fibers. These arise segmentally from the vertebral nerve that follows the vertebral artery up the neck after the vertebral nerve has left the stellate ganglion near the thoracic inlet.

When the skin of the lateral neck of a horse just above the jugular groove from the level of the atlas to the shoulder is prodded firmly with a blunt probe, the cutaneous coli muscle contracts, which causes skin flicking. The sternocephalicus and brachiocephalicus muscles often contract also, causing the shoulder to be pulled cranially and even the head to be flexed laterally. This response tends to be less obvious in other species. In horses, there also is flicking of the ear rostrally, blinking of the eyelids, and contracture of the labial muscles inducing a smile when the test is performed. Originally introduced by Rooney [24], these are termed the local cervical and cervicofacial responses, respectively. The precise anatomic pathways are not known, although they must involve several cervical segments and the facial nucleus in the medulla. Cervical lesions that involve gray and white matter can cause depressed or absent cervical responses however interpretation of abnormal responses may need to be expressed as imprecisely as for example "consistent with a caudal cervical lesion" or "consistent with a cranial cervical lesion". In contrast, the *cutaneous trunci* reflex can be very useful in delineating the precise cranial extent of a thoracic spinal cord lesion particularly when such a lesion is asymmetrical.

Sensory perception from the neck and forelimbs must be assessed. This can be difficult to evaluate accurately in stoic animals. Perception of a noxious stimulus is noted by observing the animal's behavioural response while observing the local cervical responses and continuing the skin prodding over the shoulders and down the limbs to include testing the autonomous zones for the thoracic limbs [Figure 2-14]. As with any cutaneous sensory test, a two-step technique is recommended [25]. This is accomplished by initially tenting and grasping a fold of skin between the jaws of heavy duty hemostats or needle holders. After pausing to allow the patient to settle, a second, sharp skin pinch is applied to determine superficial sensation. There may be reflex withdrawal of the part with or without a cerebral response, such as vocalization or moving the whole body away from the stimulus; the latter taken as representing conscious perception of the noxious stimulus.

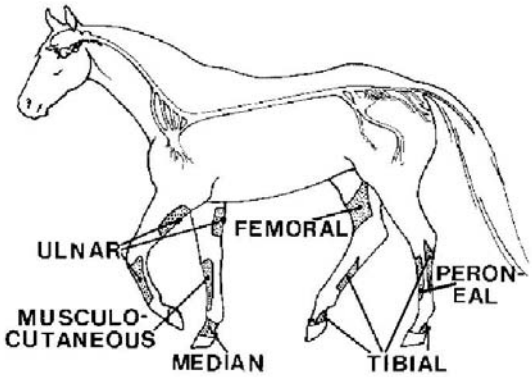

Figure 3: Autonomous zones for areas of desensitivity expected with individual peripheral spinal nerves to the limbs are not functioning [after Blythe, 1998].

If a large, adult animal has significant gait abnormality and it is feasible to cast it, then this should be done to assess the spinal reflexes. If the animal is ambulating well, it may be assumed that the spinal reflexes are intact. These reflexes can be studied in all smaller patients.

When evaluating wobblers with evidence of a neurologic abnormality in both thoracic and pelvic limbs and no evidence of brain disease, one should allow for a lesion to be present anywhere from C1 through T2. Conversely, when there is evidence of a mild neurologic gait abnormality in the pelvic limbs but not the thoracic limbs then the possibility of a lesion anywhere from C1 through S2 must be considered. If the signs of ataxia and/or paraparesis are moderate or even marked then a lesion can be considered anywhere in these segments especially C6 through S2. The reason to include lesions sites at C6-T2 is because such lesions, when intramedulary, can be very selective and spare tracts involving the thoracic limbs resulting in no definitive thoracic limb signs. Such has been the case in adult horses suffering from *S. neurona* myelitis, fibrocartilage thromboembolism, granulomatous meningoencephalomyelitis and migrating helminth parasites affecting C6-T2 spinal segments.

Trunk and Hindlimbs

If the examination of the head, gait and posture and neck and thoracic limbs reveals evidence of a lesion, then an attempt should be made to explain any further signs found during examination of the trunk and hindlimbs to have been caused by that lesion [Figure 2-15]. If there are only signs in the trunk and hindlimbs, then the lesion(s) must either be between T_2 and S_2, or in the trunk and pelvic limb nerves or muscles. This part of the examination helps localize such lesions more precisely. However, the examiner must remember that with a subtle, grade 1+ neurologic gait abnormality in the pelvic limbs, the lesion may be anywhere between the midsacral spinal cord and the rostral brainstem.

The trunk and hindlimbs must be observed and palpated for malformation and asymmetry. Lesions affecting thoracolumbar gray matter cause muscle atrophy, which is a helpful localizing finding. With asymmetric myelopathies scoliosis of the thoracolumbar vertebral column often occurs, initially with the concave side opposite the lesion. Once again, evidence of muscle atrophy, especially common over the gluteal region [Figure 4], should be taken as evidence of an underlying lameness until there is additional evidence that it is neurologic in origin.

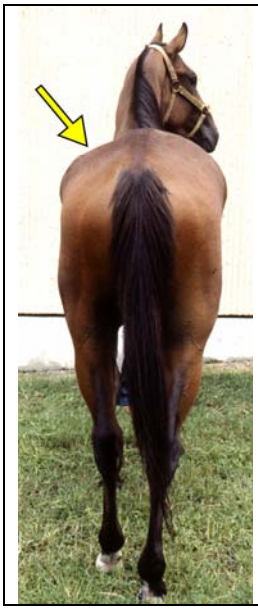

Figure 4: Proximal limb atrophy more often is due to disuse [mostly orthopaedic disease] than to neurogenic causes. However selective middle gluteal muscle atrophy in the absence of lameness and atrophy of other proximal limb musculature, as seen on the left side in this horse, is likely to be due to lower motor neuron disease such as that caused by *S. neurona* myelitis, as was the case here.

Sweating in the horse over the trunk and hindlimbs, excluding the neck and face, can be a helpful localizing sign. Ipsilateral sweating caudal to the lesion signals involvement of the descending sympathetic tracts in the spinal cord caudal to T3. Lesions involving specific pre- or postganglionic peripheral sympathetic fibers that are second and third order neurons cause patches of sweating at the level of the lesion.

Firm prodding of the skin over the trunk, particularly the lateral aspects of the thoracic wall, causes a contraction of the cutaneous trunci muscle, which is seen as a flicking of the skin over the trunk. The sensory stimulus travels to the spinal cord in thoracolumbar spinal nerves at the level of the site of

stimulation. Transmission is then cranial in the spinal cord to C_8-T_1, where the lower motor neuron cell bodies of the lateral thoracic nerve are stimulated resulting in contraction of the cutaneous trunci muscle. Lesions anywhere along this pathway may cause suppression of the response, which is easiest to detect with an asymmetric lesion. In addition to this, an assessment of sensory perception from the trunk and hindlimbs must be made. This appears as a cerebral, behavioural response to a two-pinch stimulus described above, that includes assessment of the autonomous zones for the pelvic limbs [Figure 3]. Degrees of hypalgesia and analgesia have been detected caudal to the sites of thoracolumbar spinal cord lesions, but only when they are severe.

Stroking firmly with a blunt probe or pinching and pressing down firmly with the fingers over the thoracolumbar paravertebral muscles causes a normal animal to extend into a slightly lordotic stance and fix the thoracolumbar vertebral column. It also resists the ventral motion and usually does not flex the thoracic or pelvic limbs. Continuing this stimulus to the dorsal sacral region results in a degree of flexion and a kyphotic stance. A weak animal usually is not able to resist the pressure by fixing the vertebral column and thus it over-extends or over-flexes the back and begins to buckle in the pelvic limbs. Prominent back pain can result in poor responses and evidence of pain perception by, say, a grunt from the patient.

Recumbent Patient.

Every effort should be made to help a recumbent patient stand and walk, unless there is suspicion of bone fracture. By so doing, one can learn as much or more about voluntary effort and lesion localization than one can from reflex testing. Heavy animals in particular should be moved early in the course of recumbency to avoid secondary problems like decubital sores, decreased blood supply to limbs and dehydration, which make evaluation difficult.

A patient that has recently become recumbent, but uses the thoracic limbs well in an attempt to get up, most likely has a lesion caudal to T_2. If such an animal cannot attain a dog-sitting posture, the lesion is likely to be in the cervical spinal cord [Figure 1]. If only the head, but not the neck, can be raised off the ground, there probably is a severe cranial cervical lesion. With a severe caudal cervical lesion, the head and neck usually can be raised off the ground, although thoracic limb effort decreases and the animal usually is unable to maintain sternal recumbency. Assessments of limb function must not be done while a heavy animal is lying on the limb being tested. Muscular tone can be determined by manipulating each limb. A flaccid limb, with no motor activity, is typical of a lower motor neuron lesion to that limb, but in heavy recumbent animals there can be poor tone and little observable voluntary effort in a limb that has been lain upon. A severe upper motor neuron lesion to the thoracic limbs at C_1-C_6 causes poor or absent voluntary effort, but there will be normal or sometimes increased muscle tone in the limbs. This is because there is a release of the lower motor neuron that is reflexly maintaining normal muscle tone from the calming influences of the descending upper motor neuron pathways. Interestingly, such a hypertonic paralysis in the pelvic limbs also can be seen with lesions between C_6 and T_2 if little or no gray matter is affected. A Schiff-Sherrington phenomenon of short duration, with excessive extensor tone in the thoracic limbs in the presence of good voluntary activity and normal reflexes, has been seen rarely in horses, and usually follows a cranial thoracic vertebral fracture [26].

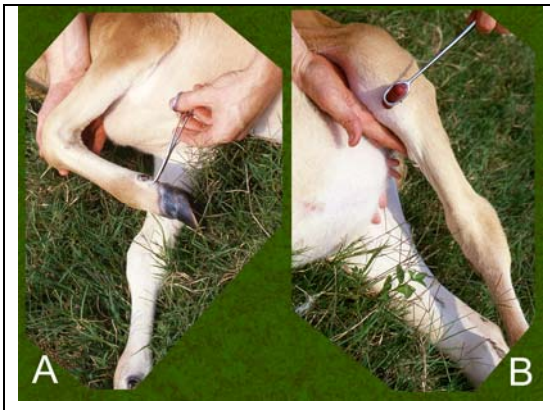

Figure 5: The three important spinal limb reflexes to perform on any patient that can be placed in lateral recumbency are the flexor reflexes in the thoracic [A] and pelvic limbs, and the extensor or patellar ligament reflex in the pelvic limb [B]. All other reflex testing can be problematic in interpretation and results do not change an anatomic diagnosis. Normal hyperactive reflexes and crossed extensor reflexes were present as expected in this normal neonatal calf. Reflexes should be tested in both pairs of limbs while uppermost and while dependant, the most prominent result being taken as real. Occasionally a particular reflex cannot be elicited in a normal patient, usually bilaterally.

Finally, spinal reflexes are tested in the thoracic limbs. The flexor reflex in the thoracic limb involves stimulation of the skin of the distal limb with needle holders and observing for flexion of the fetlock, knee, elbow, and shoulder [Figure 5]. This reflex arc involves sensory fibers in the median and ulnar nerves, spinal cord segments C_6 to T_2, and motor fibers in the axillary, musculocutaneous, median and ulnar nerves. Lesions cranial to C_6 may release this reflex from the calming effect of the upper motor neuron pathways and cause an exaggerated reflex with rapid flexion of the limb. The limb may remain flexed for some time and even show repetitive movements or clonus. Such lesions also may result in a crossed extensor reflex, with synchronous extension of the untested limb. This usually occurs only with severe and chronic upper motor neuron lesions. Thus, an animal affected by such a lesion may demonstrate considerable reflex movement following stimuli, but usually will have little voluntary motor activity in the limbs being tested. A spinal reflex can be intact without the animal perceiving the stimulus and the latter must be observed for independent to the local reflex movement. Cerebral responses associated with perception include changes in facial expression, head movement and phonation. Conscious perception of the stimulus will be intact only as long as the afferent fibers in the median and ulnar nerves, the dorsal gray columns at C_6-T_2, and the ascending sensory pathways in the cervical spinal cord and brainstem are intact.

Interpreting results of testing the tendon reflexes in the thoracic limbs is problematic and does not usually assist in defining the site of neurologic lesion, perhaps with the exception of neonatal animals. Also, patients with profound diffuse neuromuscular paresis can have reflexes that are at least present. However a general description of two of these reflexes follows, testing the remainder being superfluous. To perform the triceps reflex the relaxed limb is held slightly flexed and the distal portion of the long head of the triceps and its tendon of insertion is balloted with a rubber neurology hammer in smaller patients or a heavy metal plexor in larger patients while observing and palpating for contraction of the triceps muscle, which causes extension of the elbow. The triceps reflex involves the radial nerve for its afferent and efferent pathways and spinal cord segments C_7 to T_1. The triceps reflexes, although present, can be extremely difficult to demonstrate in heavy, adult, recumbent patients. The musculotendinous portion of the extensor carpi radialis muscle can be balloted to produce extension of the knee when the relaxed limb is held in a partially flexed position. This extensor carpi radialis reflex involves afferent and efferent fibers also in the radial nerve but the reflex may not always be present in normal adult animals.

All these reflexes usually are active in normal neonates and there is a prominent crossed extensor reflex present, and these slowly subside through the first weeks of life.

The pelvic limb spinal reflexes may also be evaluated in all animals that can be restrained in lateral recumbency and in all recumbent patients. In addition, the amount of voluntary effort and muscle tone present in the pelvic limbs is assessed in recumbent patients. As described for the thoracic limbs, this can be done while watching the animal attempt to get up, or by observing its struggle in response to stimuli while lying in lateral recumbency. Consideration must be given to possibly exacerbating a fracture.

The patellar reflex and the flexor reflex are the two most clinically important spinal cord reflexes involving the pelvic limbs. The patellar reflex is performed by supporting the limb in a partly flexed position, tapping the intermediate patellar ligament with a neurologic hammer or a heavy metal plexor, and observing for a reflex contraction of the quadriceps muscle resulting in extension of the stifle [Figure 5]. The sensory and motor fibers for this reflex are in the femoral nerve and the spinal cord segments involved are primarily L_4 and L_5. The flexor reflex is performed by pinching the skin of the distal limb with needle holders and observing for flexion of the limb. The afferent and efferent pathways for this reflex are in the sciatic nerve and involve spinal cord segments L5 to S3.

Although two other reflexes can be elicited in most neonatal animals, they frequently are not clearly reproducible in adult patients and thus results of testing them do not usually contribute to defining the site of neurologic lesions. The gastrocnemius reflex is performed by balloting the gastrocnemius tendon and observing and palpating for contraction of the gastrocnemius muscle, which is accompanied by extension of the hock. This reflex involves the tibial branch of the sciatic nerve and spinal cord segments L_5 to S_3. Secondly, the cranial tibial reflex causes contraction of the cranial tibial muscle with hock flexion occurring when the muscle is balloted and the relaxed limb is held partially extended. Variable limb

movement in response to mechanical stimulation may be interpreted falsely as a positive reflex muscle contraction when both these reflexes are tested.

Distinguishing characteristics of lower motor neuron paresis and paralysis and upper motor neuron paresis and paralysis are given in Table 2-5. Such distinctions often are straightforward and assist in anatomically localizing the site and extent of spinal cord and peripheral nerve lesions in many patients but in recumbent heavy patients and those with chronic disease and disuse these classic characteristics can merge such that this distinction can be problematic.

Skin sensation of the pelvic limbs should be assessed independently from reflex activity using the two-pinch technique. The femoral nerve is sensory to the skin of the medial thigh region, the peroneal nerve to the dorsal tarsus and metatarsus, and the tibial nerve to the plantar surface of the metatarsus. As for the thoracic limbs, lesions of the peripheral nerves to the pelvic limbs, such as the femoral and peroneal nerves, result in specific motor deficits; however, the precise sensory deficits can be difficult to define.

The patellar reflex is hyperactive in newborn foals, calves and kids, and probably in all large animal neonates. Also, the cranial tibial and gastrocnemius tendon reflexes are easily performed in healthy, cooperative newborn patients. As with the forelimbs, these patients have normal, strong, crossed extensor reflexes. In addition, an extensor thrust reflex is obtained, in very young foals at least, by rapidly overextending the toe while the limb is already partially extended. This results in forceful extension of the limb, and possibly represents a Golgi tendon organ reflex.

Interpretation of Signs in Peripheral Nerve Disease

For accurate interpretation of signs of peripheral nerve disease some consideration must be given to the neuropathological classification of damage to peripheral nerves that can result in degrees of loss of function:

- ***Neurapraxia*** is temporary loss of function with no morphological changes.
- ***Axonotmesis*** is damage to axons with preservation of myelin sheaths resulting in prolonged loss of function until axonal regrowth re-establishes innervation of muscle.
- ***Neurotmesis*** is severance of axons and their myelin sheaths with prolonged to permanent loss of function, sometimes with partial re-innervation depending on both the distance between the proximal and distal nerve segments and between the lesion and the muscle.

With loss of somatic efferent innervation due to axonal or whole nerve fiber damage there is muscle atrophy, which occurs relatively rapidly although in horses it may take one to three weeks to become clinically prominent. Electromyographic changes indicating denervation of muscle may take even longer, and be 3 to 6 weeks to become prominent in the horse. Surprisingly, disuse atrophy appears to occur quite rapidly in the horse and therefore distinguishing neurogenic atrophy from disuse atrophy clinically can be fraught with problems. A good example of the significance of this would be an unusual asymmetric hindlimb gait abnormality in a horse with accompanying gluteal muscle asymmetry. Unless profound, such asymmetric muscle atrophy should be taken as evidence for disuse due to lameness until proven otherwise.

From a practical point of view peripheral nerves are very difficult to injure directly or to stretch unless they are fixed *in situ*, they overlie a bony structure such as the case for portions of the facial and suprascapular nerves or there is a penetrating injury.

Presumed peripheral nerve irritation and vascular compromise can result in unusual syndromes in horses. Perhaps the best example of these is the abrupt onset of distress involving one limb when the horse will kick out and repeatedly stomp the foot on the ground that can be referred o as a form of claudication. This occasionally is seen following an intramuscular injection, presumed to be adjacent to a peripheral sensory or mixed nerve. The other example would be the similar syndrome that can appear upon recovery from general anesthesia wherein there is no evidence of a myopathy or motor neuropathy, the most likely explanation being the onset of paresthesia or as it is referred to in humans, pins and needles. Such unusual syndromes can occur spontaneously in horses sometimes with no associated or predisposing incident, sometimes associated with exertion. Most often these signs dissipate rapidly, with occasional notable exceptions.

Compared to small animals, the specific areas of desensitivity relating to each major spinal nerve, referred to as autonomous zones, are quite variable from horse to horse [Figure 3]. The variable analgesic

zones found following tibial and peroneal, and medial and ulnar nerve blocks undertaken during orthopedic evaluations attest to this. Determining such precise areas of analgesia can be extremely useful, albeit often frustrating, in helping to localize a peripheral neuropathy although their absence should not exclude such syndromes. On the trunk and proximal limbs the two pinch technique outlined above is preferable for sensory testing.

With the exception of those affecting the *cauda equina*, peripheral nerve lesions usually result in a gait abnormality involving only one limb. Classically the further the lesion is away from the central nervous system the more selective are any motor and sensory deficits. This is less true in the horse for several reasons particularly because of peripheral nerve anastamoses and secondly because incomplete peripheral neuropathies frequently occur.

The gait abnormalities present after several days following onset of selective median or ulnar neuropathies are minimal. The same can be said of tibial and peroneal nerve lesions although sometimes there will be a change in stride with occasional stumbling.

The radial nerve is probably rarely damaged alone. However, the commonly recognized signs of typical proximal radial nerve paralysis, including lack of carpal and fetlock extension and an inability to bear weight on the limb with a dropped elbow, usually results from partial brachial plexus involvement. Theoretically this syndrome should be distinguishable from myopathy involving the triceps or the *extensor carpi radialis* muscles, elbow joint lameness, humeral fracture and bicipital bursitis. However in practice such distinction can be difficult to achieve without a thorough evaluation usually requiring ancillary aids including ultrasonography, radiography, synovial fluid analysis and electromyography. Because partial brachial plexus lesions are quite common following thoracic limb injury this problem should be considered foremost when an inability to bear weight on an otherwise pain-free thoracic limb is being evaluated.

Musculocutaneous nerve problems are rarely, if ever, encountered alone and after some initial stumbling any permanent gait abnormality may be difficult to detect.

Immediately after an episode of shoulder injury, signs of damage to the purely motor suprascapular nerve often include a degree of lameness, presumably associated with adjacent soft tissue and periosteal damage. Suprascapular muscle atrophy will ensue in a week or two but the shoulder abduction that occurs on weight bearing, or so-called shoulder slip, that is seen with thoracic limb trauma and is presumed to be lateral laxity to the shoulder joint likely results from loss of lateral support of the shoulder due to suprascapular paralysis alone [27]. Other signs, such as sensory deficits over the caudal neck and shoulder and ensuing muscle atrophy elsewhere in the limb, must make the clinician suspicious of more than suprascapular nerve involvement such as additional damage to the brachial plexus.

The femoral nerve is incredibly well protected from external injury although damage to it will ultimately result in quadriceps atrophy. Even with moderate muscle atrophy and posturing with the pelvis flexed and back arched, horses with partial unilateral femoral nerve lesions can have a remarkably normal gait at the gallop but athletic performance probably is curtailed. Femoral nerve lesions must be quite proximal in the limb before medial thigh hypalgesia resulting from saphenous nerve involvement can be detected.

Cauda equina involvement most frequently results from a fractured sacrum or from polyneuritis equi. Such signs may begin acutely or may be delayed following onset of the disease. A slightly abnormal gait may be detected in the pelvic limbs but the cause may not be identified until the perineal region is evaluated closely when other signs of cauda equina involvement became apparent.

Other characteristic gait abnormalities do strongly suggest peripheral nerve disease. Stringhalt is one example where there is exaggerated flexion of the limb during protraction with excessive hock flexion and digital extension resulting from excessive contraction of the digital extensor muscles or lack of opposition from digital flexor muscles. This syndrome can occur with spinal cord disease as well as peripheral nerve disease and probably lesions at other sites. A thorough musculoskeletal examination including radiographic and ultrasound evaluation of the affected limb may reveal abnormalities detected within the lateral digital extensor muscle, tendon or sheath, or in the hock. Any abnormalities detected often are assumed to initiate the abnormal neural reflexes thus increasing tone in the digital extensor muscles during protraction. Another interesting gait that results from mechanical interference to contraction of caudal thigh muscles, or perhaps sometimes because of reflex hypertonia involving these muscles, is referred to as fibrotic myopathy. In this syndrome the gait classically results in excessive slapping of the foot to the ground at the end of protraction, thus shortening stride length. Mild to moderate fibrotic myopathy

usually does not appear to interfere with high speed performance, however, dressage horses, show horses and trotters and pacers do show an abnormal gait during performance.

Variations in these characteristic gait abnormalities occur. These include repetitive or intermittent mild abduction of the hindlimb during protraction and caudal jerking of the distal hindlimb after the initiation of protraction. It is possible to explain these and other movement disorders by initiation of abnormal muscle spindle activity, as in Stringhalt, with a result that certain muscles or groups of muscles contract too early or too late, or excessively or poorly at a particular phase of the stride. Thus, intermittent abduction and caudal jerking in the hindlimb may result from hypertonia and hypereflexia involving the *biceps femoris* muscle during the swing phase of stride.

Cantering with synchronous movement of the hindlimbs is referred to as bunny-hopping and is seen with numerous musculoskeletal problems. It is rarely the result of primary neurologic disease but can occur with certain congenital or acquired spinal cord malformations. Overt evidence of peripheral nerve or spinal cord disease, or identification of bilateral and synchronous hindlimb reflexes determined during recumbency need to be present before a neurologic cause for bunny hopping can be confirmed.

Finally, horses diagnosed as shiverers demonstrate a wide variety of signs including slightly excessive flexion of the hindlimbs along with thigh muscle and tail trembling at the onset of backing, reluctance to have the hindlimbs picked up with degrees of thigh muscle trembling, inability to back-up and spontaneous and induced episodes of muscle trembling with hindlimb and forelimb and neck extension that may wax and wane. Interestingly, an acquired lameness can abruptly exacerbate the syndrome. A few horses demonstrating shivering suffer from mild spinal cord disease, others from marked lumbar arthropathy, others from destructive lesions of the lumbosacral vertebrae and others from painful conditions involving the distal hindlimbs. More often than not, no site or cause of the lesion is determined in such shiverers.