

THE NEUROLOGIC EXAMINATION

As with all other examining procedures, the neurological examination should be carried out in a systematic fashion and with patient comfort in mind. No examination should be performed without consideration to the patient. If there exists an unstable patient condition, the exam must be altered to ensure patient safety. A thorough exam may not be very important if indeed the patient has been rendered worse by it. Therefore, care should always be taken to maximize the information obtained without patient neglect. At the same time, many patients are under extreme duress and care must be taken to assure the safety of the examiner and all helpers. See the Neurological Examination Form.

It is usually best to start with the head and work caudally in as systematic manner so that all areas receive the necessary attention. One should note the animal's attitude, level of consciousness, and gait prior to actually beginning the exam, however; and all abnormalities of such be noted. One can rapidly check the cranial nerve function if the lesion is obviously in other areas of the nervous system; but the cranial nerves should always be inspected, even if only briefly.

Cranial Nerve Examination:

CNI, or the olfactory nerve, is checked by moistening a wisp of cotton with a volatile oil. This can be positioned near the animal's nostril to observe for any response which might indicate the animal "smelled" the compound. With ether as the compound, the response is usually to try to move away from the cotton. There are some obvious problems with this technique and the results are quite subjective. However, a general idea as to the animal's "ability to smell" may be detected. In many ways, it is unfortunate that we cannot tell more about an animal's ability to smell. Since animals are so dependent upon olfaction for their perception of their environment, it is not easy to speculate on the importance of this function to them. This olfactory dependence is even difficult for us to fully appreciate. There are newer techniques using evoked potential recordings with computer averaging whereby more definitive information can be gained about an animal's ability to smell. Complex behavioral pattern recognition will perhaps be available in the future. Today, even the role of olfactory mechanisms in the pathogenesis of focal seizure activity is poorly defined. In my opinion, this is an area that should be considered closely for future research.

CNII, or the optic nerve, can be evaluated as to the ability of the animal to see. If the animal can follow moving objects, such as a dropped cotton ball, and will react to noxious stimuli, such as rapid movement of the hand toward the animal's eye, then the animal probably can see to some degree. Again, these tests are subjective evaluation. The ability of the animal to move in its environment without bumping into things may also aid in assessing the nervous system. Only computer assisted evoked potential studies enable direct evidence of vision. ERG's, pupillary reflexes, and fundoscopic (ophthalmic) examination may also be necessary to detect whether a cause of blindness is related to the nervous system per se. At least, some evidence can be collected as to the function of sight in the animal by use of the neurologic exam. If vision is deficient, then additional tests will be warranted.

CNIII, IV, and VI, or the oculomotor, trochlear, and abducts nerves, respectively, can usually be lumped together. If the animal can move the eyes normally and no abnormal movements are detected, the function of these nerves is usually within normal limits. Knowing

the muscles innervated by each of the nerves, one can detect abnormalities in each of the nerves, separately. The oculomotor nerve supplies all the extrinsic eye muscle except those supplied by the trochlear and abducens nerves and supplies the parasympathetic fibers to the intrinsic muscles of the eye. Damage of the oculomotor nerve results in inability to move the eye except for some retraction, lateral, and ventrolateral movements and dilation of the pupil. Pupillary reflexes can be used to test in part for the function of the oculomotor nerves. Remember, though, that this is best done in a quiet, dark room, for animals that are frightened in the presence of bright light may not show normal pupillary reaction. Accentuated oscillation in the size of the pupil (Hippus) may be one indication of irritation of the oculomotor nucleus with resultant fluctuations in nervous tone. The trochlear nerve supplies the dorsal oblique muscle and results in inability for the eye to gaze ventrolaterally. Although this is seen commonly in man, this is a difficult deficiency to detect. The abducens nerve supplies the lateral rectus and part of the retractor oculi muscles. With dysfunction of CNVI, one can see the inability of lateral gazing and some decrease in the ability of the eye to retract. The abducens nucleus is tonically driven by the vestibular apparatus and, therefore, is an important component of nystagmus production, especially in connection with peripheral vestibular disease. It can easily be seen how dysfunction of these nerves could affect the mobility of the eye and, possibly, how dysfunction of each may be detected by the abnormal movements seen.

CNV, or the trigeminal nerve, is sensory to the face and motor to the muscles of mastication. A deficiency will result in anesthesia of the face and weakness of the muscles of mastication. If the lesion is acute, the jaw may hang loosely; but, if atrophy has set in the masseter or temporalis muscles, the jaw may be "frozen in position". One must remember, that the inability of an animal to respond to a painful pinprick or other sensory test is tied closely with the motor output for the area. Therefore, the animal may feel the painful stimulus but be unable to respond to it by motor movement. This can confuse the result of sensory testing of the face, but careful scrutiny will differentiate between sensory (CNV) versus motor (CNVII) dysfunction. The trigeminal nerve is also sensory to the cornea and this function can be tested by the corneal reflex. This consists of lightly stroking the cornea with a small wisp of cotton to observe for a blinking of the eye. One should be careful not to allow the cotton to enter the animal's visual field or the eye blink may become a visual response instead of tactile. It is also wise to be careful in testing a weak or absent reflex, as overzealous manipulation may result in abrasion of the cornea. Brushing of the vibrissae (whiskers) will result in a reflex eye blink and can be used to test CNV (sensory) and CNVII (motor). The trigeminal nerve is sensory to much of the oral cavity and nasal cavity (except the nasopharynx caudally) which may be evident in the ability of an animal to sneeze, etc. Because the pattern for the sensory distribution of the trigeminal nerve is so complex, the careful examiner can often find regional changes in sensation even when gross abnormalities aren't apparent. The regional differences may be of great importance when dealing with central lesions, as the trigeminal nucleus lies in a laminar pattern within the brainstem also. One can dissect the area of lesion down to a very fine degree. As I have alluded to before, the motor function of the trigeminal nerve is closely tied to the muscles of mastication. Palpation of these muscles and, sometimes, visualization of these muscles following clipping away of the hair in longhaired breeds is essential for the examination of the trigeminal nerve. Following acute damage of the motor nucleus of the trigeminal nerve (or the peripheral motor branch), there will be rapid atrophy of the muscles which may result in immobility of the jaw and inability of the animal toprehend and chew food. One must be careful, though, as many muscular disorders can mimic trigeminal nerve damage, including eosinophilic myositis, many

muscular dystrophies, and several metabolic myopathies. It can be difficult to differentiate between these various conditions, although the presence of additional associated nervous dysfunction in the absence of additional muscular disease may favor the neurogenic type of dysfunction. An EMG, blood tests, and muscle biopsy may be required to diagnose the condition. Typically, a lesion on one side of the brain stem will result in ipsilateral (same side) anesthesia and paralysis of the face and contralateral (opposite side) anesthesia and paralysis of the remainder of the body. This is termed "alternating trigeminal paralysis". Fortunately, many of the neurological causes of trigeminal palsies are temporary in nature and recovery will occur in time as long as care is taken to maintain the animals nutritional state and other general health. The trigeminal nerve certainly is complex in nature, but can contribute a tremendous proposition of knowledge about the functional status of the brain stem if examined scrupulously.

CNVII, or the facial nerve, supplies the motor fibers for the muscle of facial expression and is closely associated, as has already been made evident, to the trigeminal nerve. Damage to the facial nerve will result in droopiness to the facial muscle due to motor weakness or paralysis which is most evident when unilaterally damaged. The eye blink reflexes will be non-existent although the eye will remain open (since the retractor palpebral muscle is still intact and is innervated by the oculomotor nerve). Movements of the lips and ear will be restricted and there will be absences of reaction to painful sensations even though they will be felt by the animal. Saliva will drip from the mouth as the facial nerve gives off parasympathetic fibers to the mandibular salivary gland via the chorda timpani nerve and CNV projections. The function the CNVII in combination with CNIX has in supplying sensory fiber from the taste buds to carry impulses for the sensation of taste maybe difficult to test. However, it may be possible with computer assisted, evoked potential studies to define this function more clearly. The facial nerve also supplies sensory fibers to the skin of the lateral ear canal and the auricular cartilage of the ear. This sensation can be carefully tested. One must also remember that the facial nerve supplies the lacrimal gland and will, therefore, influence tear secretion. The most common cause of extra cranial facial nerve damage is secondarily associated to inner ear infection. It is unlikely that the facial nerve could be damaged intracranially without concurrent damage to other cranial nerves in the same area. Therefore, seemingly isolated facial nerve damage is most likely to be extracranial in origin.

CNVIII, or the cochlear and vestibular nerves, is composed of two components which result in quite different disorders. The cochlear nerve is sensory for the sensation of hearing and damage will result in deafness. It is very difficult to test defects in hearing, particularly if unilateral. There may be a decrease in responsiveness to loud noises or a decrease in ability to seemingly localize the direction from which the sound comes. The startle reaction will be absent if bilateral, assuming the tegmentum is intact. One must be careful in testing for sound so that the clap of the hands (or whatever) is not felt by the animal in some other manner, such as air currents or floor vibrations. Again, with computer assisted, evoked potentials, one may be able to detect the animal's ability to hear as well as other information. The vestibular nerve is associated with equilibrium and related reflex connections. Loss of vestibular function will result in circling in tight circles, head tilt to the side involved, loss of balance and righting reflexes, vertigo, rolling motions, and altered eye movements like nystagmus. The nystagmus may be in any direction; but, with peripheral dysfunction, is often horizontal in nature with the slow component to the side of the lesion in ablative disorders. Vesicle or positional nystagmus is usually central in origin. For partial vestibular dysfunction, the abnormality may be made more obvious by the caloric test or by rotation of the animal to check for a normal response. These tests contain many

difficulties in performance of them and interpretation of the results, but may aid in diagnosing the obscure case. The procedures for each are described adequately elsewhere. If vestibular disease is central in origin, there are usually other neurological signs associated with the disorder which have already been described in another portion of this paper.

CNIX, X, and XI, or the glossopharyngeal, vagus, and spinal accessory nerves, respectively, are usually examined together. In general, if the animal can swallow normally, these nerves can be presumed to be functional as they supply, together, all of the pharyngeal and laryngeal musculature. The ability to swallow tasteless liquids may be one of the best indicators of these functions. They lie in close association with one another as they emerge from their cranial foramen and are often damaged together or included together in a neoplastic process at that site. The glossopharyngeal nerve, besides those fibers to the pharyngeal and those fibers for the sensation of taste (as described with CNVII), also carries parasympathetic fibers to the remaining salivary glands and sensation of the inner surface of the tympanic membrane and the Eustachian tube. The glossopharyngeal nerve also receives nervous input from the carotid sinus and body which can be hyperactive upon occasion. Pressure upon the carotid body will normally result in reflex slowing of the heart and a decrease in blood pressure; but can result in fainting if overactive even from relatively minor stimulation, such as pulling upon a lease. The vagus nerve supplies not only the laryngeal musculature, but all of the parasympathetic fibers to the thoracic and abdominal viscera except those supplied by the pelvic nerve. This associates many reflexive phenomena (gag reflex, cough reflex, etc.) and much autonomic nervous activity to this nerve. The numbers of abnormalities are tremendous by large and far beyond the scope of this paper. Examination of the vagus must be done in concert with the physical exam findings and include heart rate, blood pressure, gastrointestinal function, etc. Pressure upon the eyeball will normally result in reflex slowing of the heart if the vagus is functioning. This simple test is not always accurate depending upon the emotional state of the animal and the level of autonomic tone that is present at the time of the test. The vagus also supplies sensory fibers to the external ear canal and the inner surface of the pinna. This can be tested for by careful sensory evaluation and is why neonatal animals will occasionally vomit upon stimulation of that region. The spinal accessory nerve can also be distinguished as the motor supply to the trapezius, sternomastoideus, cleidomastoideus, cleidocervicalis, and omotransversarius muscles. Atrophy of the trapezius muscle (without other disease) is classically associated with damage of the spinal accessory nerve. This may result from high cervical spinal cord damage. So, although CNIX, X, & XI act as a functional unit during deglutition, they have individual properties that can separate them from each other.

CNXII, or the hypoglossal nerve, supplies motor fibers to the intrinsic and extrinsic muscles of the tongue. Damage of CNXII will result in the inability to move the tongue or abnormal movements if unilateral. Unilateral damage will result in deviation away from the lesion initially and deviation toward the lesion later (often 4-5 days) once muscular atrophy has set in. Watching the movements of the tongue will show many of the abnormalities. Placing a "tasty" substance upon the lips will quickly show if the animal can move the tongue to the area. Unfortunately, many injuries to the hypoglossal are the result of overzealous pulling of the tongue forward during intubative procedures and this should be carefully avoided.

Postural Response Examination:

A basic knowledge of spinal pathways is important for understanding the spinal cord

reflexes and this will be stressed where this understanding is essential. Unfortunately, this information is more extensive than this paper is meant to present; but a basis of understanding will hopefully be laid. Spinal cord reflexes are broken down into two major categories, postural or attitudinal reflexes and segmental reflexes. Postural reflexes involve functions requiring higher center interaction whereas segmental spinal cord reflexes involve functions that are contained and controlled within the spinal cord itself. Making this distinction now; will allow the reader to see which reflexes are postural and which are segmental without listing the reflexes as such per se. It is best to take each reflex separately and discuss them in their entirety. One must remember that many of the reflexes are interrelated and that any one reflex is no more important than any other. The assimilation of all the reflexes data, along with other neurologically gathered data, is how the diagnosis is made. The patient must always be treated as a whole organism so that one doesn't lose sight of the overall picture in disease state. The postural reflexes shall be considered first and the segmental reflexes second, much in the sequence in which I routinely perform them during an examination. Remember that any part of the exam that is deemed potentially hazardous to the patient may be necessarily deleted.

Once the animal has been observed in the examination room and the cranial nerves evaluated. The gentle palpation of the animal from the base of the skull caudally is important to develop a "feel" for the animal. This also allows the quick observation for gross body abnormalities or dissymmetry, presence of muscle atrophy, and often areas of tenderness or pain. The presence of regional atrophy or pain can often be the first step in localizing a lesion, particularly if the lesion is minor. Bony deformities if not previously noted in the physical exam should be recorded and any physical abnormality found previously should be rechecked with its potential role in the neurological dysfunctions noted. After this overview, the specific examination can begin.

Conscious proprioception is one of the first tests to perform. This reflex is partially responsible for many of the other postural reflex responses and is a good indicator of spinal cord dysfunction. The test is performed by the turning over of each paw, one at a time, so that the animal is standing upon the dorsum of the foot. This is not a normal position for the foot and the normal animal will almost immediately reposition the foot to the normal position, so that it is once again standing upon the plantar surface. The information for conscious joint kinesthesia (or the ability to know where the extremities are in space) travels up the spinal cord within the dorsal funicular white matter in the fasciculus gracilis (thoracolumbar) and fasciculus cuneatus (cervicothoracic). These nerve fibers synapse in the medulla in their respective nuclei and the neurons from the medullary nuclei continue upwards, after first decussating as the internal arcuate fibers, as the medial lemniscus. As these fibers pass through the brain stem toward the ventrolateral thalamic nucleus, they are joined by the conscious proprioceptive fiber from the trigeminal nucleus (mesencephalic portion of the sensory nucleus of V). Therefore, the conscious proprioception for the entire contralateral half of the body and head eventually reach the thalamus. Finally, the nerve fibers arising in the thalamus are dispersed to the cerebral cortex in the primary sensory area. Throughout the course of this pathway (the medial meniscal system) exists an organized pattern so that each part of the body is recognizable in topographical relationship to the other parts. This is true of the majority of nerve pathways and is of utmost importance in the development of spinal signs. Since the medial lemniscal system does not decussate until reaching the medulla, damage within the spinal cord will result in signs on that side of the body. However, after crossing to the opposite side of the brain stem in the medulla, a lesion will affect the contralateral side of the body. Thus, the level of the lesion is an important

fixture in interpreting the results for this test. As I have mentioned, there is a topographical representation of the body in the medial lemniscal system. As the fibers are added to the fasciculus gracilis (and cuneatus further forward), they build up on the midline from medial to lateral so that the coccygeal segments are added first, sacral segments second, lumbar segments third, and so on. Therefore, the coccygeal representation is most medial and cervical representation most lateral. Once the fibers decussate, the opposite representation exists with the coccygeal segments being the most lateral. In the spinal cord, this topographical pattern plays a major role in the signs that are seen. For example, ventral compression of the spinal cord, as would occur with a disc protrusion, will cause upward pressure upon the ventral funiculus. However, the dentate ligament, which attaches the spinal cord to its surrounding meningeal structure on each side, will be stretched so that the major force will be exerted upon the medial position of the dorsal funiculus and the junction between the lateral and dorsal funiculus. Thus, the first pathway damaged will be the medial lemniscal system especially in the most caudal representation.

As another example, however, if the spinal cord has come under pressure from a nerve root tumor (such as a neurofibroma) encroaching upon the cord via the dorsal nerve root, these forces will be exerted on the most lateral structures first causing proprioceptive deficits in the more cranial areas first. Therefore, the way in which the medial lemniscal system is affected by the disease process can indeed aid in defining the location and nature of the pathological condition. Although this may seem to be reading an awful lot into such a minute difference, the ability to make such conclusions is not only clinically feasible but also extremely important. Setting aside this system for the present, one should remember that this pathway is proprioception a sensitive indicator of spinal cord dysfunction. If the question as to whether an animal has an arthritic disorder versus a neurological condition (as the differential diagnosis between hip dysplasia and degenerative myelopathy of German Shepherds), the presence or absence of conscious proprioception may be the deciding factor. If no deficit exists in conscious proprioception, the condition probably isn't neurological in origin. Conversely, if there is a deficit in conscious proprioception then the condition most likely is due a to nervous system disease.

The **extensor postural thrust reaction** is elicited by lifting the animal off the ground and lowering the animal rear legs first until the rear legs gently touch the surface. Normally, the animal will, upon touching the ground with the rear feet, move the legs underneath him in an effort to begin to bear his weight. Lowering the animal too fast can interfere with the results by bringing local spinal reflexes into play, but careful performance of the test will give consistent results. The reflex tests whether afferent information can travel up the spinal cord to the higher centers and efferent information can travel down the spinal cord from the higher centers to initiate motor movements. Some of this information travels within the medial lemniscal system and other impulses ascend in the spinothalamic pathway (and a few other minor pathways), so the extensor postural thrust reaction provides only relatively crude information about the function of the spinal cord. Due to the diffuse manner by which the information ascends to the higher centers, and returns from them, little localization value can be gained. Also, it can be difficult to rely upon the results in weak patients who respond poorly anyway. Even so, the extension postural thrust reaction can, at times, be an important part of the neurological examination.

The **placing reaction** is similar to the extensor postural thrust reaction in that it provides general information about the nature of the spinal higher center connections. Again, this

information is rather crude and travels by diffuse mechanisms; but the information is quite useful at times. Placing reactions should be performed both as tactile placing and visual placing responses. It is best to perform the tactile response prior to the visual response as animals quickly learn what is happening and will many times lift the feet each time the examiner moves them in the same manner as when performing the test. However, one can usually outsmart the patient by turning around or presenting a different surface than previously used in the test. One must carefully perform and carefully interpret the results; but, by doing so, reliable results can be obtained. The tactile response is performed by blindfolding the patient (or covering the eyes with your hand) and move the animal slowly toward the edge of a surface, such as the table top, so that the dorsal surface of the foot brushes against the edge. Normally, an animal will hold the foot up and place it onto the top of the surface. Both forelegs are tested individually and the results noted. If the animal fails to respond, then a lesion should be suspected somewhere between the cerebral cortex and the lower cervical spinal cord. After the tactile response has been carried out, the visual response is performed similarly but allowing the animal to visualize the edge and surface. Ordinarily, the patient will raise the foot over the edge and place it firmly upon the surface. It is possible to test the visual field to a slight degree by presenting the edge and surface from different angles. Again, each foreleg is tested. The results, if abnormal (i.e. the animal doesn't place the foot upon the surface), suggests that a lesion is present in the visual pathways to the cerebral cortex, in the connection between the visual and motor cortexes, or the motor outputs to the cervical plexus. By carefully considering the results of both the tactile and visual placing responses, it is possible to determine, in a general sense, where the lesion may be. That is, if both are abnormal, then the defect is probably in an area common to both responses. If only one is abnormal, then the lesion is probably in an area that is uncommon to the two responses. One must look at other neurological signs to make a definitive decision as to where the lesion exists. One should remember that the animal must be able to see to check the visual response and also must be able to move the forelegs or no response would be expected. Although some suggest the performance of this test in the rear legs also, placing reactions are of little value when tested in the rear legs. Therefore, I only check this reaction in the forelimbs where the results are most meaningful.

The **hopping reaction** is performed by holding the animal so that only one leg is in contact with the surface and the animal is using the leg to support a substantial amount of its body weight. The examiner can, then, move the animal in a forward, backward, medial and lateral direction to assertion the animal's ability to move that leg in a hopping manner so that the animals weight bearing is maintained. By slowly moving the animal in various directions, an assessment of the animal's ability to perform this reaction can be made. The pathways which carry this information from the leg to the higher centers and return to the LMN's from the higher centers are, again, fairly diffuse in nature making the exact placement of a lesion in this system quite difficult. However, general information about UMN connection to LMN's can be made.

The **wheelbarrow response** is done by picking up the rear legs (or forelegs) so that only the forelegs bear weight. You should not pick them up too high as this could inhibit the test. The animal is the pushed along (forward or backward) using only the weight-bearing legs. This can help determine or accentuate asymmetry between the motor and sensory functions of the left and right limbs.

The **hemiwalk response**, like the wheelbarrow response, helps look for asymmetries. In this case, both legs on one side of the body are elevated from the floor and the animal is pushed to the side. By comparing the response between the left and right sides, subtle motor and sensory

deficits can be found.

Additional Sensory Pathways:

Unconscious proprioceptive information is carried in four paired pathways, the dorsal, ventral and rostral spinocerebellar and spinocuneocerebellar tracts. The dorsal spinocerebellar and the spinocuneocerebellar tracts appear to be similar. The former for the caudal part of the body and the latter for the neck and head. Both travel ipsilaterally and enter the cerebellum through the caudal cerebellar peduncle terminating in the anterior lobe of the cerebellum. In the case of the dorsal spinocerebellar tract, information enters the spinal cord by way of the dorsal nerve root, travels cranially or caudally in the substantia gelatinosa, and penetrates the dorsal horn to synapse in Clark's column (a nuclear group within the gray matter). The second order neuron in this pathway passes out into the dorsolateral aspect of the lateral funiculus. In the case of the spinocuneocerebellar tract, axons pass in the nerve roots and follow the course of the fasciculus cuneatus up the spinal cord in the ventrolateral aspect of the dorsal funiculus until they synapse in the accessory cuneate nucleus. The ventral and rostral spinocerebellar tracts also appear to be similar with the former for the caudal half of the body and the latter for the cranial half. The rostral spinocerebellar tract passes ipsilaterally up the spinal cord, some of its fibers entering the caudal cerebellar peduncle while the remainder transverse the brainstem to enter the cerebellum with the ventral spinocerebellar tract by way of the rostral cerebellar peduncle. The ventral spinocerebellar tract is made up of fibers which enter locally through the nerve root, synapse in the dorsal gray matter and cross to come up the contralateral spinal cord in the ventrolateral aspect of the lateral funiculus. These fibers pass up to the mesencephalon where they re-cross to enter the cerebellum by the rostral cerebellar peduncle.

Unconscious proprioception cannot be tested in a paralyzed animal, for it can be assessed only during locomotion. When the animal is walking, signs of spinocerebellar dysfunction are manifested by dysmetria, either hyper- or hypometria. Since the spinocerebellar tracts are so peripheral in the spinal cord, they are affected early in many spinal cord disorders.

Pain sensation is carried in the lateral spinothalamic tract. Unmyelinated fibers from pain, pressure and thermoreceptors enter through the nerve roots and pass 1-2 segments caudally and 3-4 segments cranially in the substantia gelatinosa. These then penetrate to synapse in the gray matter of the dorsal horn. Some fibers innervate locally the motor neurons of the spinal segment (including those on the contralateral half of the spinal cord), while the remainder of these second order neurons pass, for the most part, across the mid-line in the ventral white commissure to build up on the contralateral spinal cord in the ventromedial aspect of the lateral funiculus. The spinothalamic tract then proceeds cranially where the sensation for the head is placed in the pathway by way of the spinal tract of CNV until it terminates in the thalamus. Along the way, many branches are given off in the reticular formation which assist in altering the cortex through the reticular activating system.

Pain is an extremely important biologic sensation. It alerts animals to hostile conditions in the environment. It makes adaptive sense that this pathway travels up the contralateral spinal cord, since if the leg is immobile from paralysis, it is important to be able to feel it, so the opposite limb can be used to get away from environmental threats. On the other hand, if a limb is moving, it may leave a hostile environment before extensive damage might be done. Superficial pain can be tested by pinching the webbing between the toes; however, deep pain is best tested by clamping a hemostat on the joints of the digits so that the periosteum will be stimulated.

Withdrawal of the limb is only a spinal reflex. Stimulation of the lateral spinothalamic tract and subsequent transfer of information to the cerebral cortex will result in a behavioral response. This may be crying, snapping or change in autonomic activities. Unless one or more of these behavioral responses is seen, deficiency of pain pathways must be considered. In some cases, palpation elicits an excessive reaction called hyperpathia.

Hyperpathia often indicates a local area of painful response. This response says that the animal is overly sensitive or painful at the site. By lightly stroking the skin (or using a pin to stimulate local skin responses), a local reaction can be found, called *hyperesthesia*. Hyperesthesia can be hyperpathic or not. In some cases, only an increased local reaction of the skin will be seen without any other behavioral response. This indicates the local nerve root and subsequent dermatome is irritated and is often present at the edge of a lesion. The cranial edge may be hyperesthetic and hyperpathic, while the caudal edge will be hyperesthetic only. The **panniculus response** is a unique reaction in quadrupedal animals. The cutaneous trunci muscle is well developed and can be wiggled in response to stimulation. This probably developed as a result of the need to scare flies and other biting insects from the animal's back. They cannot reach their arms behind them. The tail does not reach far enough forward. Upon stimulation of the dermatome, information is carried up the spinal cord (above L4 usually) to the origin of the lateral thoracic nerve. The motor units of this nerve are stimulated to result in reflex contraction of the cutaneous trunci muscle. To test the panniculus response a hemostat is used to pinch the skin hard enough to get the response. A change in the level of stimulation needed or the loss of the response caudally indicates there is a lesion 1-2 segments cranially. (This is due to dermatome overlap.) By utilizing local hyperpathia, local hyperesthesia and the panniculus response lesions in the thoracolumbar region of the spinal column can be localized.

Motor Pathways (Upper Motor Neuron Tracts):

Animals possess many of the same motor pathways as in human beings; however, the relative importance of the cerebral cortex and its pathways is less in most domestic species. Animals have locomotor automatism, meaning that the basics of walking are hardwired into the spinal cord of domestic species. The brainstem adds the remaining ingredients for rudimentary, voluntary locomotor activity. The cerebellum adds smoothness while the cerebral cortex provides behavioral direction. While a normal gait requires a functioning spinal cord, brainstem, cerebellum and cerebral cortex, complete paralysis usually cannot occur unless the brainstem or spinal cord are affected.

Corticospinal and Rubrospinal tracts. These pathways, one from the motor cortex and the other from the red nucleus of the mesencephalon, travel down the spinal cord in the dorsomedial portion of the lateral funiculus. Their fibers penetrate into the gray matter and through internuncial neurons alter the activities of flexor muscles of the digits. It is my opinion that the corticospinal and rubrospinal tracts developed around food prehension, the corticospinal tract being a later development over the rubrospinal tract. In human beings and other animals (such as sea otters and raccoons), the corticospinal tract has direct synaptic contacts with alpha motor neurons providing extremely fine motor control of digital movements. This is necessary in these species to manipulate their food. In horses and cows, the loss of corticospinal (and corticobulbar) tracts results in inability to manipulate the lips and tongue. In small animals, this deficit is most seen in the fore legs, which are used to play with and paw their food and toys. The corticospinal tract can be tested by performing the **Babinski's response**. To do this, the leg

is extended and digital pressure is applied medial and lateral at the metacarpal (or metatarsal) - phalangeal joints. A blunt instrument is then stroked along the back of the metacarpals from medial to lateral and distal to proximal. A flaring or dorsal extension of the digits is positive. This should be repeated with the leg flexed. In animals, a positive Babinski's sign in either leg position indicates corticospinal pathway damage.

Superficial cutaneous responses such as the cremaster response, vulvar response, preputial response, or umbilicus response also test the descending corticospinal tract and can help identify subtle lesions in this motor system. They can also help localize the disease since they leave the spinal cord at different locations. For example, if the umbilicus response is absent, the lesion is above the T12 spinal segment. The umbilicus response is determined by gently stroking the skin of the abdomen with the blunt end of a reflex hammer from the umbilicus outward. A normal reaction is for the muscle to tense slightly in the direction of the stimulus. The cremaster and vulvar responses are performed by gently stroking the inner thigh near the pubis and observing movement of the vulva toward the stimulus or contraction of the cremaster muscle on the side of stimulation. The preputial response is performed by stroking the abdominal surface away from the prepuce and observing a slight deviation of the prepuce in the direction of the stimulation. Loss of these responses indicates damage of the corticospinal system.

Vestibulospinal tract. The vestibulospinal tract runs in the middle of the ventral funiculus and is tonically active. It innervates extensor muscles and resists gravity. As such, muscle strength and the ability to stand are managed by the vestibulospinal tract. Testing the function of this spinal pathway can be done by determining the animal's ability to stand and walk. In addition, pressure can be applied over the back or shoulders of the animal to examine motor strength. By lifting one leg, difference in strength between each leg can be determined.

Lateral Reticulospinal tract. The lateral reticulospinal tract originates in the medulla of the brainstem and is the major inhibitor tract of the spinal cord. As such, the majority of the inhibitory control of locomotion occurs through this pathway. When the lateral reticulospinal pathway is damaged, the resultant disinhibition of motor neurons results in increased reflex activity, spasticity and impaired motor movement. Damage to this pathway can be assessed by testing tendon tap responses, for the presence of crossed extensor responses and by the presence of an accentuated extensor thrust reflex.

Medial Reticulospinal tract. Originating in the pontine reticular formation, the medial reticulospinal tract is a facilitatory pathway. In conjunction with the lateral reticulospinal tract, it regulates movement. Since it is facilitatory in nature, it represents the "on switch" while the lateral reticulospinal tract is the "off switch". Removal of the "on switch" results in diminished reflexes and a lower motor neuron-like, upper motor neuron dysfunction. Since the medial reticulospinal tract is protected by lying deeply in the medial portion of the ventral funiculus, damage to this tract is usually outweighed by damage to the laterally located lateral reticulospinal tract. As such, most spinal cord lesions show increased reflexes typical of upper motor neuron disease. Selective damage of the medial reticulospinal tract; however, can occur.

Additional Motor Reflexes:

The **crossed extensor reflex** is a normal spinal mechanism which is inhibited from being demonstrated by the lateral reticulospinal tract. When upper motor neuron damage occurs, this reflex can be seen when the animal is in lateral recumbency. By gently flexing the toe of the down leg, creating in that leg a progressive painful stimuli, the upper leg is observed for

extension which occurs while the down leg is flexing. This should be repeated on both sides for all the legs. The side which extends has the damage. The presence of a crossed extensor reflex immediately after an injury indicates severe upper motor neuron damage. The development of a crossed extensor reflex 7-10 days following an acute injury may indicate normal accentuation of spinal automatism following injury. A crossed extensor response in a walking dog usually indicate chronicity of the disease process. In addition, a crossed extensor reflex in all four legs in a walking dog indicate a lesion in the low medullary to high cervical spinal cord. Crossed extensor responses are often exaggerated in older animals. This is thought to be due to decreased tone of descending motor control pathways from the cerebral cortex which lead to alterations in the activity of the brainstem and an apparent upper motor neuron problem at the spinal cord level.

The **extensor thrust reflex** is initiated by spreading the foot pads from the ventral surface and pushing slightly toward the spinal column. Normally, an animal will push against your force. If the extensor thrust reflex is exaggerated, the animal may kick out upon even slight stimulation. The significance of this reflex is similar to that of the crossed extensor reflex.

The **Schiff-Sherrington response** is seen when the animal is in lateral recumbency. With damage to the propriospinal tract (the peri-gray white matter in the T3 to L3 spinal cord), the front leg exhibit extensor hypertonicity. The fore legs are not paralyzed; but, when left alone, will extend. The other component of the Schiff-Sherrington response is paralysis caudal to the lesion. This is due to the damage of the other white matter tracts. This is generally a sign of severe spinal cord damage, if both fore leg extensor hypertonicity and rear leg paralysis are seen, since the propriospinal tract is so deeply located within the white matter.

Reflex Testing:

Reflex examination of tendon (muscle stretch receptor) responses can be performed upon any accessible tendon or muscle belly. In general, the animal is laid on it's side so that the muscle can be relaxed. In large animals or animal that resist this, the leg can be elevated and supported by the examiner. The joint across which the reflex is to be tested is flexed or extended to put tension upon the tendon or muscle belly. (This makes certain that the load on the stretch receptor is "standardized".) The tendon is then struck with a reflex hammer and the reaction of the muscle observed and felt. (A finger or other "reflex" tester can be used, but consistency is what allows accurate interpretation of the results.) It is best to check both legs (even in lateral recumbency), although the "free" or up leg is what is recorded. The reflex can be graded from 0 to 4+ based upon the response: 0 = areflexia; 1+ = diminished reflex; 2+ = normal reflex; 3+ = hyperactive reflex; and, 4+ = hyperactive reflex with clonus. Reflexes may be altered by the mental state of the dog, size of the dog, or the disease process.

Foreleg Reflexes

Triceps - Segments C7-T2

Biceps - Segments C6-C7

Extensor carpi radialis - Segments C7-T2

Digital flexors - Segments C7-T2

Withdrawal - Segments C6-T2

Rear Leg Reflexes

Patellar - Segments L4-L5

Achilles (gastrocnemius muscle) - Segments L6-S2

Cranial tibialis - Segments L6-S2

Sciatic Notch - Segments L6-S2

Withdrawal - Segments L6-S2

Miscellaneous Reflexes

Anal - Segments S1-S3

Volvo- or bulboanal - Segments S1-S3

Jaw - CN V (mesencephalic trigeminal nucleus)