**THE NEUROLOGIC EXAMINATION**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 as 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 conscious, 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**Cranial Nerve Examination**:**

**CNI**CNI**,** or the olfactory nerve, is checked by moistening a wisp of cotton with a volatile oil. this can be positioned near the animals 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 animals "ability to smell" may be detected. In many ways, it is unfortunate that we cannot tell more about an animals' 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**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**CNIII, IV, and VI, or the oculomotor, troclear, 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 troclear 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, darken 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 trochear 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**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 animals 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 nasopharnyx 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 find degree. As I have eluded 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 to prehend 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**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**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**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 timpanic 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**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**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** 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 in 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 gracilus (thoracolumbar) and fasciculus cuneatus (cervicothoracic). These nerve fiber synapse in the medulla in their respective nucleic 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 important 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 gracilus (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 menningeal 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** 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** 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** 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** 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**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 bewteen the left and right sides, subtle motor and sensory deficits can be found.

**Additional Sensory Pathways**Additional Sensory Pathways**:**

**Unconscious proprioceptive** 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 paralyze 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** Pain sensation is carried in the lateral spinothalamic tract. Unmyelinated fibers from pain, pressure and thermoreceptors enter through the nerve roots an 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 make 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 reation called hyperpathia.

***Hyperpathia*** Hyperpathia often indicates a local area of painful response. This response says that the animal is overly sensittive 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. 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** panniculus response is a unique reaction in qudripedic 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 bitting 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** 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**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 deficits 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**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 decending 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 absence, the lesions is above the T12 spinal segement. 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 muslce to tense slightly in the direction of the stimulus. The cremaster and vulvar responses are performed by gently stoking 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**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**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**Medial Reticulospinal tract**.** Originating in the pontine reticular formation, the medial reticulospinal tract is a facilitory pathway. In conjunction with the lateral reticulospinal tract, it regulates movement. Since it is facilitory 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**Additional Motor Reflexes**:**

The **crossed extensor reflex** 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** 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** 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 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+ = diminshed 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***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*** Rear Leg Reflexes

Patellar - Segments L4-L5

Achilles (gastronemius muscle) - Segments L6-S2

Cranial tibialis - Segments L6-S2

Sciatic Notch - Segments L6-S2

Withdrawal - Segments L6-S2

***Miscellaneous Reflexes*** Miscellaneous Reflexes

Anal - Segments S1-S3

Volvo- or bulboanal - Segments S1-S3

Jaw - CN V (mesencephalic trigeminal nucleus)

**SELECTED NEUROLOGIC DISEASES**SELECTED NEUROLOGIC DISEASES

Neurologic diseases can be complex in terms of their diagnosis and therapy. On the other hand, through evaluation of the patient, observation of its behavior and performing specific diagnostic tests, a clear clinical picture can be formulated. Acupuncture probably works through its effects upon the nervous system and it is, therefore, reasonable to assume that the nervous system is affected by acupuncture. In fact, acupuncture probably needs an intact nervous system to work. In this course, we will review the signs of neurologic disease and discuss both the TCM and Western diagnoses which may respond to acupuncture. Sample therapies will be provided as a guide in handling patients with neurologic disease. In some cases herbal therapy may be more beneficial, in the long run. However, the information on treatment of neurologic diseases by TCM will be excluded fro examination purposes, except where specifically noted.

Whenever looking at a new patient, it is important to determine whether they have a neurologic disease. This can often be determined by observing the patient in its environment, watching it gait and performing some simple tests. A history can also be helpful, since seizures are a sign that the nervous system (cerebral cortex) is involved, even if there are no other signs. Paralysis of a part of the body can certainly indicate neurologic disease. The presence of dysmetria, conscious proprioceptive deficits, tremors, head tilt, and nystagmus are other signs which can be seen with various neurologic diseases. Other signs may be seen, but can be non-specific or occur with non-neurologic diseases, too.

Knowing that the patient has a neurologic disease and where it is located will help determine the likely causes of the problem. Coupled with a TCM diagnosis, the patient can be monitored for progress and the clients informed as to the prognosis and response to therapies initiated. Some acute conditions can still benefit from a Western medical approach in combination with TCM, while some chronic conditions may respond better to TCM. Combining knowledge of both TCM and Western medicine will probably help the patients better than any single approach.

**Neurologic History & Mechanisms of Disease**Neurologic History & Mechanisms of Disease**:**

***The Neurologic History***The Neurologic History

Part of the minimum data base for evaluation of any proposed neurologic patient is the neurologic history. Not only can this help describe the type of condition and possible causes of the problem, it can also help confirm that the problem is a neurologic disorder. It can be one of the most important parts of the initial examination, leading to the formation of the appropriate differential diagnosis. The owners description may lead to determining the exact nature of the problem, how long it has been present and whether the problem has been progressive.

**Signalment**Signalment**:**

The signalment includes the species, breed, age, sex and color. While many conditions affect all animals, certain diseases are unique to some species and even to certain breeds of that species. Wobbler’s disease is most common in the horse and dog. Moreover, in dogs, it is most often recognized in young Great Danes and older Doberman Pinchers. One would not think of feline leukemia, if treating a dog.

The **age** of the animal can also be important. Younger animals are more prone to congenital problems, infections and toxicities. Older animals are more likely to have degenerative, metabolic, infectious and neoplastic diseases.

The **sex** and **color** of the patient can alter the differential list as well. Hypocalcemia is more common in females around the time parturition. Mammary neoplasia is more common in females, while prostatic disease is most common in male dogs. Blue-eyed, white cats are often congenitally deaf.

**Specific History**Specific History**:**

The diet, exercise, living conditions (outdoor or indoor), past illnesses, vaccination records, and medications can all be important in developing the differential diagnosis. If the diet is improper, nutritional or secondary metabolic diseases may develop. Animals who lack exercise may hasten the development of degenerative diseases. Having access to other animals and potential trauma from living outside may increase the risk of infectious or traumatic disease. Seizures secondary to canine distemper generally occur after the patient has recovered from the original infection. Lack of preventative medication (such as heartworm prevention) may lead to neurologic symptoms secondary to developing the disease. On the other hand, certain medications may allow manifestation of a previously sub-clinical problem. For example, certain heartworm preventatives can lower the seizure threshold. Treatment with aminoglycoside-antibiotics can lead to disorders of cranial nerve VIII. Occurrence of the disease process following pesticide application or the availability of such pesticides may help determine the nature of intoxication.

***Mechanisms of Disease***Mechanisms of Disease

The underlying causes for neurologic diseases are similar to those causes for disease within the body in general. The basic mechanisms of neurologic disease are congenital, inflammatory, metabolic, toxic, nutritional, traumatic, vascular, degenerative, neoplastic and idiopathic. (See Table 1) **Congenital** diseases will, for the most part occur in young animals or in older animals who de-compensate for the condition. The most common **metabolic** diseases in small animals are hypoglycemia and hepatoencephalopathy. The most common **nutritional** disease is thiamine deficiency. **Toxicities** in small animals are usually secondary to organophosphate intoxication, lead poisoning or ethylene glycol ingestion. Knowing the basic mechanisms of disease can help identify the cause of the patient’s neurologic disease.

|  |  |
| --- | --- |
| **Table 1. Mechanism of Neurologic Disease and Some Common Examples.** | |
|  | |
| **Congenital Disorders** | Hydrocephalus, True Epilepsy, Cerebellar Hypoplasia, Congenital Deafness, Vertebral Column or Neural Tube  Defects, Lysosomal Storage Diseases |
|  | |
| **Inflammatory (Infectious) Disorders** | Viral Infection (Canine distemper, Feline infectious peritonitis, Feline leukemia, Panleukopenia, Rabies), Bacterial Infection (meningitis, discospondylitis, Lyme’s disease), Fungal Infection (cryptococcosis, aspergillosis), Protozoal Infection (toxoplasmosis, neosporidiosis), Rickettsial Infection (Rocky Mountain spotted fever, ehrlichiosis), Granulomatous Meningoencephalitis, Polyradiculoneuritis, Myasthenia gravis, Polymyositis |
|  | |
| **Metabolic Disorders** | Hypoglycemia, Hepatoencephalopathy, Electrolyte Disturbances (hyper or hypocalcemia), Hypoxia, Hypothyroidism, Osmolality Disturbance, Acid-Base Disturbance |
|  | |
| **Toxic Disorders** | Organophosphates, Lead, Ethylene glycol, Chlorinated hydrocarbons, Aminoglycoside antibiotics |
|  | |
| **Nutritional Disorders** | Thiamine deficiency, Vitamin E deficiency |
|  | |
| **Traumatic Disorders** | Head injury, Spinal Cord injury, Traumatic Disc rupture, Peripheral Nerve injury |
|  | |
| **Vascular Disorders** | Fibrocartilaginous infarction, Septicemia, Vasculitis |
|  | |
| **Degenerative Disorders** | Degenerative myelopathy, Intervertebral disc disease, Cerebellar degeneration |
|  | |
| **Neoplasia** | Gliomas, Astrocytomas, Oligodendrogliomas, Meningiomas, Neurofibromas, Metastatic neoplasia |
|  | |
| **Idiopathic Disorders** | Cranial nerve syndromes, Self-mutilation syndrome, Acquired epilepsy |

Diseases may be symmetrical or asymmetrical. While metabolic, nutritional and toxic disorders are almost always symmetrical, inflammatory, traumatic, vascular and neoplastic diseases are almost always asymmetrical. This can help rule/out certain diseases from the differential. In addition, traumatic and vascular diseases are more commonly acute and non-progressive; whereas inflammatory, degenerative and neoplastic diseases are either acute or chronic, progressive diseases. (See Table 2.)

|  |  |
| --- | --- |
| **Table 2. Onset and Progression of Disease Mechanisms** | |
|  | |
| *Acute, Non-progressive* | |
|  | 1. Traumatic Disorders  2. Vascular Disorders |
| *Acute, Progressive and Symmetrical* | |
|  | 1. Metabolic Disorders  2. Nutritional Disorders  3. Toxic Disorders |
| *Acute, Progressive and Asymmetrical* | |
|  | 1. Inflammatory (Infectious) Disorders  2. Neoplasia |
| *Chronic, Progressive and Asymmetrical* | |
|  | 1. Inflammatory (Infectious) Disorders  2. Degenerative Disorders  3. Neoplasia |

**Localization of Lesions**Localization of Lesions**:**

One of the important aspects of evaluating any neurologic patient is to determine the location of the lesion. Luckily, the function of the nervous system is intimately tied to its structure. As such, when a function is lost, the structure involved is uncovered. Signs of neurologic disease can be divided into those representing diseases above the foramen magnum (head signs) and those below the foramen magnum (spinal cord signs). Head signs include seizures, head tilt, cranial nerve deficits, whole body and head tremors, and ataxia. Spinal cord signs include quadriparesis and paraparesis. The peripheral nervous system shows signs consistent with the distribution of the nerve involved. (See Table 3) Once the disease process is localized, the differential diagnosis can be made and the diagnostic approach determined.

|  |  |  |
| --- | --- | --- |
| **Table 3. Neurologic Signs and Lesion Location.** | | |
|  | | |
| ***Neurologic Sign*** | | ***Location of Lesion*** |
|  | | |
| Change in Behavior or Personality | | Cerebral Cortex, Thalamus, Hypothalamus |
|  | | |
| Seizures | | Cerebral Cortex, Thalamus, Hypothalamus |
|  | | |
| Visual Dysfunction | | Retina, Optic nerve, Visual pathways, Cerebral Cortex |
|  | | |
| Signs of Cranial Nerve Dysfunction | |  |
|  | Anisocoria | Sympathetic or Parasympathetic innervation |
|  | Strabismus | CN III, CN IV, CN VI |
|  | Dropped jaw | CN V |
|  | Changed facial sensation | CN V |
|  | Paralysis of eyelid, lip or ear | CN VII |
|  | Dysphagia | CN IX, CN X |
|  | Megaesophagus | CN X |
|  | Laryngeal paralysis | CN X, CN XI |
|  | Paralysis of tongue | CN XII |
|  | Head tilt, circling, nystagmus | CN VIII (vestibular system) |
|  | Deafness | CN VIII (auditory system) |
|  | | |
| Incoordination of the Head and Body | | Cerebellum |
|  | | |
| Paraparesis or Paraplegia | | TL Spinal Cord |
|  | | |
| Paralysis of one Limb | | Peripheral nerve |
|  | | |
| Flaccid Anus, Tail and Bladder | | Cauda Equina |

**Ancillary Diagnostic Tests for Neurologic Patients**Ancillary Diagnostic Tests for Neurologic Patients**:**

After determining that the patient has a neurologic disease, localizing the disease process, and forming a differential diagnosis, a diagnostic plan can be developed. This will include tests to ascertain the nature of the neurologic disease, but also include tests to evaluate any discrepancies in the physical examination. Some test should be performed on every neurologic patient while other tests must be selected based upon the location of the neurologic lesion or lesions. The former tests are called the minimum data base.

**The Minimum Data Base**The Minimum Data Base**:**

A **complete blood count** (CBC) including a measure of chronic inflammation such as plasma fibrinogen should be performed on all patients. The presence of polycythemia or anemia, the presence of alterations in plasma proteins and the presence of inflammatory disease or possibility of disseminated intravascular coagulation (DIC) can be assessed, initially, through the CBC. The presence of reduced or elevated white blood cells (WBCs) may indicate infection with viral or bacterial pathogens. Myeloproliferative diseases may produce characteristic changes in the WBC. Increases in circulating nucleated red blood cells (RBCs) may indicate lead poisoning or the presence of hemangiosarcoma.

**Serum chemistry profiles** allow screening for metabolic and toxic conditions which could result in neurologic sequela. Since any disease which effects the body can affect the nervous system, wither directly or indirectly through metabolic intoxication, assessment of the bodies health through screening tests is important in understanding neurologic disease. As will be seen in seizure disorders, the changes reflected in the chemistry profile may help differentiate between an active seizure disease and epilepsy. To this end, the electrolytes (Na, K, Cl, Ca and PO4) are important in muscle and nerve strength and reactivity. Assessments of BUN, cholesterol and albumin can help assess liver function. If all of these parameters are low, one should suspect a portosystemic shunt with diminished liver function. Elevations of cholesterol may help suggest endocrine abnormalities such as hypothyroidism or Cushing’s disease. Elevated globulins might indicate autoimmune disease or, in the case of cats, the presence of feline infectious peritonitis.

Additional serum chemistries beyond routine screening tests may be indicated based upon the location of the lesion and the nature of the neurologic disease. For example, in seizures, all cases should also have serum cholinesterase levels run (to rule out organophosphate intoxication) and serum bile acid levels determined (to rule out liver dysfunction and as a base-line for possible future examines after anticonvulsant medications have been started). Dogs and cats with muscle pain or weakness may need additional serum muscle enzyme tests and determination of serum T4, T3 and TSH concentrations.

A **urinalysis** can help complete the assessment of the patient’s health. Since many neurologic patients exhibit urinary retention or incontinence, this can be important in monitoring for urinary tract infection. Examination for ammonium biurate crystals can help establish diminished liver function, while the presence of calcium oxalate crystals might confirm ethylene glycol intoxication.

Appropriate **parasite screens** should be performed where indicated. Heartworm infection can result in neurologic and muscular diseases in endemic areas. Heavily parasitized young animals can become anemic or hypoglycemic as a result of the infestation, resulting in seizures or other neurologic conditions.

**Routine radiographs** of the chest and abdomen are indicated where disease is suspicioned based upon the physical examination. They may also be indicated in animals over 6-8 years of age, even in the absence of overt physical changes. When neoplasia is on the differential, then they are warranted. If the chest or abdomen are riddled with cancer, extensive workup for the concurrent neurologic disease may not be indicated. In addition to abdominal radiography, **abdominal ultrasound** examination may help determine the cause of the problem, even when abdominal radiographs do not show obvious lesions.

**Other Physical Examinations**Other Physical Examinations**:**

**Fundoscopic examination** may provide important information about the nervous system, since the retina and optic disc are the only parts of the nervous system which can be directly visualized. With CNS infection, active chorioretinitis might be seen. In the dog, this may mean fungal infection (aspergillosis or cryptococcoses), protozoal infection (toxoplasmosis or neosporidiosis) or canine distemper. In cats, it may lead to the diagnosis of cryptococcoses, toxoplasmosis or viral diseases (FeLV or FIP).

**Otoscopic examination** may help in diagnosing problems in the ears and is especially important in assessing animals with vestibular disease.

**Specific Neurologic Tests**Specific Neurologic Tests**:**

Despite the many different disease processes which can assault the nervous system, there are a limited number of tests which can be used to help make the diagnosis. Many are indicated not matter what the nervous system disorder, while others are indicated for specific neurologic conditions. The include CSF tap and analysis, electroencephalogram (EEG), electromyogram (EMG), brainstem auditory evoked response (BAER), skull or spinal radiographs, myelography and magnetic resonance imaging (MRI). Skillful use of these test will, however, allow for the diagnosis of the majority of neurologic conditions. Definitive diagnosis may be achieved by biopsy techniques, including muscle, nerve or brain biopsies.

The **CSF tap and analysis** CSF tap and analysis is one of the most important tests which can be performed in assessing neurologic disease. It might be contraindicated in cases of recent or ongoing hemorrhage and in cases the intracranial pressure is increased. However, in most cases, it provides direct information about the CNS with minimal risk, being less than that of anesthesia. Evaluation of CSF should include pressure (for cisternal taps), protein determination and cytology. Additional test on CSF might be beneficial in certain diseases, such as acetylcholinesterase levels and 2-D electrophoresis in degenerative myelopathy. In cases where infection is suspected, titers can also be helpful in diagnosing the cause. CSF can be collected from the cisterna magna or the lumbar cistern between L5 and L6. For most animals, a 22 ga spinal needle is best for achieving the tap, varying in length between 1.5 to 3.5 inches. Allowing the CSF to flow by gravity and collecting into a syringe as it drips from the hub of the needle, one cc of CSF can be collected for every 10 pounds of body weight. To run routine CSF analysis and titers, requires approximately 1.5 cc of CSF. Cytologic examination plays an important part of CSF analysis. Total counts can be useful, but we have found that close inspection of the “reactivity” of the cells on cytology may be more important than the total count. The best method to perform cytology is with the use of a cytocentrifuge. Since the cells deteriorate rapidly in CSF, cytology and cells counts must be performed within 20 minutes of drawing the sample.

The **EEG**EEG tests the outer 3 mm of the cerebral cortex and measures the electrical potentials between scalp electrodes. It can be used to test the forebrain and is an important diagnostic tool for diseases characterized by changes in behavior and seizures. To perform the EEG, the patient is anesthetized for any other neurologic tests which are being performed and, then, the scalp electrodes are inserted and connected to an EEG machine (a filtered, amplifier connected to a recording device). Once the connections are made, the recording is started and the anesthesia is turned off. The EEG is then recorded while the patient recovers from anesthesia. Performing the EEG in this manner induces some artifacts from the effects of anesthesia (however, these are minimized by using the same anesthesia in all patients and becoming familiar with the artifactual changes). On the other hand, it removes artifacts from EMG activity and movements, typical of awake EEG recordings. The normal EEG has fast, low amplitude activity (15-30 Hz and 5-15 V, respectively). The presence of slow waves (alpha, delta and theta waves) with high amplitude indicates abnormality.

**Electromyographic** examination Electromyographic examination test the integrity of the lower motor unit. The needle EMG is performed by inserting an exploring electrode into the muscle to examine its intrinsic electrical activity. It is best performed under anesthesia, whereby nerve stimulation studies can also be performed. The presence of fibrillation potentials, fasciculation and bazaar high frequency discharges indicates increased irritability of the muscle membrane, occurring in disorders of the motor neuron, motor nerve, neuromuscular junction or muscle. Based upon the distribution of the EMG changes, the location and nature of the neurologic disorder may be indicated. Since muscle membrane irritability requires time to develop following denervation, the needle EMG may be normal for 5-7 days following acute injury of the motor unit.

Another important part of the EMG is determined by electrical stimulation of peripheral nerves. By stimulating at multiple sites along a motor nerve and recording the latency between the stimulation and the beginning of the compound action potential, the motor nerve conduction velocity can be determined. The distance between the stimulating electrodes at the two sites is divided by the difference between the latencies from the 2 sites to give the motor conduction velocity in meters per second (normal conduction is greater than 50 m/s). In addition to motor conduction velocity, repetitive nerve stimulation can be performed. Normally, the muscle can maintain activity at stimulation rates between 5-10 per second. In myasthenia gravis or sub-acute organophosphate intoxication, there is a decremental response to repetitive stimulation. The F wave is a low-amplitude wave seen several milliseconds following the compound action potential and is thought to be produce by antedromal spread of the stimulation pulse to the cell bodies of the nerve where it results in a secondary pulse traveling down the nerve to the muscle. The H wave is another low-amplitude response several milliseconds after the F wave and represents stimulation of the sensory fibers in the nerve and subsequent reflexive stimulation of the motor neurons. Both the F wave and H wave may help examine the integrity of the central connections of the peripheral nerves. In addition to motor nerve conduction velocities, sensory nerve conduction can be measured. The sensory nerve is stimulated and a recording electrode place proximally along the nerve records the passage of the impulse up the nerve. The distance to the recording electrode is divided by the latency of the impulse recording to determine the sensory conduction velocity.

The **BAER**BAER records the electrical activity in the brainstem caused in response to auditory clicks in the ears. The BAER is not affected by sedation or anesthesia, so patients who are fractious can be sedated without affecting the results. The recording is made by placing a ground electrode in the untested ear, a reference electrode in the ear to be tested and a recording electrode over the vertex. The click is introduced in the ear to be tested and the electrical activity generated is averaged to reduce random noise. Generally, 5-7 middle-latency, waves are recorded, representing the transmission of auditory information through the vestibulocochlear nerve, the cochlear nucleus, the nucleus of the trapezoid body, the lemniscal nucleus and caudal colliculus, respectively. The BAER is used most frequency to test young animals for congenital deafness, but may also be used to test the integrity of the brainstem auditory system.

**Neuroradiology and imaging** Neuroradiology and imaging include routine radiographs of the skull and spinal column. All neruo-imaging techniques are best performed under general anesthesia. Routine radiographs of the skull may reveal fractures, congenital defects, otitis media and interna and obvious neoplasia affecting the osseus structures of the skull. Routine spinal radiographs can help identify fractures, congenital malformations, evidence of degenerative disc disease, discospondylitis and neoplasia of the vertebra. However, many times the effects of the bony changes on routine radiographs do not provide sufficient information about the neural damage without the addition of special imaging techniques.

The most common of these techniques is **myelography**, performed by injecting contrast agent into the subarachnoid space through a spinal needle. Most of the time, the injection is made at the lumbar cistern and the contrast agent (Iohexol 180) is allowed to flow forward to fill the subarachnoid space to beyond the lesion. For diseases in the thoracolumbar region 0.33 cc/kg of body weight is used, while 0.45 cc/kg of body weight is used for cervical disease. It is best to use image-intensification to monitor the flow of the contrast agent and the dosage given adjusted to effect. Since most contrast agents are irritative, most neurologist believe they should not be performed in the face of obvious inflammation of the nervous system. In addition, this irritation can result in seizures upon recovery from anesthesia, another reason not to inject more than necessary to fill the subarachnoid space to the level of C1. Giving methylprednisolone immediately following the contrast injection can reduce the incidence of pot-myelographic seizure, probably due to helping to maintain intercellular glucose concentrations.

A number of other special imaging techniques have been applied to neuro-imaging including computer assisted tomography (CAT) scans, radioisotopic brain scans, cerebral angiography and ventriculography. Of these, only the **MRI** provides anatomic detail when examining the nervous system. All portions of the CNS can be imaged by MRI. The MRI provides evidence of increased tissue density and fluid accumulation, demonstrates anatomic shifts in CNS structures, and (coupled with contrast studies) demonstrates breaks in the blood-brain barrier. For CNS neoplasia and for lumbosacral stenosis, MRI is the imaging method of choice.

**Diagnostic Plans**Diagnostic Plans**:**

Although the neurologic tests above can help diagnose neurologic disease, not all are indicated for all conditions. For simplicity, the problems of the nervous system can be broken into 1) diseases above the foramen magnum (diseases with head signs), 2) diseases of the spinal column (diseases of quadriparesis or paraparesis) and 3) diseases of the peripheral nerves and muscle.

For diseases of the headdiseases of the head, the diagnostic plan includes:

1) minimum data base,

2) fundoscopic or otoscopic examination,

3) CSF tap and analysis,

4) skull radiographs,

5) EEG or BAER (EMG if cranial neuropathy), and

6) MRI or CT scan.

For diseases of the spinediseases of the spine, the diagnostic plan includes:

1) minimum data base,

2) CSF tap and analysis,

3) spinal radiographs,

4) myelography,

5) EMG, and

6) MRI or CT scan.

For diseases of the peripheral nerves or musclediseases of the peripheral nerves or muscle, the diagnostic plan includes:

1) minimum data base,

2) EMG,

3) special muscle enzymes, and

4) muscle and nerve biopsy.