Learn to do the neurological examination on a cooperative small breed dog and then you can adapt it to accommodate the size and attitude of other dogs and cats as well as large animals and exotics. When asked how I do a neurological examination on a maniacal aggressive cat or dog my pat answer is: I don’t! However you can make many reliable observations by just observing the animal while it is caged as long as you understand what the normal examination is determining.

Why do the neurological examination?
- To determine if the nervous system is affected in a disease process.
- To establish as accurate an anatomic diagnosis as possible when the nervous system is affected.

Making the anatomic diagnosis should always precede consideration of the differential diagnosis. Despite the tremendous contribution of imaging to neurological diagnosis and one can only guess what innovations lie ahead in the future, the basic hands-on neurological examination is the most valuable cost effective determinant of the clinical diagnosis.

In most cases the anatomic diagnosis is a regional diagnosis and there are essentially 8 regions of the nervous system for consideration.

The prosencephalon (forebrain) - includes the two cerebrums and the diencephalon which is the most rostral part of the brain stem and is comprised of the thalamus and hypothalamus. The pons and medulla are usually considered together as they are the source of the upper motor neuron responsible for the generation of the gait. The cerebellum may be the sole anatomic diagnosis or often is considered together with the pons and medulla. This region is sometimes referred to as the caudal fossa which is the space these 3 anatomical areas occupy. The spinal cord is divided into 4 anatomical regions: C1 - C5, C6 - T2, T3 - L3, L4 - Cd.

The peripheral nervous system components are usually considered collectively as the neuromuscular system, realizing that there are important sensory systems here as well. Any of these 8 areas can be further divided into smaller components but this serves as a starting point in your anatomic diagnosis.

There are 5 components to the neurological examination:

1. Sensorium
The owner is the best observer of any change in the patient's behavior. Significant changes will be obvious but you will often notice subtle changes as you examine the animal. Expressions used to describe these subtle changes include - a vagueness - seems out of touch with reality - is in a world of its own. More acceptable medical terms for the progressive loss of a patient's sensorium are: dullness, lethargy, obtundation, semicoma (stupor) and coma. In my experience the most common site for a focal lesion to cause progressive obtundation or stupor is the diencephalon - presumably from the interruption of the ascending reticular activating system (ARAS) at this level.

2. Gait
The most important aspect of the gait examination is to be able to walk the dog on a non-slippery surface. These are often scarce in most hospitals. If there is no convenient built in carpet in the hospital then purchase a reasonable sized indoor - outdoor carpet that can be rolled out for the exam and rolled out of the way afterwards and can be hosed off in your runs after your patient excretes on it - which is a guarantee. This is just as important for evaluating orthopedic lameness cases. A major objective of the gait evaluation is to determine if a lameness is caused by a neuromuscular disorder or an orthopedic
problem. Lower motor neuron disease can mimic an orthopedic lameness and is often overlooked as the latter problems are so much more common. As you gain experience you will recognize specific gait patterns that suggest the anatomic diagnosis i.e., the "two engine" dog with short strides in the thoracic limbs and long delayed strides in the pelvic limbs that has a C6 - T2 disorder. The following is an attempt to dissect what it is that you are looking for when you evaluate the gait of a patient with a neurological problem. From a neurological perspective, you are assessing the gait for both paresis and ataxia.

A - Paresis (weakness) can be defined as a deficiency in the generation of the gait or the ability to support weight. There are two qualities of paresis - upper motor neuron and lower motor neuron.

1 - Lower motor neuron (LMN) paresis is seen as an inability to support weight and the patient walks short-strided - "lame". Other signs include a tendency to collapse, trembling, bunny hopping, and neck flexion. Thoracic limb support requires neurons in the radial nerve to be intact whereas those in the femoral nerve are necessary for pelvic limb support. Lesions that affect specific peripheral nerves to the limbs excluding the radial and femoral nerves will cause abnormal limb postures but still the ability to support weight.

2 - Upper motor neuron (UMN) paresis is seen as a delay in the onset of protraction (the swing phase) and a longer stride with a variable degree of stiffness - spasticity to the stride. Because the UMN tracts and the general proprioceptive (GP) tracts are adjacent to each other at every level of the spinal cord and caudal brain stem lesions at any of these levels usually will cause dysfunction in both systems, therefore, the gait reflects both deficits. GP lesions cause an ataxia in which the patient loses awareness of where its limbs are in space. This also may contribute to the delay in the onset of protraction and be the cause of excessive flexion, adduction or abduction of the limb during protraction and the tendency to bear weight on the dorsal aspect of the paw often referred to as "scuffing or knuckling".

The pattern of gait observed reflects the loss of both of these functional systems and it is not necessary to distinguish between the two systems for your anatomic diagnosis. Patients that have C1 - 5 lesions and are still ambulatory often have a prolonged stride with the limb kept in extension that appears as if the patient is overreaching its landing site. This is especially evident on turns. Although by strict defintion this is a form of hypermetria, I avoid calling it that as there is a strong tendency to relate any hypermetria to a cerebellar disorder which this is not. I refer to this overextension as overreaching or floating. Cerebellar hypermetria has a sudden bursting quality to the onset of protraction and an overflexion of the joints as opposed to the overextension seen here.

B - Ataxia is incoordination and comes in three qualities-general proprioceptive, vestibular (special proprioceptive) and cerebellar.

1 - General proprioceptive ataxia represents a loss of awareness of where the limbs are in space and was discussed above with the UMN system which it accompanies.

2 - Vestibular ataxia is a loss of balance reflected in a head tilt, and a tendency to lean, drift, fall or roll to one side. The ataxia is often accompanied by an abnormal nystagmus.

3 - Cerebellar ataxia reflects the inability to modulate the gait generating systems in the brain stem resulting in abnormal "uncontrolled" limb movements that usually are excessively abrupt in onset with an overflexion of the limbs on protraction and abnormal sites of limb placement. These excessive movements are usually referred to as hypermetria. This abnormal gait is usually accompanied by vestibular signs with a loss of balance because there are significant components of the central portions of the vestibular system in the cerebellum.

3. Postural Reactions - Muscle Size
I usually assess these at the same time by standing over the patient with both of us headed in the same direction. In assessing muscle size it is important to try to have the patient bearing the same amount of weight on the two limbs that are being compared. I palpate both thoracic limbs simultaneously from proximal to distal and then flex and extend each for range of motion and as an assessment of muscle tone. When I place the paw back on the floor I place it on its dorsal surface to test for its return to a normal supporting position - the paw placement response. I then move caudally palpating the axial muscles and then palpate and move the pelvic limbs in a manner similar to the thoracic limbs and complete it with the paw placement test. To test the hopping responses I then move back to the thoracic limbs and while still standing over - straddling - the patient, I pick up one thoracic limb and hop the patient laterally on the other limb then I shift limbs and hop it back on the first limb. Only hop the dog laterally on the limb. I keep doing this back and forth shifting limbs when the patient reaches the limit of
my stance. I do not move during this procedure and with heavy animals I brace my supporting elbow on my thigh to avoid the strain on my back. The patient does not have to be lifted off the floor for this, only supported so that it is bearing as much weight as possible on the limb being hopped.

To test the hopping responses in the pelvic limbs I stand beside the patient and place my forelimb between the thoracic limbs of the patient so I can lift it up off the floor by its sternum. I then pick up the pelvic limb on the side that I am on and push the dog away from me making it hop on the opposite pelvic limb. I have to change sides to test the other pelvic limb. For heavy animals these hopping responses can be evaluated as you make the patient hemiwalk. I stand beside the patient and pick up both limbs on that side and push the dog away from me. It is important to compare one thoracic limb with the other and one pelvic limb with the other as they are usually faster in the thoracic limbs normally.

These hopping responses essentially test all components involved in voluntary limb movement from sensory receptors in the limb, to ascending spinal cord tracts, to medullary relay proprioceptive nuclei, to thalamic relay nuclei to the thalamocortical pathways, to the internal capsule, to the sensory cortex and the return of the UMN pathways. The latter begin in projection neurons in the adjacent motor cortex, pass into the internal capsule and crus cerebri, to descending UMN pontomedullary systems, into the spinal cord in pyramidal and extrapyramidal UMN tracts, to the ventral grey column LMN, to the muscles in the limb. One might conclude that this is fairly non-specific!! Correct - so why is it so useful? Because first - it tells you if there is an abnormality somewhere in the nervous system and therefore, is a reliable screening test. Second - its importance in localizing lesions is dependent on what else is abnormal. A patient with a normal gait in the environment of your examination that has hopping deficits on one side most likely has a contralateral prosencephalic lesion. This is a very common relationship and may be the only indication of a prosencephalic lesion. This is one of only 3 tests that you can use in your neurologic examination to determine if a prosencephalic lesion is present and on which side. If you have a patient with a head tilt and a mild loss of balance to one side but otherwise has a normal gait and there is a hopping deficit then if the lesion is focal it is central at the medulla and not in the inner ear. Postural reactions are normal with inner ear - peripheral vestibular system - disorders.

Misleading neurologic description - It is important to be clear and precise with your neurologic descriptions. What does it mean when you read a description in a published case study that describes an 8 year-old Beagle dog as having a right hemiparesis? If the author means that this dog has a normal gait but a right side postural reaction deficit then I would make an anatomic diagnosis of most likely a left prosencephalic lesion. If the author means this dog has a right sided gait deficit with a delay in protraction of the right limbs with spasticity and a tendency to float with the right thoracic limb then my anatomic diagnosis is right C1 - C6. It is important to remember that animals with neuromuscular disorders that still have voluntary movements will hop fast as long as their weight is held up because their proprioception is normal. This observation may help you distinguish between a subtle LMN and UMN paresis.

There are many other postural reactions that can be tested but in my experience the hopping responses are the most reliable and they are all that I routinely perform in this examination.

Many clinicians rely solely on what they call the CP - conscious proprioception response. This is an incorrect term as this tests more than just conscious proprioception. The late Ralph Kitchell published a paper in which he made a point of this common mistake describing that in this test there are somatic afferents that are responding to light touch and pressure in addition to the general proprioceptive neurons. In reality the failure to return the paw to its normal position can be caused by a LMN denervation of the digital extensors, an UMN paresis, or a loss of any of the sensory innervation just described. In addition to this lack of specificity it is my experience that there are some normal patients that when their paw is placed on its dorsal surface they will continue to stand on it until you make them move. This paw placement test should not be relied on in the absence of testing the hopping responses.

Recumbent Animals - It is very important in evaluating these patients to pick them up and hold them in a standing position. Get help if it is a heavy patient. By holding them in this position and lifting them up and down you can determine the quality of muscle tone ie., whether they have a flaccid or spastic paralysis as well as determine if any voluntary movements can be elicited. If there are voluntary movements, while still supporting them you can determine the presence and quality of the hopping response.

4. Spinal Reflexes - Muscle Tone

Ideally these spinal reflexes and muscle tone will be diminished to absent in LMN disorders and increased in UMN disease. The degree of hypertonia that results from UMN disease will be determined by the amount the lesion interferes with the upper motor neurons that are inhibitory to extensor motor neurons. It is important to evaluate the tone and spinal reflexes together with the gait abnormality. Dogs can exhibit profound neuromuscular paresis with myasthenia gravis and still have normal tone and reflexes. Similarly some dogs with T3 - L3 lesions often have normal muscle tone and reflexes. For evaluating the spinal reflexes the patient should be placed in lateral recumbency and be as relaxed as possible. The limbs
can be flexed and extended to assess the degree of muscle tone that is present. The only reliable tendon reflex in my experience and the only one that I routinely test is the patellar reflex. Holding the stifle in partial flexion the patellar ligament is struck lightly with a hard object. The human pediatric patellar hammer is the best size for our small animals. Both the sensory and motor components of this reflex are contained in the femoral nerves and their components in the L4, 5 and 6 spinal nerves, roots and segments. If you do not get this reflex in either the recumbent limb or non-recumbent limb do not consider it absent until you can not get it in the other position. For some reason that I do not know, this reflex is occasionally absent on either the recumbent or the non-recumbent side. You only need to get it once to know it is intact. If the patient will not relax you may not be able to elicit this reflex. It is my experience that the other tendon reflexes are not consistently present in normal dogs and I do not routinely test them.

The withdrawal (flexor) reflex is done on both limbs by squeezing a digit with enough pressure to elicit the reflex and a conscious response in a normal patient. Sometimes your digital pressure may be enough. Otherwise use a pair of forceps on the base of the toenail adding enough pressure to get the response or not get it if there is a lesion. Remember that you can have a reflex loss without loss of nociception so you must use care in the amount of pressure you apply to avoid excessive discomfort to the patient and injury by the patient!!

This is a more complex reflex. The sensory neurons tested depend on the digit being tested or the autonomous zone that you select for this stimulus and the motor response involves primarily the sciatic nerve in the pelvic limb (stifle flexion) and its branches, the tibial nerve (digital flexion) and peroneal nerve (tarsal flexion). Beware that the hip flexion that results involves the femoral nerve and most all the ventral branches of the lumbar spinal nerves to the psoas major muscle. An animal with a complete sciatic nerve lesion can flex the hip when the medial aspect of the paw is stimulated (saphenous nerve - sensory branch of the femoral nerve). The segments of spinal cord, roots and spinal nerve ventral branches involved with the sciatic nerve are L6 L7 and S1.

In the thoracic limb there are multiple nerves involved with the withdrawal reflex thus it is a crude test of the entire brachial plexus and cervical intumescence. The sensory nerve or nerves tested depend on the autonomous or cutaneous zones selected. Squeezing the base of the 2nd or 3rd digital nail stimulates the sensory components of the radial nerve dorsally and the median and ulnar nerves on the palmar aspect. The motor neurons involved are in the axillary nerve (shoulder flexion) musculocutaneous nerve (elbow flexion) and median and ulnar nerves (digital flexion). Both the sensory and motor neurons that are involved are associated with the C6 to T2 spinal cord segments - the cervical intumescence.

These flexor responses only require the peripheral nerves and the segments of spinal cord where synapses occur between the afferent and efferent components. A transverse lesion in the spinal cord cranial to these segments that isolates the segments from the rest of the CNS will not cause a loss of these reflexes. They can persist independent of the rest of the CNS.

Nociception

By increasing the amount of pressure on the digit the stimulus becomes a noxious one and in the normal animal will elicit a conscious response. This response is the patient’s manifestation of pain. As an anatomist I try to strictly adhere to the approved nomenclature to avoid ambiguity. This is published in the Nomina Anatomica Veterinaria. No such Bible exists for medical terminology and is sorely needed. Therefore I appreciate it when I am corrected for an improper use of terminology. In my textbook I refer to this noxious stimulus as the pain stimulus which is incorrect. Once again, my neuroanatomical critic Ralph Kitchell pointed out to me the error of my ways and I applaud him for that. Pain is not a sensory modality. Pain is the subjective response of the patient to a noxious stimulus and varies between individual patients and is dependent on many other factors surrounding the origin of the noxious stimulus. We should all adhere to this terminology! Having clarified that - the conscious perception of the noxious stimulus known as nociception is primarily at the level of the sensory (somesthetic) neocortex in the area of the postcruciate gyrus. To reach this level the entire pathway from the intumescence involved with receiving the noxious stimulus to this sensory cortex must be intact. In general when the afferents that have been stimulated by the noxious event enter the spinal cord dorsal grey column, they synapse on projection neurons there. Most of these will cross to form an ascending pathway in the opposite lateral funiculus but some will form a similar pathway on the same side as the source of the stimulus. In reality there are nociceptive pathways in all funiculi. However there are enough that are contralateral that in a cooperative patient with a prosencephalic lesion involving this pathway or the sensory neocortex there will be a degree of hypalgesia in the limbs on the opposite side. Only a transverse spinal cord lesion cranial to the intumescence involved will produce analgesia. Such a lesion in the cervical spinal cord is usually lethal due to the interruption of UMN respiratory tracts. Recognizing a hypalgesia in the limbs and trunk on one side in a patient with a normal gait is one of the 3 tests used to localize a prosencephalic lesion. It is easier to appreciate in the nasal mucosa which will be described with the cranial nerve part of this examination.

Because there is so much variation between animals in their response to noxious stimuli I do not believe I can reliably recognize the difference between the response to a mild and more severe noxious stimulus - referred to incorrectly as superficial and deep pain. Even if I could, I am not convinced it contributes to my ability to make the anatomic diagnosis. Obviously the presence or absence of nociception with severe transverse thoracolumbar spinal cord lesions is important for
prognosis as well as to specifically locate the site of the transverse lesion. There is one more reflex that I usually test and always test when I am concerned about a possible transverse T3 - L3 lesion in the spinal cord. This is the cutaneous trunci reflex - which I have incorrectly called the panniculus reflex. The sensory neurons stimulated by lightly squeezing or poking the skin over the epaxial muscles of the thoracolumbar vertebrae are contained in the dorsal branches of the spinal nerves innervating the skin at about the level of your stimulus. Synapse occurs in the spinal cord dorsal grey column on long interneurons that then project cranially in the fasciculus proprius. These interneurons terminate on LMN cell bodies in the ventral grey column at C8 and T1 which in turn enter the lateral thoracic nerve that innervates the cutaneous trunci muscle causing the skin to twitch. Rarely this reflex can not be elicited in a normal dog. Starting at the L7 region and stimulating the skin over each successive vertebrae the reflex in most animals does not start to about the midlumbar level but there are many individual variations here. In patients with complete transverse T3 - L3 lesions, this reflex will be absent caudal to the lesion - and more specifically about 2 spinal cord segments caudal to the lesion because of the normal short caudal course of the dorsal branches after they leave the spinal nerve. This reflex will also be absent with lesions that affect the lateral thoracic nerve or its origin from the C8 and T1 spinal nerves ie., avulsion of the roots of the brachial plexus, nerve sheath neoplasms of these spinal nerves.

The tail should be moved to assess the tone of its muscles and the anal tone should be determined. The perineal reflex can be performed by mild digital pressure on the anus or with the blunt end of closed forceps or by squeezing the anal or adjacent perineal skin with forceps and observing contraction of the anal sphincter and tail flexion. The degree of stimulus can be gauged to avoid upsetting the patient when this innervation is still intact. This reflex is dependent on the sacral segments and their spinal nerves and the branches of the pudendal nerves. The tail response is dependent on the caudal segments and nerves. LMN bladder dysfunction is often indirectly assessed by loss of the perineal reflex because of similar involvement of sacral segments and the proximal sacral spinal nerves.

5. Cranial Nerves
The cranial nerve exam should be done when the patient is the most relaxed. With very young animals this is often before you handle them at all. In most instances with these young animals - the less restraint the better. For larger patients I prefer to do this cranial nerve exam while standing over the patient as I have been for the postural reactions. For small dogs and all cats I prefer to sit on the floor with my back against the wall - all very comfortable - flex my knees and place the patient with its back lying on my thighs. It is very easy to control the patient this way and especially its head that you are going to examine. Aggressive cats can be rolled in a towel before placing them in this position. The cranial nerve exam can be done "by the numbers" or by region. I much prefer the latter. Either part or all of cranial nerves II thru VIII are evaluated in the region of the eyes.

Menace - Vision - Pupils
I always start with the menace response and cover one eye as I menace the other. This is a learned response and may not occur until 10 to 12 weeks in puppies and kittens in which case I have to use their ability to follow objects moving in their environment to assess vision. Anatomically this is a II - central visual pathway - VII response. The majority of the central visual pathway is contralateral to the eye being menaced. Some normal animals need a mild stimulus to get a response. I usually tap their orbital region with my hand a couple of times before I do the menace. Then be sure you are not too close with your menacing hand so that you avoid long vibrissae or a sudden air movement that stimulate sensory components of cranial nerve V. If I do not get a response then I immediately touch the eyelids and look for the palpebral response to be sure the facial nerve is functioning. If it is not, then I have to look for eyeball retraction or a head movement as a response to the menace if the patient is visual. Occasionally it is necessary to set up a maze of objects in the animal’s environment to see if they can avoid the objects when walking around them.

Immediately following the menace test, the pupil size and response to light should be examined. Some pupil size can be seen in room light - I love cats that have a yellow iris. Most patients have a dark iris which will require some additional light to see the borders of the iris. Hold your pen-light on the midline over the nose to give each eye the same amount of light to look at pupillary size and determine if any anisocoria is present. Then place the light source as close to the eye as possible and if no response occurs move the light around the fundus to be sure all areas are stimulated. After observing this in one eye quickly swing the light into the other eye, observe that eye’s response and then swing the light source back to the first eye and keep repeating this. In the normal patient the pupil will constrict rapidly (depending on the species - this is always slow in horses) and as you move the light source from one eye to the other the pupils in both eyes will stay constricted. This is how I observe the indirect or consensual response rather than try to see the response in the opposite eye while I hold the light in the stimulated eye.

When I am teaching, writing examinations or publications I never use the terms direct response (eye stimulated) or indirect - consensual (the other eye) as these terms can be confusing unless you are very careful in your description and in many publications this care is absent. Avoid this confusion by indicating that when the light is directed into OS what happens to the pupil in OS and what happens in OD and do the same for the light directed into OD.
This light reflex is mediated through the rostral brain stem. The retinal ganglion layer neurons involved with this reflex in each optic nerve presumably are directed at the chiasm either into the opposite optic tract (about 75% dog, 65% cat) and the remainder enter the ipsilateral optic tract. These light reflex processes pass over the lateral geniculate nucleus and enter the dorsal thalamus to synapse on neurons in the pretectal nucleus on that side. The majority of these pretectal neurons project thru the caudal commissure to terminate in the oculomotor nucleus on the opposite side of the rostral mesencephalon. Based on this anatomy, light directed into one eye will have a greater influence on the ipsilateral oculomotor nucleus and the response in the stimulated eye may be more rapid and complete than the indirect response in the opposite eye. This is not always obvious in your examination. You would also expect that a lesion limited to one optic tract would cause a decreased response when the contralateral eye was stimulated but this too may be difficult to appreciate.

**Examples**

If the menace response is absent in one eye with a normal palpebral response and pupils are normal and equal in size and have normal pupillary light reflexes then the lesion causing the unilateral menace deficit is most likely in the contralateral optic tract, lateral ciliary nucleus, thalamocortical fibers, optic radiation part of the internal capsule or visual neocortex primarily in the occipital lobe. This is the central visual pathway for perception. In the dog about 75% of the pathway is contralateral to the eye menaced after the optic chiasm and about 25% remains ipsilateral. Therefore, lesions in this central visual pathway on one side cause a 75% loss of vision in the contralateral eye and 25% loss in the ipsilateral eye but the owners rarely recognize this deficit. The menace test can only determine the contralateral 75% deficit and it is fairly reliable. Even though the contralateral optic tract contains the majority of the pupillary light reflex fibers - assuming that their portion that cross in the optic chiasm is similar to the visual perception pathway - there usually will be no recognizable loss of pupillary light response in the eye tested. This menace test is one of the 3 examinations to determine structural disorders in the prosencephalon.

A patient has normal menace responses. The pupil OD is widely dilated. Light in OD only causes the pupil to constrict in OS. When you cover OD with your hand, the pupil in OS dilates to its full extent.

**Where is the lesion?**

**Answer** - Right oculomotor nerve - parasympathetic visceral efferent fibers, or ciliary ganglion or its ciliary nerve branches. A retrobulbar tumor or abscess could do this.

A patient has no menace OD with a normal palpebral reflex. There is no anisocoria. Light directed into OS causes no response OU (in both eyes or in each eye). Light directed into OD causes a normal response OU. As you swing the light from OD, where the pupil constricted, back to OS, the OS pupil which was constricted from the OD stimulation is now dilating back to its original size. This asymmetry is repeated as you swing the light back and forth between the two eyes. When you cover OD with your hand, the pupil in OS dilates to its full extent.

**Where is the lesion?**

**Answer** - In OS or the left optic nerve. Most of the time with these lesions there is enough room light entering the normal eye to keep the pupil in the abnormal eye constricted. Occasionally it will be slightly larger than the normal pupil in room light. A patient has no menace responses. The pupil OD is widely dilated. Light in OD only causes the pupil to constrict in OS. Light in OS only causes the pupil to constrict.

A patient has no menace response OS with a normal palpebral reflex. Light directed into OS causes no response OU (in both eyes or in each eye). Light directed into OD causes a normal response OU. As you swing the light from OD, where the pupil constricted, back to OS, the OS pupil which was constricted from the OD stimulation is now dilating back to its original size. This asymmetry is repeated as you swing the light back and forth between the two eyes. When you cover OD with your hand, the pupil in OS dilates to its full extent.

**Where is the lesion?**

**Answer** - Right oculomotor nerve - parasympathetic visceral efferent fibers, or ciliary ganglion or its ciliary nerve branches. A retrobulbar tumor or abscess could do this.

A patient is blind OU - no menace OU - with normal palpebral reflexes. In room light the pupils are mildly dilated. Light directed into OS causes the pupils to constrict OU. Light directed into OD causes the pupils to constrict OU.

**Where is the lesion?**

**Answer** - Both eyeballs, optic nerves optic chiasm or optic tracts. The two most common disorders that cause these specific signs are a retinal degeneration (SARDS-sudden acquired retinal degeneration) and optic neuritis.

From my clinical experience it appears that animals with lesions in the sites just described can lose their visual perception and be clinically blind but still have light responsive pupils when a bright light is directed into the eyes. However, in room light there is insufficient light to permit normal constriction. This may reflect that the disease processes involved tend to spare the pupillary light reflex neurons in cranial nerve II or more likely to lose the light reflex completely it is necessary to interfere with the function of all of these neurons whereas vision is lost after a certain threshold percentage of retinal ganglion layer neurons are dysfunctional. In other words the pupillary light reflex neurons are the last to go when lesions disrupt the retina or optic nerve.

Anisocoria can result from many intraocular disorders. Iris atrophy is fairly common in older animals and creates dilated
unresponsive pupils with no interference with vision. Neurological causes of anisocoria include disturbances to cranial nerves II, III and the sympathetic ocular innervation.

Complete sympathetic paralysis of the head (Horner’s syndrome) causes a miosis, smaller palpebral fissure and a protruded third eyelid. Facial hyperthermia and decreased nasal air flow on the affected side are very difficult to appreciate in small animals. This sympathetic paralysis most commonly involves some component of the pre or postganglionic sympathetic LMN. In very acute severe C1 - C8 spinal cord lesions an UMN Horner’s syndrome may occur. This is most commonly seen in hemiplegic dogs associated with ischemia or infarction caused by fibrocartilaginous emboli. A persistently miotic pupil in a small animal with the signs of an avulsion of the components of the brachial plexus localizes the injury to the level of the roots or spinal nerves at the vertebral column.

It is important to remember that in general the size of the pupils represents a balance between the amount of light entering the eye and stimulating the oculomotor neurons that innervate the iris constrictor muscle and the emotional state of the patient which influences the sympathetic innervation of the iris dilator muscle.

**Strabismus**

While examining the eyes you can appreciate whether they are normally positioned in the orbits. Abnormal eye positions reflect a lack of innervation of the extraocular muscles or a disorder with the vestibular system. The latter is most common and the vestibular strabismus only occurs in some positions of the head. Somatic efferent neurons in the oculomotor nerve prevent a lateral and slightly ventral strabismus. The abducent neurons prevent a medial strabismus. The trochlear neurons prevent an excessive extorsion of the eye which can only be seen in the cat with the lateral positioning of the dorsal aspect of its vertical pupil. In the dog you would have to do a fundic exam and look at the position of the normally vertical superior medulla. For other areas of facial nerve innervation I look for normal flaring of the nostrils on inspiration, hold the head and neck in extension and look at the corners of the lips for evidence of mucosa showing on the paretic side and abnormal drooling on that side. I also assess the ability to move the ears in those patients with erect ears but do not spend much time on...
flop-eared dogs to avoid frustration. Sometimes a normal ear will move when you blow air into it. There is no need for the examiner to get excessively stressed in this process. Remember that dogs and cats with facial paralysis will not have a smaller palpebral fissure (ptosis) unlike the herbivores nor will their nose deviate to the normal side. When you see the nose deviated to one side in a dog this usually is a reflection of excessive contraction of the facial muscles on that side referred to as hemifacial spasm but it is not spasmodic-episodic as it is described in humans. It is a continual deviation. This is most commonly associated with a presumed irritation of the facial nerve from an otitis media. These dogs will have a narrowed palpebral fissure and an ear pulled dorsally and medially on that side. On testing the palpebral reflex there may be slight movement of the eyelids. This has been described as a denervation contracture of the facial muscles but these muscles will often relax during local or general anesthesia which refutes that consideration at least for most cases. This rarely occurs in the cat.

To complete my evaluation of the trigeminal nerve I palpate the muscles of mastication. The only evidence of a unilateral motor trigeminal nerve deficit will be the denervation atrophy that can be palpated. They can still bite!! You need bilateral loss of this mandibular nerve innervation to get a loss of the ability to use the jaw. When this occurs the lower jaw will be dropped so the mouth is continually open and can not be closed. The most common cause of a sudden onset of a dropped lower jaw is an immune-mediated trigeminal neuritis. The most common cause of unilateral atrophy of these muscles is a nerve sheath neoplasm in the dog and more likely lymphoma in the cat. Bilateral atrophy of the muscles of mastication is often seen in older dogs with no evidence of any dysfunction. One cause may be a chronic myositis. Occasionally this atrophy and accompanying fibrosis is severe enough to prevent the jaw from opening.

On a routine examination I always touch the nasal septum with the end of my closed forceps as a test of both the sensory innervation by the trigeminal nerve - specifically ophthalmic branches via the ethmoidal nerve, but also as a very sensitive test for nociception and therefore this projection pathway which involves the contralateral prosencephalon. This is one of the three tests that I have described that will evaluate prosencephalic function. With prosencephalic lesions this nasal septum will never be analgesic just hypalgesic because some of the nociceptive pathway stays ipsilateral and the incomplete crossing occurs in the pons and medulla.

When you determine that there is nasal hypalgesia the lesion responsible for this can either be in the ipsilateral trigeminal nerve or in the contralateral prosencephalon. You differentiate between the two locations based on the rest of the clinical signs that are present. Are they related to the caudal brain stem and therefore this is a trigeminal nerve problem or are they prosencephalic and that is the basis for the hypalgesia?

IX - X - XII
These 3 cranial nerves are examined together with the so-called "gag reflex". This is done rapidly as the patient usually objects to the manipulation that is necessary and especially cats. You grasp the upper jaw with one hand and pull down on the lower jaw with the other hand opening the mouth. This effort will test the tone - resistance in the muscles of mastication (CN V). You quickly look at the size of the tongue for atrophy - hypoglossal nerve (CN XII) and push the tongue with your finger to see if it moves. Then insert your finger deep into the oropharynx to assess the tone and sensory perception that you will stimulate. These latter functions are dependent on the innervation by the pharyngeal branches of the glossopharyngeal (IX) and vagal (X) nerves. This assessment of the gag response is difficult to evaluate and is usually very subjective. A more reliable indication of dysphagia usually comes in the form of a complaint by the owner as they watch their pet try to eat and swallow.

The following is an outline of the order of the cranial nerve exam just described:

- Menace Response II - central visual pathway to occipital lobe
  VII = closure of palpebral fissure
- Pupil Size - Light Response II - brain stem
  III parasympathetic-ciliary ganglion - nerves = pupil constriction direct and indirect
- Eye Position Strabismus - III = ventrolateral
  Strabismus - VI = medial
- Eye Movements Normal vestibulo-ocular nystagmus
  VIII - brain stem - III = addiction
  VIII - brain stem - VI = abduction
- VIII - Vestibular Strabismus in some eye positions
  Abnormal nystagmus (head not moving)
- Facial Muscles VII - position, tone, movement: eyelids, ears, lips, nose
<table>
<thead>
<tr>
<th>Test</th>
<th>Visceral Nerve (Branch)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Menace Response</td>
<td>II - VII</td>
</tr>
<tr>
<td>- Palpebral Reflex</td>
<td>V - VII</td>
</tr>
<tr>
<td>- Facial Sensation</td>
<td>V</td>
</tr>
<tr>
<td>- Palpebral Reflex</td>
<td>V - VII - cutaneous, autonomus zones</td>
</tr>
<tr>
<td>- Nociception</td>
<td>Nasal mucosa - Ophthalmic branch V</td>
</tr>
<tr>
<td>- Masticatory muscles</td>
<td>V - Mandibular branch V</td>
</tr>
<tr>
<td></td>
<td>Muscle size, tone-jaw closure</td>
</tr>
<tr>
<td>- Gag Reflex</td>
<td>Jaw tone - V</td>
</tr>
<tr>
<td>- Tongue Size Movement</td>
<td>XII</td>
</tr>
<tr>
<td>- Reflex Gagging, Swallowing</td>
<td>IX, X</td>
</tr>
</tbody>
</table>

All rights reserved. This document is available on-line at www.ivis.org. Document No. B0201.1001.

Leading the way in providing veterinary information