Unravelling the Mysteries of the Neurological Examination

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The Neurological Examination

History

Before touching the patient, it is important to let them become accustomed to the consulting room environment if it is safe to do so. This can be done whilst the history is being taken from the owner. It allows them to relax and often a more accurate examination is obtained.

I would normally start by asking about the patient's general health; appetite, drinking, urination, vomiting, diarrhoea, weight change, personality change, exercise, travel history, toxin exposure before moving on to the problem they have been presented for.

A thorough history about the problem is invaluable. Ask the owners to describe in their own words what the problem it but make sure that you understand exactly what they mean and clarify any ambiguity. When and how did the problem start? During exercise or at rest? Was there a specific trigger? How has the problem progressed since the onset? Waxing and waning, consistent deterioration or improving? Have they trialled any previous treatment either that they had at home or that was prescribed from a previous veterinary surgeon. Now more and more people have phones which can readily take video footage it is extremely helpful if it is a paroxysmal event if the owner has been able to video it.

Clinical Examination

A thorough routine clinical examination should always be performed as part of the neurological examination as there are a number of non neurological diseases, which can readily be confused with a neurological lesion. For example, bilateral cruciate disease can often appear as if the patient has a spinal disorder especially if they have become non ambulatory. In some circumstances syncope can be confused with a seizure i.e. cats with intermittent third degree atrioventricular block. The patient may have a metabolic reason for the neurological signs i.e. hypothyroidism, diabetes mellitus, Cushing's disease or hepatic encephalopathy. Or there may be a severe concurrent disease which may affect the investigation or that you may need to factor into it i.e. a severe heart murmur or suspicious lumps.

Neurological Examination

The aim the neurological examination is to ideally localise the patient's problem to one part of the neurological system. However, some patients will have multifocal or diffuse problems.

Mentation and Posture

Before touching the patient, there are some important questions to answer. If it is safe to do so, I allow the patient to walk around the consulting room. Is the patient's mentation

appropriate for the surroundings baring in mind their signalment? Are they circling? If so, is it always in one direction and are they wide or tight circles? Are they head pressing? Excessively vocalising? Staring at the walls? Do they constantly bump into objects that they should be able to see?

What is their posture like? Do they have a head tilt? If so in which direction? By convention, the side of the head tilt is the one where the ear is the closest to the ground. Is there a head turn? Body turn (pleurotonus)? Are they palmigrade or plantigrade? Do they have a wide based stance? Is there a spinal curvature?

- Kyphosis – dorsal curvature of the spine
- Lordosis – ventral curvature of the spine
- Scoliosis – lateral deviation of the spine
- Torticollis – twisting of the neck

The curvature can be congenital i.e. secondary to a vertebral malformation. Alternatively, could be seen with a disease process i.e. intervertebral disc disease or syringomyelia. No type of curvature is pathognomonic for a particular disease.

The more severe postures include decerebrate rigidity, decerebellate rigidity and Schiff Sherrington.

Decerebrate rigidity

The patient is in lateral recumbency with dorsal extension of the head and neck (opisthotonus) and extension of all four limbs. The mental status of the patient will also be severely impaired (stupor/coma). A lesion within the rostral midbrain will cause this posture.

Decerebellate rigidity

The patient will be in lateral recumbency with dorsal extension of head and neck (opisthotonus), extension of the thoracic limbs, the hips maybe flexed (increased tone in iliopsoas muscles). The mental status will be normal. The posture can be episodic. A lesion in the rostral cerebellum will cause this posture.

Schiff-Sherrington posture

The patient is in lateral recumbency with increased extensor tone in the thoracic limbs. If there is no possibility of a spinal fracture/luxation then when the patient is adequately supported in a standing position there will be normal voluntary movement and normal conscious proprioception. The pelvic limbs will be paralysed and should typically have UMN signs (i.e. increased tone and spinal reflexes). However, within the first few hours the tone and spinal reflexes may be reduced (so the patient will look as though the lesion is LMN to pelvic limbs) due to the phenomenon of spinal shock. The mental status is normal.

A lesion within the thoracolumbar spinal cord will cause this posture. Border cells (interneurons) are present within the dorsolateral border of the ventral grey matter in spinal segments L1-5. Their axons run cranially in the fasciculus proprius to the cervical intumescence (C6-T2 spinal cord segments). They terminate on LMNs, which supply the thoracic limb extensor motor neurons to inhibit their function. Damage to the border cells

will cause disinhibition of these motor neurons resulting in extension of the thoracic limbs. As the lesion is caudal to the cervical intumescence, the voluntary movement in the thoracic limbs is preserved. The posture has no prognostic significance.

Gait

It is important to assess the patients gait from both a neurological and orthopaedic point of view. Lame animals have a short stride on the affected limb as they try to avoid weight baring and a longer stride on the opposite limb or if severe, enough they will also hold the limb up. Dogs with a nerve root signature due to entrapment of the nerve from a lateralised intervertebral disc extrusion or due to infiltration of the nerve from a neoplastic process can cause the patient to appear lame.

Dogs with bilateral cruciate disease or bilateral hip disease may be non-ambulatory and appear as if the lesion is a T3-L3 or L4-S3 myelopathy when actually there is an orthopaedic cause. If they remain, ambulatory the gait can appear stiff and stilted which can appear neurological.

Patients with LMN disease will have a stiff stilted gait, which can make them appear lame in the affected limb(s).

Paresis is defined as a deficiency in the generation of gait (UMN) or the inability to support weight (LMN). For patients with UMN paresis there is a delay in the protraction phase of the gait and the stride will typically be longer. For patients with LMN paresis the gait is often stiff/stilted and can appear as if the patient is lame. If both pelvic limbs are affected then the limbs maybe used simultaneously resulting in bunny hopping. This is not pathognomonic for a neurological lesion however, and orthopaedic disease still needs to be excluded on the examination

Tetraparesis/tetraplegia – reduced/absent voluntary movement in all limbs
This could be due to a cervical lesion from C1-T2 spinal cord segments
Or from a lesion within the brain from the cerebrum to brainstem
Or from a generalised LMN lesion

Hemiparesis/hemiplegia – reduced/absent voluntary movement in the thoracic and pelvic limb on the same side is affected.
This could be due to an ipsilateral cervicothoracic lesion (C1-T2 spinal cord segments) or within the brain from the caudal aspect of the midbrain to the brainstem
This could be contralateral to a lesion within the brain located from the cerebrum to the rostral midbrain

Paraparesis/paraplegia – reduced /absent voluntary movement in the pelvic limbs
This is due to a lesion from T2-S3 spinal cord segments

Monoparesis/monoplegia – reduced/absent voluntary movement in one leg (either thoracic or pelvic). Typically caused by a LMN lesion to the affected leg. A very lateralised lesion from T3-S3 spinal cord segments may cause paresis/plegia in a pelvic limb. For a lesion from C1-T2 spinal cord segments to cause paresis/plegia in the thoracic

limb then there will also be deficits in the ipsilateral pelvic limb so it would be a hemiparesis/plegia.

The gait should also be assessed for the presence of ataxia (incoordination). There are three types of ataxia.

General proprioceptive (Sensory/spinal) ataxia
The protraction of the affected limb(s) is delayed. The stride may be longer than normal and is often described as a floating gait. During the stride the affected limb(s) may sway medially under the body (adduct) or laterally away from the body (abduct). The paw may drag during the protraction phase and the patient may stand with the paw knuckled over In the normal animal, the CNS needs to know where the neck, body and limbs are within space as well as the amount of muscle contraction in each particular muscle at that time. Interruption of this information due to a lesion within the peripheral nerve, dorsal nerve root, spinal cord or brainstem can result in this type of ataxia. Lesions affecting the UMN pathways will also affect the general proprioception so a UMN tetraparetic or paraparetic patient will also have general proprioceptive ataxia in the affected limbs

Vestibular ataxia
There is a loss of coordination between the eyes, neck, body and limbs resulting in the patient leaning/tracking or falling to one side. The strength is maintained within the limbs. A head tilt and pathological nystagmus may also be seen. Patients with bilateral peripheral vestibular dysfunction will typically have side to side head movements, loss of physiological nystagmus, no apparent head tilt and will tend to remain crouched rather than walk. It is caused by lesions within the vestibular system.

Cerebellar ataxia
There is marked over flexion of the limbs on protraction leading to a high stepping and overreaching stride. The patient is unable to control the rate or range of the movement The gait is often referred to as hypermetric or dysmetric. Most commonly this type of ataxia would occur due to a cerebellar lesion. Less commonly a lesion involving the spinocerebellar tracts could also cause this type of ataxia

Postural Reactions
These are further tests to assess whether the patient knows where their limbs are as well as how strong and coordinated they are.

Paw positioning: Pick up each paw in turn and turn the paw over so that the dorsal surface is on the ground. The normal reaction is for the patient to immediately turn the foot over and place it correctly underneath them. The test can be repeated until you are confident that the result is repeatable. Failure to support the body weight will cause the limb to flex which will stimulate joint receptors which are not part of the test and so a false test maybe achieved. It is also important to support their weight whilst performing the if they have concurrent orthopaedic disease otherwise it may be too painful for them to turn their foot over and you may have a false result. The patient must still put some weight through the limb so that they feel the paw is turned over. Failure to do so will

mean some patients will leave the paw turned over even when their proprioception is normal.

Hopping: Lift the left thoracic limb so that the patient has the majority of their weight on the right thoracic limb. Then move the patient towards the right. As the patients centre of gravity is moved to the right the normal response is for the patient to hop to correct the body position. Those with a mild hopping deficit will leave the limb underneath them for a longer time before correcting the position. Those patients with a marked hopping deficit may not move the limb at all and may collapse. If you are concerned that the hopping may cause them to collapse then stand astride the patient so that you are in a better position to support them. I usually perform the hopping on both thoracic limbs so that I can compare left and right before moving on to the pelvic limbs. The hop should be smooth and coordinated and is likely to be slightly slower in the pelvic limbs compared to the thoracic limbs. The test can be repeated until you are confident that the result is repeatable.

Hemi standing and walking: On a non slip surface pick up both the thoracic and pelvic limb on the same side and gently push the patient away from you. The patient should hop so that the thoracic and pelvic limb remains underneath them. The patients response should be assessed for how quickly and coordinated they are able to do the test.

Wheel barrowing: This is a postural reaction for the thoracic limbs, which can be performed in most patients except those, which are very large. The pelvic limbs are lifted off the ground and the patient is encouraged to walk forwards. The thoracic limbs should be assessed for any signs of weakness or ataxia. If the head and neck are extended, the vestibular system is challenged and this test can show subtle abnormalities in the vestibular system

Extensor postural thrust: This can only be performed in cats and small dogs. Hold the patient around the chest and lower the patient to the table so that the pelvic limbs contact the table and then walk the patient backwards. The patient should be able to support their own weight when they touch the table and walk backwards in a coordinated fashion.

Visual placing and tactile placing: These are really only suitable for cats and small dogs.

Tactile Placing: This can only be performed in small dogs and cats, which can be readily lifted. Cover the patient's eyes with one hand. Lift the patient up and approach the table. As the patients foot touches, the table they should pick the foot up and place it correctly upon the table. Repeat the test for all limbs.

Visual Placing: The patient is lifted up brought towards the table this time with their eyes uncovered. The patient should be able to lift the feet up in anticipation of reaching the table and to place the feet correctly upon it. With dogs that are used to being carried around, they can falsely have a delayed response.

Reflex step/ Sliding paper test: Tests the proprioception in the proximal limb. The patient's foot is placed upon the paper and the paper is pulled laterally. The patient should pick up the foot and place it correctly underneath them.

In the majority of cases only the paw positioning and hopping are performed. If there is a doubt about a result then the other postural tests can be performed.

The grading system most commonly used is 0 = absent, 1 = reduced and 2 = normal.

Cranial Nerve Examination

Eyes

Vision (CNII): Does the patient bump into objects in the consulting room? Does the history lead you to a suspicion that the patient maybe blind? You can set up obstacle courses for the patient to negotiate. Then blind fold each eye in turn and take them through the obstacle course again. Alternatively, cover each eye in turn and drop a cotton wool ball in front of the open eye. Does the patient watch it? Be careful not to use anything that might make a sound otherwise you will be testing their hearing rather than just their vision.

Palpebral reflex (CN V and VII): Touch the medial and lateral canthi of each eye in turn. The medial canthus is innervated by the ophthalmic branch of the trigeminal nerve where as the lateral canthus is innervated by the maxillary branch of the trigeminal nerve. The patient should blink and should fully close the eyelids. Repeat 2-3 times until you are confident of the result. Those patients with a subtle facial paresis may not be able to fully close the eyelids. This reflex may fatigue in patients with Myasthenia gravis.

Menace response (CN II and VII): Make a threatening gesture with your hand towards the eye, the patient should blink and should fully close the eyelids. By doing the palpebral reflex response first, they are more likely to menace as they realise that you will touch them! Be careful not to waft air onto the cornea or touch any long hairs, as this will assess the trigeminal nerve. This is a learned response and so will be absent in animals less than 10-12 weeks old. The input is via optic nerve and output via facial nerve but pathway runs through the visual cortex, motor cortex, pons and cerebellum.

Eye position (CN III, IV, VI, VIII): When you face the patient head on are their eyes in a normal position? Do they deviate laterally (divergent strabismus), medially (convergent strabismus)? Is there any spontaneous nystagmus? If so is the character horizontal, rotatory or vertical? By convention, the direction of the nystagmus is defined as the direction of the fast phase. The presence of any nystagmus when the patient's head is still is abnormal. Elevate the head and assess the eyes for any positional strabismus or nystagmus. Are either now present? Has the character/direction changed?

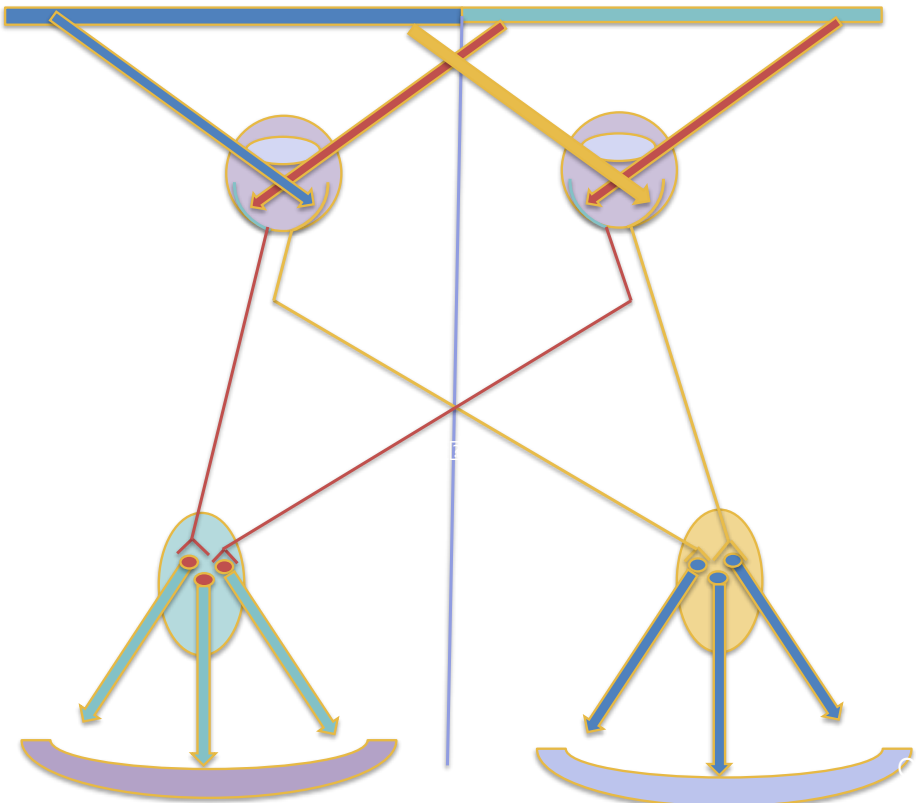
Physiological nystagmus/vestibular ocular reflex: I tend to assess canine patients by standing astride them and looking down on the head. I gentle pull back the upper eyelids so that I can see the sclera. Move the head from left to right and assess eyeball movement The eyes will jerk to keep a stable image on the retina. The fast phase of the jerk will be in the direction of the movement of the head. The slow phase of the jerk will be opposite to the movement of the head. The movement should stop as soon as the patients head stops moving. In cats, I usually hold them around the thorax at arms length with them facing me and move them from side to side.

Anisocoria: Assess the patients pupil size in both bright and dim light considerations as this can make anisocoria due to certain causes more evident i.e. in Horner's syndrome the miosis will be most obvious in a darkened room. Holding the pen torch about 1m away from the patient and shining the light into both eyes simultaneously can also identify anisocoria. There are a small proportion of patients who may have a resting anisocoria without a pathological underlying cause.

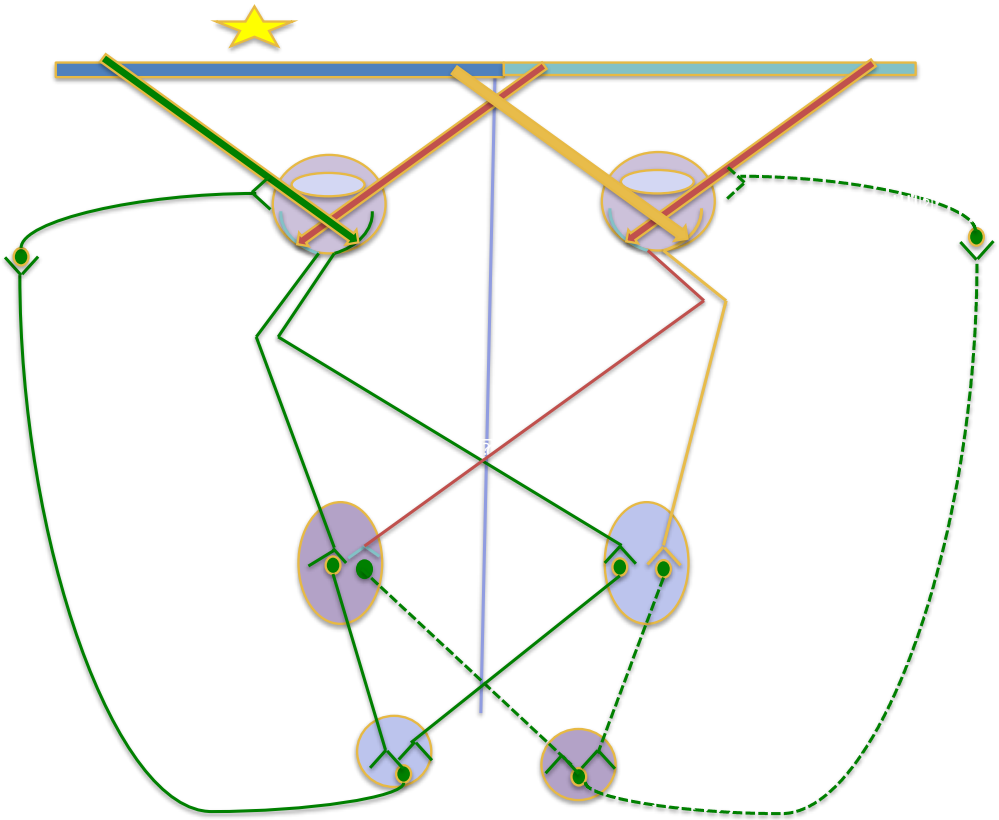
Pupillary light reflex and swinging flash light (CN II and III): It is important to use a very bright pen torch as you will need to overcome the high resting sympathetic tone of the patient which will cause pupil dilation. When the light is shone into the eye the pupil

should constrict (direct response). Then move the light to the opposite eye. The pupil should already be partly constricted (consensual response) and then should constrict fully (direct response). Then move the light back to the left eye, which should have partly constricted due to the light being shone in the right eye (consensual response). The pupillary light reflex is very useful in blind animals as it can help localise the lesion. The rostral part of the pathway for the pupillary light reflex and vision are the same. They deviate after the optic chiasm as the axons involved in vision will synapse in the lateral geniculate nucleus where as those for the pupillary light reflex will be synapse in the pretectal nucleus. Therefore, if the patient is blind with an absent pupillary light reflex the lesion will be between the retina and the lateral geniculate nucleus/pretectal nucleus.

Schematic diagram of the pathway for vision.



Schematic diagram for the pathway for the pupillary light reflex



The bold green line indicates the pathway of the direct pupillary light reflex
The dashed green line indicates the pathway of the consensual pupillary light reflex

Corneal reflex (CN V, VI, VII): This reflex is not commonly tested unless there is a concern about the general facial sensation. Using a damp cotton bud gently touch the cornea, the patient should blink and retract the globe. I tend to prefer testing the nasal sensation with my artery forceps as this also tests the ophthalmic branch of the trigeminal nerve.

Horner's Syndrome (Sympathetic supply to the eye): There are 4 parts to this syndrome, not all of them have to be present as in some cases the patient may only have a partial Horner's. The components are enophthalmus; third eyelid protrusion; ptosis (drooping of the upper eyelid) and miosis. In partial Horner's, there may only be miosis. The anisocoria will be more noticeable in dim light. The pathway is very long. It originates within the hypothalamus/rostral midbrain, extends down the cervical spine in the tectotegmental spinal tract to the T1-3 spinal cord segments. It exits here and course back up the neck in the vagosympathetic trunk to the level of the tympanic bulla. From there, the axons pass through the middle ear before entering the cranial cavity with CN IX. There is a short intracranial course before the axons exit the skull via the orbital fissure with the ophthalmic branch of CN V. The axons supply the smooth muscle of the iris, orbit and eyelids (including the third eyelid). Damage anywhere along this pathway can cause Horner's syndrome and so you need to use the rest of the neurological examination to help you localise the lesion further.

Face

Facial sensation (CN V): Touch all over the patients face with closed artery forceps. Does the patient react? Cover their eyes and test up each nostril. Is the response the same both sides?

Facial symmetry (CN VII): When you look at the patient head on is the face symmetrical? Is one side droopy? Do the nostrils move symmetrically? Is the ear position symmetrical? When you lift the patient's head up are the commissures of the mouth level? When the lips are touched/ gently pinched are they retracted by the patient?

Temporal muscle mass (CN V): Palpate the temporal muscles. Are they symmetrical? Are the atrophied? Are they swollen or painful? Many older animals or those on steroids will have symmetrical atrophy of their temporal muscles.

Jaw tone (CN V): Is the patient able to hold the jaw closed? Open the patient's mouth and assess the resistance. Is the jaw tone normal, reduced or increased?

Gag reflex (CN IX and X): There are two ways to do this depending on whether you trust the patient not to bite. If you trust the patient then open their mouth and place your fingers at the back of their throat, they should swallow, gag or retch. If you don't trust the patient then gentle, squeeze the hyoid apparatus and watch to see if they swallow.

Tongue (CN XII): When their mouth is open assess the tongue, is it deviated? Is it symmetrical and feel it to assess the tone, are there any abnormal movements?

Hearing (CN VIII): Does the patient respond appropriately to noises in the environment? Clap to one side of the patient; does the patient turn towards the noise? Repeat for the other side.

Muscle tone, atrophy, spinal reflexes and withdrawals

Try to have the patient standing as square as possible and feel down their shoulders and hips then down each limb individually. Feel for any asymmetry in the muscle size and tone. Then with the patient in lateral recumbency move the upper most limbs one at a time to assess their range of motion and tone.

Spinal reflexes and withdrawals

Biceps reflex: Pull the thoracic limb caudally to extend the elbow. Place your finger on the biceps tendon where it inserts on the radius. Tap your finger with the tendon hammer. The normal response is to see a contraction of the biceps brachii muscle. There will also be a flexion of the elbow if your grip on the leg is reduced. This reflex can be difficult to obtain in the normal patient or may be difficult to see if the patient has very long/thick fur The reflex evaluates musculocutaneous nerve and C6-C8 spinal cord segments

Triceps reflex: Flex the elbow and rotate the shoulder medially. The triceps tendon is struck just proximal to the olecranon. The normal response is to see a contraction of the triceps muscles and extension of the elbow or carpus. This reflex can be difficult to obtain in normal animals. The reflex evaluates the radial nerve and C7-T1/2 spinal cord segments.

Extensor carpi reflex: The limb is supported under the ante brachium. The carpus is slightly flexed. The tip of the tendon hammer is used to strike the proximal aspect of the extensor carpi radialis muscle. The normal response is an extension of the carpus. The reflex evaluates the radial nerve and C7-T2 spinal cord segments.

Withdrawal reflex in the thoracic limbs: The limb is extended and the skin between/at the base of the digits is lightly pinched, the stimulus is increased if there is no response. The normal response is flexion of the joints of the thoracic limb. It is important to consider the cutaneous innervation as this can affect the presence of the reflex. The skin over the dorsal aspect of the first digit is innervated by the radial nerve (palmer surface by the ulnar and median nerves). The skin over the dorsal aspect of fifth digit is innervated by the ulnar nerve (palmer surface by the ulnar and median nerves). The sensation is transmitted via the median, ulnar and radial nerves to the C6-T2 spinal cord segments (within the C5-T1 vertebrae). The motor output from the C6-T2 spinal cord segments is transmitted through the axillary, musculocutaneous, and median and ulnar nerves. The axillary, radial and thoracodorsal nerves are responsible for flexion of the shoulder. The musculocutaneous nerve is responsible for flexion of the elbow. The median and ulnar nerves are responsible for flexion of the carpus and digit. It is important to observe the contralateral limb for reflex extension during the testing of the withdrawal reflex. No extension in the contralateral limb should be seen in the normal patient. Patients with a lesion cranial to C6 will show a reflex extension of the contralateral limb – crossed extensor reflex

The presence of the withdrawal reflex does not indicate that there is a conscious perception of the stimulus

Patella reflex: The patella reflex maybe absent either unilaterally or bilaterally in patients over 10 years of age due to non specific age related changes. Previous stifle disease may also cause a reduced or absent patella reflex. It may also be difficult to elicit a patella reflex in very tense animals. If you can not get them to reflex then you can test the patella reflex in the recumbent limb, as they will not be able to tense this leg to such and extent. In order to perform the patella reflex a hand is placed under the thigh to support the pelvic limb. The stifle should be partially flexed. The patella tendon is stuck with the tendon hammer. There should be a single, quick extension of the stifle.

The reflex maybe exaggerated (pseudohyperreflexia) if there is a lesion within the sciatic nerve or L6-S1 spinal cord segments. The muscles responsible for stifle flexion would normally dampen the patella reflex. If there is a lesion in the sciatic nerves or L6-S1 spinal cord segments then these muscles have a reduced tone and so are unable to dampen the reflex as effectively

To differentiate a pseudohyperreflexia from an increased patella reflex the tone, other reflexes and gait should be assessed to form a complete picture. Patients who are very anxious may also have an increased patella reflex. In these patients, the gait and postural reactions will obviously be normal. The increased reflex does not mean there is an UMN lesion. This reflex evaluates the femoral nerve and the L4-6 spinal cord segments.

Gastrocnemius reflex: The stifle should be in extension and the hock flexed. The common calcaneal tendon is struck just proximal to the calcaneus. There should be a contraction of the caudal thigh muscles. It may not be possible to elicit the reflex even in normal animals. The reflex evaluates the tibial branch of the sciatic nerves and the L7-S1 spinal cord segments.

Cranial tibial reflex: The limb should be supported under the thigh. The tarsus should be in slight extension. The tip of the tendon hammer is used to strike the proximal aspect of

the cranial tibial muscle. The response should be flexion of the tarsus. The reflex evaluates the peroneal branch of the sciatic nerve and L6-S1 spinal cord segments.
Withdrawal reflex in the pelvic limbs: The limb is extended and the skin between or over the base of the digits is lightly pinched, the stimulus is increased if there is no response The normal response is flexion of the joints of the pelvic limb. It is important to consider the cutaneous innervation as this can affect the presence of the reflex. The saphenous branch of the femoral nerve innervates the skin over the first digit. The skin over the fifth digit is innervated by the sciatic nerve. The sensation is transmitted via the peroneal and tibial branches of the sciatic nerve (lateral, dorsal and ventral aspect of foot) as well as the saphenous branch of the femoral nerve (medial aspect of limb and first digit) to the L4-S1 spinal cord segments (within the L4-5 vertebrae). The S2 spinal cord segment innervates the pelvic muscles and so is not assessed by testing this reflex. The motor output from the L4-S1 spinal cord segments is transmitted through the femoral and sciatic (peroneal and tibial branches) nerves. The femoral nerve is responsible for flexion of the The sciatic nerve is responsible for the flexion of the stifle and hock. The presence of the withdrawal reflex does not indicate that there is a conscious perception of the stimulus It is important to observe the contralateral limb for reflex extension during the testing of the withdrawal reflex. No extension in the contralateral limb should be seen in the normal patient. Patients with a lesion cranial to L4 will show a reflex extension of the contralateral limb – crossed extensor reflex.

The grading system commonly used for spinal reflexes is 0 = absent, 1 = reduced, 2 = normal, 3 = increased, 4 = clonic.

Nociception (Pain perception)
The withdrawal reflex is purely a reflex and relies on the local spinal cord segments involved in the reflex to be intact. It does not rely on conscious input from the brain. In patients with completely severed spinal cords in the thoracolumbar region, the withdrawal in the pelvic limbs will be good.
Due to the order of loss of function in with spinal cord lesions, it is not necessary to test deep pain perception in patients with good voluntary movement. However, those who are paraplegic or tetraplegic will need to have their pain perception assessed in all limbs and tail.
In order to test deep pain perception firstly pinch the toes with your finger to achieve a withdrawal then gradually increase the pressure and monitor for a response. In some patients the response will be unequivocal i.e. vocalisation or trying to bite. In some, it can be more subtle i.e. stopping panting, dilation of the pupils. Also, be careful that the patient is not responding to some other stimulus i.e. a noise in the neighbouring room. In addition, during the withdrawal some patients will change their body position, which they then can react to rather than the stimulus you are causing. You want to achieve an unequivocally painful stimulus so if there is no response with digital pressure you need to place an artery forceps across the nail bed and gradually increase the pressure. Always be mindful that you do not want to press so hard that you crush the tissues. The deep pain perception is carried by spinal cord tracts located in all funiculi (dorsal, lateral and ventral) that run both ipsi and contralaterally. The most significant tract is the

spinothalamic tract. Therefore, for a lesion to cause a loss of deep pain perception bilaterally it would have to be a severe transverse spinal cord lesion

Order of loss of function with progressive spinal cord disease

- Loss of conscious proprioception
- Loss of voluntary movement
- Loss of bladder function
- Loss of deep pain perception

Bladder

Palpate the abdomen, assess the bladder size, tone, and then gently apply some pressure to see how easy it is to express.

Perineal (anal) reflex

The anus should be tightly closed in the normal patient. Touch the left and right sides of the perineum with the tip of a closed artery forceps. The normal response is for the anal sphincter to contract and for the tail to flex (clamp down). The reflex evaluates the pudendal nerve (perineal and caudal rectal branches) and the S1-3 spinal cord segments (perineum) as well as the caudal segments (Cd1-5) (tail flexion).
In some patients with severe UMN lesions within the thoracolumbar spinal cord stimulation of the perineal reflex may also cause flexion of both pelvic limbs and tail. The patients may also urinate. This is referred to as a mass reflex.

Panniculus reflex/Cutaneous trunci

The patient should be standing square. Gently pinch the skin (using fingers or artery forceps) just lateral to the spine starting at the wings of the ileum. In normal patients, the reflex is usually present from the mid lumbar area although there is a degree of individual variation. If the reflex can be obtained at the lumbosacral region bilaterally there is no need to proceed cranially. If the reflex cannot be obtained then the test should be repeated moving cranially by one vertebra at a time on one side. Once the reflex has been obtained the level should be recorded before the opposite side is similarly tested. The normal reaction is for a bilateral twitch of the skin over the thorax and abdomen due to contraction of the cutaneous trunci muscle. The skin over the thoracolumbar region is innervated by spinal nerves. The dorsal branches of the spinal nerve only extend for approximately two vertebrae caudal to the intervertebral foramen from which the spinal nerve arose. Therefore, if there is a cut off the lesion will be approximately two vertebrae cranial to the level of the cut off. Depending upon the level within the vertebral column this may correspond to 1-4 spinal cord segments. If the reflex were completely absent on one side, this would suggest a lesion in the ipsilateral brachial plexus. In normal animals, there is no cutaneous trunci reflex in the cervical or sacral regions. If the reflex is absent but there are no other neurological deficits then the loss of the reflex is unlikely to be significant as this can occur in normal patients. The reflex evaluates the lateral thoracic nerve and the C8-T1 spinal cord segments.

Palpation

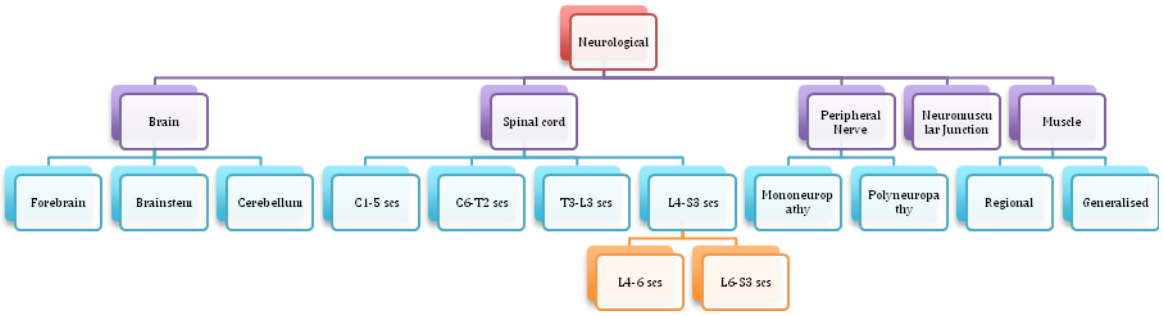
Gently palpate along entire length of the spine. Then turn the neck from side to side; the patient should be able to touch his/her flank with their nose. Then lift the head up and

then down. The patient should be able to put their nose in between their thoracic limbs. Note down any resistance or areas of pain. Be very cautious with ventroflexion of the neck especially in toy breeds where Atlantoaxial subluxation is a possibility. Other breeds can be affected by Atlantoaxial subluxation including Cavalier King Charles Spaniels. A gentle squeeze just in front of the wings of C1 can often detect subtle meningeal pain.

Also, palpate the skull assessing for the presence of an open fontanel, asymmetry or pain. Squeezing just rostral to the zygomatic arches can detect pain in some patients with intracranial disease.

Localisation

Once you have performed the neurological examination and recorded your findings you now need to determine where in the nervous system it is likely to be. This is important in helping to work out an accurate differential diagnosis list and making sure that if further investigation is required, the right part of the patient is investigated. It is important to also use information gleaned from the history and physical examination.



Brain

Forebrain

Neuro exam	***Dysfunction***
Mentation	*Behavioural change*
	Altered mental status (i.e. obtundation, d
Cranial nerve	*Blindness (contralateral)*
	Decreased/absent menace response (cont PLR
	Possible anisocoria (contralateral)
	Possible facial paresis (contralateral)
	Possible facial hypoalgesia (contralatera
Gait/posture	*Normal/near normal gait*
	Pleurotonus (body turn) towards the lesio
	Head turn towards the lesion
	Circling in wide circles (usually towards
	Pacing
	Aimless wandering
	Hemi-inattention/hemineglect syndrome
	Narcolepsy/cataplexy (rare)

Forebrain continued

Neuro Exam	**Dysfunction**
Postural reactions	Contralateral deficits
Spinal reflexes	Normal to increased in contralateral limbs

Muscle tone	Normal to increased in contralateral limbs
Sensation	Hypoaesthesia to contralateral side of the bod
Possible other things reported in the history	Seizures
	Hemi-inattention/hemineglect syndrome
	Narcolepsy/cataplexy (rare)

Brainstem

Neuro exam	**Dysfunction**
Mentation	Altered mental status (i.e. obtunded, stup
Cranial nerve	Deficits in the function of CN III to XII
Gait/posture	Tetraparesis/tetraplegia
	Or hemiparesis/hemiplegia (ipsilateral)
	Opisthotonus (possible)
	Decerebrate rigidity (possible)

Brainstem continued

Neuro Exam	**Dysfunction**
Postural reactions	In all four limbs
	Or ipsilateral thoracic and pelvic limbs
Spinal reflexes	Normal to increased in all four limbs
	Normal to increased in ipsilateral thoraci
Muscle tone	Normal to increased in all four limbs
	Normal to increased in ipsilateral thoraci

Sensation	Possible cervical hyperaesthesia
Possible other abnormalities	Respiratory dysfunction
	Cardiac abnormalities

Cerebellum

Neuro exam	**Dysfunction**
Mentation	Normal
Cranial nerves	Ipsilateral menace response deficit
	Vestibular dysfunction (possible)
	Anisocoria, widened palpebral fissure and eyelid (possible)
Gait/posture	Intention tremors (head and eye)
	Hypermetria (normal strength)
	Ataxia
	Broad based stance
	Decerebellate rigidity (possible)

Cerebellum continued

Neuro Exam	**Dysfunction**
Postural reactions	Initiation is delayed and response is exag
Spinal reflexes	Normal
Muscle tone	Normal to increased
Sensation	Normal
Possible other abnormalities	Increased frequency of urination (possibl

	UMN	LMN
Posture	Can be normal unless lesion severe ıuse recumbency. The affected limb (s) may be held adducted, abducted, crossed or knuckled over.	Difficult to support weight Crouched stance

Spinal cord
Upper Motor Neuron vs. Lower Motor Neuron

Gait	Delayed protraction phase General proprioceptive ataxia Stiffness Paresis/plegia	Short strides No ataxia Often collapse Paresis/plegia
Muscle tone	Normal/increased tone (Hypertonia)	Reduced/absent tone (Flaccid)
Spinal reflexes	Normal/increased (Hyperreflexia)	Reduced/absent
Range of motion	Increase resistance when moving limb	Decrease resistance when moving li
Muscle atrophy	Late onset Mild Disuse atrophy	Early onset Severe Neurogenic atrophy

Spinal Cord Segments	Thoracic Limbs	Pelvic Limbs
C1-5	UMN	UMN
C6-T2	LMN	UMN
T3-L3	Normal	UMN
L4-S3	Normal	LMN

C1-5 spinal cord segments

Neuro Exam	**Dysfunction**
Mentation	Normal
Cranial nerve examination	Horner's syndrome (ipsilateral) possible
Gait	Tetraparesis/tetraplegia or ipsilateral hem
Posture	Torticollis/scoliosis if asymmetrical weak muscles
Postural reactions	Reduced in all four limbs or ipsilateral th
Spinal reflexes	Normal/increased in all four limbs
Muscle tone	Normal/increased in all four limbs
Muscle mass	Normal
Spinal pain	Possible in cervical region
Respiration	Poor respiration in tetraplegic patients
Urination	Urine retention possible
Defecation	Faecal incontinence possible

C6-T2 spinal cord segments

Neuro Exam	**Dysfunction**
Mentation	Normal

Cranial nerve examination	Horner's syndrome (ipsilateral) possible
Gait	Tetraparesis/tetraplegia or ipsilateral hem monoparesis of one thoracic limb
Posture	Torticollis/scoliosis if asymmetrical weak muscles
Postural reactions	Reduced in all four limbs or ipsilateral th or affected thoracic limb
Spinal reflexes	Reduced/absent in thoracic limbs or limb Normal/increased in pelvic limbs Reduced/absent cutaneous trunci if lesion segments
Muscle tone	Reduced to absent in thoracic limbs or lir Normal/increased in pelvic limbs
Muscle mass	Muscle atrophy in thoracic limbs Normal mass in the pelvic limbs
Spinal pain	Possible in caudal cervical/cranial thoraci
Respiration	Poor respiration in tetraplegic patients
Urination	Urine retention possible

T3-L3 Spinal Cord Segments

Neuro Exam	**Dysfunction**
Mentation	Normal
Cranial nerve examination	Normal
Gait	Paraparesis/paraplegia
Posture	Schiff-Sherrington possible if severe and
Postural reactions	Normal in thoracic limbs Reduced in pelvic limbs
Spinal reflexes	Normal in thoracic limbs Normal/increased in pelvic limbs Cutaneous trunci cut off caudal to the lesi
Muscle tone	Normal in the thoracic Normal/increased in pelvic limbs

Muscle mass	Normal in all four limbs
Spinal pain	Possible in thoracolumbar region
Sensation	Superficial or deep pain perception mayb
Urination	Urinary retention possible (UMN bladder
Defecation	Faecal incontinence possible

L4-6 spinal cord segments

Neuro Exam	**Dysfunction**
Mentation	Normal
Cranial nerve examination	Normal
Gait	Paraparesis/paraplegia or Monoparesis/pl
Postural reactions	Normal in thoracic limbs Reduced in on or both pelvic limbs
Spinal reflexes	Normal in thoracic limbs Patella reflex reduced/absent in one or bo Normal pelvic limb withdrawals
Muscle tone	Normal in the thoracic Reduced/absent extensor tone in pelvic li
Muscle mass	Normal in thoracic limbs Atrophy affecting the quadriceps femoris
Spinal pain	Possible in lumbar region
Sensation	Sensory loss affecting specific dermatom specific nerves possible
Urination	Urinary retention possible (UMN bladder
Defecation	Faecal incontinence possible

L6-S3 spinal cord segments

Neuro Exam	**Dysfunction**

Mentation	Normal
Cranial nerve examination	Normal
Gait	Paraparesis/paraplegia or Monoparesis/pl Paresis/paralysis of the tail Able to walk but difficulty in rising if par
Postural reactions	Normal in thoracic limbs Reduced in pelvic limbs or limb
Spinal reflexes	Normal in thoracic limbs Pseudohyperreflexia of patella reflex in o Reduced/absent withdrawal in pelvic limb Reduced/absent perineal reflex
Muscle tone	Normal in the thoracic Reduced/absent in pelvic limbs and tail Dilated anal sphincter
Muscle mass	Atrophy affecting caudal thigh muscles, r distal pelvic limb muscles
Spinal pain	Possible in lumbosacral region or on palp during rectal examination
Sensation	Superficial or deep pain perception mayb perineal region and tail
Urination	Urinary retention possible (LMN bladder

Spinal cord segment summary slide

Neuro Exam	**C1-5 scs**	**C6-T2 scs**	**T3-L3 scs**	**L4-6 scs**
Postural reactions – thoracic	Reduced	Reduced	Normal	Normal
Postural reactions – pelvic	Reduced	Reduced	Reduced	Reduced
Spinal reflexes - thoracic	Normal/increased	Reduced/absent	Normal	Normal
Spinal reflexes - pelvic	Normal/increased	Normal/increased	Normal/increased	Patella absent, withdrawal ok
Muscle tone - thoracic	Normal/increased	Reduced/absent	Normal	Normal

Muscle tone - pelvic	Normal/increased	Normal/increased	Normal/increased	Reduced/absent

Peripheral Nerve

- 12 pairs of cranial nerves
- 36 pairs of spinal nerves
- Contain sensory and motor axons
- Skin region innervated by one spinal nerve-dermatome
- Muscles innervated by one spinal nerve-myotome
- Brachial plexus formed by branches of the C6-T2 spinal nerves to supply the thoracic limb
- Lumbosacral plexus formed by the branches of L4-S3 spinal nerves to supply the pelvic limb

Neuro Exam	**Dysfunction**
Mentation	Normal
Cranial nerve examination	CN VII, IX and X maybe affected
Gait	Paresis/paralysis of affected limb or limb
Postural reactions	Reduced in affected limb or limbs (senso
Spinal reflexes	Reduced/absent in affected limb or limbs
Muscle tone	Reduced/absent in affected limb or limbs
Muscle mass	Atrophy of affected limb or limbs (motor
Sensation	Paraesthesia (possible self mutilation)
Nociception	Reduced/absent in affected limb or limbs

Neuromuscular Junction

There are three main parts which can be affected within the neuromuscular junction.

- Presynaptic – decreased ACh released
    - i.e. Botulism
    - LMN all 4 limbs, dysphagia, dysphonia and facial weakness possible
- Postsynaptic – interference with ACh receptor activation
    - i.e. Myasthenia gravis
    - Exercise induced weakness

- Enzymatic – chemicals interfere with AChE
    - i.e. organophosphate and carbamate toxicity
    - Autonomic overstimulation
    - Stiff gait, muscle tremors and exercise intolerance

Muscle

Neuro Exam	Dysfunction
Mentation	Normal
Cranial nerve examination	Abnormal if the facial, masticatory or affected
Gait	Stiff and stilted Exercise induced weakness/stiffness
Postural reactions	Dependent on severity (normal to red
Spinal reflexes	Normal (unless contractures)
Muscle tone	Normal/reduced/increased dependent
Muscle mass	Atrophy/hypertrophy dependent on di Contractures possible
Sensation	Myalgia possible

Differential Diagnosis List

I usually use the mnemonic DAMNITV to ensure I remember the different disease processes, which I should be considering. I try to order them in order of likelihood. Using the differential diagnosis list, I then compile a list of diagnostic tests, which will help me, rule in or out the differentials within the list.

- Degenerative
- Anomalous
- Metabolic
- Neoplastic, Nutritional
- Inflammatory, Infectious, Idiopathic
- Traumatic, Toxic
- Vascular