Neck Pain
In Smaller and Medium Breed Dogs

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Neck pain is a common presenting clinical problem encountered by the general practitioner. Localizing the lesion is actually quite logical, although diagnostic tests identifying the cause can be more extensive and specialised.

Introduction
Neck pain is a common presenting clinical problem encountered in practice and is often referred to a specialist for further investigation and treatment.

When evaluating these patients allow them first to move freely in the examination room. A painful animal guards its movements and will look around with exaggerated eye movements or by turning the whole body in an effort not to bend the neck. The clinical presentation is very typical with the head held low, either tucked back into the shoulders or extended, a slow deliberate gait, reluctance to walk up or down stairs, reluctance to jump, intermittent sharp “crying out” in pain, avoidance of head touching (head shy) and aggression towards other dogs.

A clinical, orthopaedic and neurological examination needs to be performed. Do the painful part last. Put pressure on the para-spinal musculature starting at the thoracic area, using the thumb and first finger, exerting first gentle then deep pressure depending on response. Work backwards to the lumbar region, supporting the patient under the abdomen. In this way you will also pick up abdominal splinting. When evaluating the neck, if the pain and splinting is severe you cannot perform range of motion. It not slowly and gently flex the entire neck, not just the head, left, right, up and down, stopping as soon as the patient shows signs of pain or resistance. It is important to recheck these responses for repeatability.

The breed and age of the dog plays an important role in drawing up the initial working differential diagnosis list and expedites the diagnostic procedures required to make a diagnosis. This article will look at the problems causing neck pain in the smaller and medium breeds, the diagnostic differentials, the diagnostic procedures and treatment considerations.

Differential diagnosis
The differential diagnosis list for neck pain in the small and medium breed dog is extensive. Just thinking of the anatomical structures can help you make a list.

Muscles - abscesses, muscle injury
Vertebrae - fractures, neoplasia
- Very painful, neurological deficits not always present
Joints (synovial) – polyarthritis (immune mediated)
- Very painful. No neurological deficits. Other small synovial joints also generally painful or swollen eg digits and carpus
Discs – IVDD or discospondylitis
- Both conditions are painful. No neurological deficits occur with discospondylitis
Nerve roots
- Nerve root compression and inflammation is very painful.
- Root signature/sign can occur in both fore and hind limbs.
- Splinting of the abdominal muscles in spinal disease may mimic the clinical appearance of an acute abdomen.
Meninges - Inflammation (infectious or more commonly, immune mediated)
- Painful with or without neurological deficits
Spinal column - neoplasia, infarcts, masses
- no sensory fibres in CNS parenchyma – so no pain with lesions.
- Neurological deficits present.

The most common causes of neck pain include:
- Disc extrusion or prolapse
- Immune mediated inflammatory meningitis/myelitis
- Seringomyelia
- Neoplasia
- Discospondylitis
- Trauma (vertebral fractures or spinal haematoma)
- Disternper myelitis
- Atlanto-axial sub luxation
A more extensive list including less common causes of neck pain includes the following:

- Vertebral Facet joint pain
- Synovial cyst
- Bicipital bursitis
- Intracranial mass lesion (neoplasia)
- Midthoracic lesions (discospondylitis/tumour)
- Polyanarthritus
- Polymyositis

Cervical spondylomylopathy ("Wobblers syndrome") has not been mentioned in the differential diagnosis list as it is a condition of large breed dogs.

Clinical Signs

The obvious clinical signs associated with neck pain have been listed in the introduction. Nerve root sign is sometimes noted with neck pain and this tends to localise the lesion to the C4-T1 area of the spinal cord. This presents with front limb lameness, with the limb often being held up, that is most often unilateral and manipulation of the lower neck will elicit withdrawal of the front limb together with a cry of pain. Traction of the limb may also elicit a pain response.

Neurological examination:

When assessing neck pain a full neurological examination is also required to help localise the lesion in the neck and exclude brain involvement which may be seen with inflammatory immune mediated conditions. Spinal reflexes should be checked in the front and back legs. In many of cases of neck pain there are no neurological deficits noted. If neurological deficits are noted then the urgency for a diagnosis is increased.

If the lesion is cranial to the nerves supplying the limb being tested the reflexes will be exaggerated and are described as upper motor neuron lesions (UMN). If the lesion is at the level of the neurons supplying the limb being tested the reflexes are generally decreased or absent and are described as lower motor neuron lesions (LMN). Figure 1

<table>
<thead>
<tr>
<th>Site of Injury</th>
<th>Thoracic Limb Deficit</th>
<th>Pelvic Limb Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>a: C1-C5 (Cervical)</td>
<td>UMN</td>
<td>UMN</td>
</tr>
<tr>
<td>b: C6-T2 (Cervicothoracic)</td>
<td>LMN</td>
<td>UMN</td>
</tr>
<tr>
<td>c: T3-L3 (Thoracolumbar)</td>
<td>NORMAL</td>
<td>UMN</td>
</tr>
<tr>
<td>d: L4-S3 (Lumbosacral)</td>
<td>NORMAL</td>
<td>LMN</td>
</tr>
</tbody>
</table>

The most consistent and reliable reflexes are the patella reflex in the back legs and tibialis cranialis reflex in the fore limbs. The tibialis cranialis reflex can be difficult to interpret as forelimbs reflexes may be quite subdued in a normal patient.

Intervertebral disc disease (IVDD)

Certain breeds are predisposed to IVDD and if one of the predisposed breeds presents with neck pain then this becomes the number one differential. The most common breed presenting with IVDD in South Africa is the Dachshund which is one of the chondrodystrophic breeds.

Other chondrodystrophic breeds are also over represented, these include the Pug, Bull dog, Bassetthound, Pekinese, Lhasa Apso, Shih Tzu, Beagle, poodle, Cavalier King Charles spaniel, Jack Russell Terrier and Boston Terrier.
There are two types of IVDD described:

Hansen Type 1 This tends to be more of an acute disease process and is associated with extrusion of disc material (nucleus pulposa) through a tear in the annulus and into the vertebral canal. The extruded disc material then causes cord compression and/or nerve root compression resulting in neck pain and neurological deficits.

Hansen Type 2 This is associated with a more chronic process and occurs secondary to tearing of the annulus fibrosis ligament and bulging of the disc dorsally with resultant compression of the spinal cord.

There is more space surrounding the spinal cord in the cervical vertebral canal so a disc prolapse in this area may only impinge on the meninges or nerve roots, causing severe pain, but minimal neurological deficits. There is very little space around the cord in the thoracolumbar region which means that any prolapsed disc material will cause cord compression and neurological deficits.

When making a diagnosis of IVDD in the neck the first diagnostic procedure following on a neurological examination is a plain cervical radiograph. Following basic radiological principles both lateral and dorsoventral views should be obtained. Radiographs are unable to identify disc prolapses or disc extrusions but can identify disc calcifications and spondylitic changes around the vertebra and also exclude other potential causes of disc pain such as fracture, atlanto-axial instability, discospondylitis, vertebral body tumours and articular facet arthritis. Narrowing of disc spaces may be associated with acute disc extrusions but this may often be a misleading sign and caused due to incorrect positioning.

Cervical contrast myelography allows evaluation of compressive lesions of the spinal cord. Before injecting the contrast CSF should be obtained for evaluation in case the myelogram is negative.

Cervical MRI is a more advanced imaging technique that allows for improved evaluation of areas of disc pathology and allows evaluation of the spinal cord itself. On a MRI study areas of inflammation, spinal cord oedema, cystic changes, syrinx formation, spinal tumours and nerve root compressions can be easily appreciated, as well as changes of the extra-spinal soft tissue which may be also associated with neck pain MRI a CSF tap can be performed if it is indicated on the MRI study.

Once a disc extrusion or prolapse has been identified treatment may revolve around pain management or surgical decompression of the spinal cord.

Conservative management can be considered if there are no neurological deficits and cord compression is mild or if nerve root compression is the primary finding. When considering pain management it should be combined with cage rest or strict confinement at home to help prevent further disc extrusion. If conservative management fails then surgery is warranted.

A multi-drug approach should be used. An effective approach uses a muscle relaxant such a diazepam (Valium®) or methocarbamol (Robaxin®), an anti-inflammatory (NSAID) and gabapentin, which manages neuropathic pain. Corticosteroids may initially be used to reduce inflammation but should be limited to the initial 2-4 days before being replaced by non-steroidal. Treatment should be continued for a month after all pain has been controlled.

The surgical approach most often used in the neck is ventral slot decompression. This is an extremely effective treatment with resolution of the underlying problem and almost immediate pain control. This should be considered immediately if there are neurological deficits or if pain is non-responsive to conservative therapy. Proper diagnostic imaging is required prior to surgical intervention and it should only be attempted by an appropriately trained surgeon.

Meningoencephalitis of unknown origin (MUO)
Infectious causes of meningitis are very uncommon in dogs when compared to immune mediated causes. The neurological examination often shows multifocal deficits in these cases. When evaluating a CSF sample titres for canine distemper, toxoplasmosis and neospora infection are checked. A bacterial culture can be considered.

Immune mediated cause of meningitis and encephalitis include:
- Steroid responsive meningitis encephalitis (SRME)
- Granulomatous meningio-encephalomyelitis (GME)
- Necrotising meningioencephalitis (NME)
- Necrotising leukoencephalitis (NLE)
- Steroid responsive meningitis-arteritis (SRMA)

GME, SRME and SRMA can cause neck pain and will be discussed. NME and NLE, common in pugs and Yorkshire terriers, do not cause neck pain and will not be discussed.

GME and SRME
There are a range of glucocorticoid responsive CNS inflammatory diseases. Some respond well to an extended course of glucocorticoids and others are poorly responsive. Diagnosis of the exact type of inflammation is impossible without a brain biopsy – so treatment is initiated and response to therapy will ultimately classify the disease as a simple steroid responsive meningitis or a GME. Prednisolone is the cornerstone of treatment and is started at immunosuppressive doses (2-4 mg/kg PO /day) for 2 to 4 weeks and then gradually reduced or tapered every 4 weeks to the lowest effective dose. If clinical signs...
GME is a devastating inflammatory disease of the CNS that affects young to middle age small and toy breed dogs. Clinical signs may be mild to severe with rapid decompensation and death. GME can be classified into three different forms which include focal, disseminated/multifocal and ophthalmic forms. The disseminated form is the most common. Clinical signs include neck pain, vestibular signs and sometimes cranial nerve deficits and seizures.

Diagnosis is based on a combination of MRI imaging and CSF analysis. The CSF analysis typically has a markedly increased protein content and a pleocytosis ranging from 50-900 WBC's/ul or more. The pleocytosis is typically mononuclear, including mainly lymphocytes (60-90%), monocytes (10-20%) and sometimes a few neutrophils. Common findings on MRI include multiple hyperintensities on T2 weight images and flair images with a predilection for white matter although grey matter may also be involved. T1 images tend to reveal more of a hypo-intensity of the lesions with variable contrast enhancement. On histopathology the lesions are aggregates of eccentric perivascular cuffing by macrophages, lymphocytes and plasma cells. These lesions may form granulomas that invade the CNS parenchyma.

The focal form of GME is a coalescence of the granulomatous lesions to form a space occupying lesion. In the disseminated form the granulomatous lesions are distributed throughout the CNS while with the ophthalmic form the optic nerve, optic disc and the retina may be involved.

The cause of GME is unknown but it is suspected that it is a delayed type hypersensitivity involving major histocompatibility class 2 and a predominance of CD3 antigen-positive T lymphocytes.

The prognosis for GME is poor. Life expectancy is anything from a few days to months and rarely years. Most dogs require continual therapy to control clinical signs. Treatment revolves around the use of immune suppressive therapy. Corticosteroids have always been the mainstay of therapy for this disease. Immune suppressive dosages of up to 2mg/kg twice daily are used initially to help gain control. Corticosteroids come with many undesirable side effects that can have a major impact on the quality of life.

Additional therapies can be added on top of the corticosteroid therapy to get improved control and help reduce the corticosteroid dosages. The chemotherapy drug cytosine arabinoside (Cytosar®) crosses the blood brain barrier in dogs. It acts on mitotically active cells by inserting into DNA molecules causing premature chain termination. It has been used in dogs and humans to treat CNS neoplasms. The dosage used in dogs with GME is 50mg/m² given subcutaneously twice daily for two consecutive days. This is initially repeated every three weeks until remission is obtained. Often it is used together with prednisolone and after the second or third treatment cycle the prednisolone dosage can be dropped to the lowest effective dosage. Once control is obtained after 4 cycles of therapy the interval between the cytosine therapies can be increased. Gloves should be worn when using the drug and white cell counts should be obtained at 2 weeks after the first treatment and then periodically throughout therapy.

Other therapies that can be considered for GME include azathioprine, methotrexate, procarbazine and cyclosporine.

SRMA
Steroid responsive meningitis-arteritis is a disease of young large breed dogs although it is a condition also found in the beagle which is a medium breed dog. The diagnosis relies on exclusion of other causes of neck pain with the use of MRI imaging and evaluation of a neutrophilic pleocytosis and an increased protein content. Treatment consists of immunosuppressive dosages of prednisolone (1-2mg/kg bid) for the first 5 days and then gradually tapered down to the lowest effective dosage over a month to 6 weeks. This is then continued for a further 3-6 months before cessation of treatment. Prognosis is excellent but relapses do occur but respond to dose adjustments. Large breed dogs are sensitive to the muscle catabolic effects of corticosteroids so taper dose more rapidly initially.

Syringomyelia
Breeds affected are the Cavalier king Charles spaniel, Staffordshire bull terrier well as also seen in Miniature Doberman Pinschers and Yorkshire terriers.

Clinical signs are secondary to the fluid filled cavity (syrinx) in the cervical spinal cord and are listed below.

- Neck pain.
- Owners report that dogs resent any touching or grooming of the ear, limb or neck of the "scratched" side.
- Lower motor neuron deficits of the thoracic limb ipsilateral "scratched " side.
- Proprioceptive deficits and ataxia of the pelvic limbs.
- Facial nerve paralysis.
- Head twisted to the "scratched" side.

Persistent scratching at one side of the shoulder/neck area seen most commonly when walking the dog on a leash. No skin contact is made with the scratching. Excitement, exertion and barking could also elicit the response. Clinical signs are first noted between 6 months and 3 years of age. Signs of pain are not well correlated with the size of the syrinx.
Diagnosis of SM can only be made on MRI imaging of the caudal brain and the spinal cord. The goal of imaging is to confirm syrinx formation in the spinal cord and also to demonstrate Chiari type 1 malformation. The caudal baso-occipital bone is short resulting in reduced caudal fossa volume with the caudal cerebellar vermis and medulla extruding through the foramen magnum. The ventricles in the brain are also often distended. These dogs often have fluid filled tympanic bulla. As a minimum both T1 and T2 sagittal sequences of the caudal brain and proximal spinal cord should be taken together with T2 transverse sequences through the proximal spinal cord.

Treatment involves both medical and surgical management. Not all patients require treatment particularly when the syrinx is small or asymptomatic.

Medical treatment revolves around the use of three groups of drugs, analgesics, drugs that reduce CSF production and corticosteroids.

- Analgesics that can be considered are the NSAIDs and oral opioids. Gabapentin may also be used as it has a dampening effect on neuropathic pain.
- Furosemide decreases CSF production and is a useful drug in the management of SM. The carbonic anhydrase inhibitor acetazolamide may also help but can have unpleasant abdominal side effects.
- Corticosteroids are very effective in reducing pain and neurological deficits but the mechanism of action is unknown.

It is recommended that furosemide is tried first for 2 weeks and response is evaluated. If response is poor then a NSAID is added. This is tried for 2 weeks. If poor response then NSAIDS are dropped and Gabapentin is tried. If clinical signs still not controlled then furosemide is discontinued and corticosteroids are tried.

Surgical treatment is reserved for those dogs with refractory pain or worsening of the neurological signs. The aim of surgery is to restore the CSF dynamics. This is achieved by sub-occipital decompression where most of the supraoccipital bone and sometimes the cranial dorsal lamina of the atlas are removed. The largest case series of 16 dogs found that 81.25% of affected dogs had improvement or resolution of clinical signs, but 25% had relapses within the followup period. The earlier the surgery was performed the better the outcome.

**Conclusion**

Neck pain is a common presenting problem in small to medium sized dogs. The breed of the dog should be taken into account as well as a proper history obtained and neurological examination performed before proceeding with further diagnostic procedures in order to expedite a specific diagnosis. Once a diagnosis is made treatment can be tailored for the patient.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of action</th>
<th>Dose in dogs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methocarbamol (Robaxin®)</td>
<td>Muscle relaxant (central action) Secondary sedative effect</td>
<td>15 – 20mg/kg po tid</td>
</tr>
<tr>
<td>Diazepam Pax® Valium®</td>
<td>Striated muscle relaxant</td>
<td>0.2 mg/kg tid</td>
</tr>
<tr>
<td>Gabapentin (Neurontin®)</td>
<td>Neuropathic pain control, Anticonvulsant, blocks calcium channels in spinal cord and supra-spinal levels</td>
<td>3mg/kg oid</td>
</tr>
<tr>
<td>NSAIDS</td>
<td>Inhibits COX - anti-inflammatory Direct spinal effect blocking substance-p and glutamate receptors</td>
<td>2 – 4 mg/kg / day</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Immunosuppressive</td>
<td>CRI 2-5μg/kg/hr for pain management CRI 5-10μg/kg/hr for peri-operative pain</td>
</tr>
<tr>
<td>Opioids</td>
<td>Fentanyl Pure mu agonist, potent, short acting</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Transdermal patches – delayed time to onset: Dogs 18 – 24 hrs Cats 6 – 12 hrs</td>
<td>Small dogs, cats (&lt;5kg): 12.5 - 25μg/hr 5-10kg: 25 μg/hr 10 – 20 kg: 50 μg/hr 20 – 30 kg: 75 μg/hr &gt;30 kg: 100 μg/hr</td>
</tr>
<tr>
<td></td>
<td>Buprenorphine (Temgesic®) Partial / mixed, agonist / antagonist</td>
<td>Dogs: 0.005 -0.02 mg/kg q6 – 12 hrs</td>
</tr>
<tr>
<td></td>
<td>Tramadol Weak mu receptor agonist</td>
<td>Dogs: 5mg/kg every 6hrs</td>
</tr>
<tr>
<td></td>
<td>Ketamine Analgesia and prevents wind up pain NMDA and α-opioid receptor binding</td>
<td>0.1 - 1 mg/kg IM/SQ every 4-6hrs CRI 2-10 μg/kg/min</td>
</tr>
<tr>
<td></td>
<td>Medetomidine Domitor Alpha-2- adrenergic agonist Analgesia in cardiovascularily stable patient Effects last only 30 – 90 minutes so CRI most effective</td>
<td>Loading dose 1μg/kg IV then maintenance at CRI 1-3μg/kg/hr</td>
</tr>
</tbody>
</table>

From Plumbs Veterinary Drug Handbook (7th edition)  
References available from author on request