Differentials for a Pain in the Neck
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Cervical spinal pain, also will be referred to as cervical spinal hyperesthesia (CSH), is an abnormal clinical sign commonly associated with compressive or inflammatory diseases of the cervical spinal region. Hyperesthesia denotes an unpleasant behavioral response to a nonnoxious stimulus. As part of a routine neurologic examination, spinal hyperesthesia is evaluated by deep palpation of the spinal epaxial musculature and by detecting movement resistance with flexion-hyperextension and lateral flexion of the neck. Anatomic structures that are pain sensitive include the meninges, nerve roots, outer 1/3rd of the disc, joints, boney periosteum and muscles.

Neurologic and orthopedic examinations of an animal with spinal or musculoskeletal hyperesthesia determine lesion localization and extent. Neurologic examination of a patient with CSH and suspected cervical spinal cord disease may demonstrate other neurologic abnormalities. Posture of an animal with CSH is typically guarded that is reflective of muscle stiffness. Neck pain may manifest with horizontal neck carriage, increased muscle tone, and intermittent spasms/jerks. Gait may be stilted or stiff and have a shortened stride length especially in the thoracic limbs. Animals with joint, muscle, or meningeal pain often appear to be “walking on eggshells”. Thoracic or pelvic limb lameness also may manifest as radicular pain (nerve root signature). Ataxia, paresis/plegia occurs with myelopathy. If spinal cord compression is present, the severity of postural reaction and motor deficits may depend on the amount and the rapidity of the compressive myelopathy. Often with compressive myelopathy, gait and postural reaction deficits are worse in the pelvic limbs reflective of longer neurotransmission and lateralization of the proprioceptive pathways. Primary meningitis results in slight or no evidence of postural reaction or motor deficits. Animals with muscle and joint pain often have no neurologic deficits but physical discomfort may be associated with decreased withdrawal reflexes and limited responses of some postural reactions. Spinal reflex evaluations localize the lesion within the cervical intumescence (C6-T2) or cranial (C1-5; intracranial).

Testing for spinal hyperesthesia should be performed as the final part of the neurologic examination. Cervical spinal hyperesthesia can be elicited by deep palpation of the cervical spinal epaxial musculature in the region of the transverse processes. Palpation begins distal to the area of suspected disease. Clinical signs include caudal flinching of the ears, twitching of the cervical spinal musculature and behavioral discomfort. The neck is manipulated by ventro-and dorsoflexion and lateral flexion. Normal animals have full range of movement with no resistance. Upon lateral flexion of the neck, the nose is manipulated to touch the truncal region. Resistance or behavioral reluctance to move is evidence of hyperesthesia. Meningeal pain often is diffuse but may predominate in the cervical spinal region. Joint and muscle pain are assessed during palpation and evaluating range of motion. The limb joints are hyper-flexed and extended to elicit evidence of pain.

Pathologic states of clinical pain can be classified as inflammatory pain or neuropathic pain. Tissue damage or inflammation produces pain through stimulation of nociceptors that are sensitive to mechanical, thermal, and chemical stimuli. Neuropathic pain occurs with injury to
neural tissue and represents abnormalities in transmission and somatosensory processing in the peripheral or central nervous system. Some disease processes encompass both nociceptive/inflammatory and neuropathic pain mechanisms. Cancers can infiltrate, and compress neural tissue and pain-sensitive structures or cause unlocalizable pain through paraneoplastic effects. Pain associated with chemotherapy and radiation may result from induced axonal injury and vascular compromise.

Determining the underlying cause for inflammatory and neuropathic pain can help guide appropriate treatment strategies and pain management. The neurologic and orthopedic examinations assist with establishment of differential diagnosis. Disorders of chronic onset and neuropathic pain can be more difficult to manage than those of acute onset and inflammatory pain. Spinal and musculoskeletal pain occur in diseases or disorders associated with compression, inflammation or trauma of pain sensitive tissues (See Table 1). General categorical differential diagnoses include degenerative disease (intervertebral disc disease, caudal cervical spondylomyelopathy, osteoarthritis, and some storage diseases), anomalous (atlantoaxial subluxation), neoplasia (bone or extradural and intradural-extradural-extramedullary masses), inflammatory disease (infectious/noninfectious meningomyelitis and discospondylitis), and trauma (spinal fractures). Common noninfectious inflammatory disorders are granulomatous meningoencephalomyelitis, breed-specific meningoencephalomyelitis and steroid-responsive meningoencephalomyelitis. Neurologic signs of CNS inflammatory disease have concurrent intracranial and spinal cord localization.

Although these general categorical differentials should be strongly considered, veterinary neurologists also recognize cervical spinal pain as a major clinical sign of a primary intracranial mass lesion. Cervical spinal hyperesthesia as a clinical sign of intracranial disease in companion animals may be analogous to the symptom of headache as described in human-beings. The term headache has been applied to brain tumors in dogs and cats. In humans, less than 1% of headaches are caused by the presence of structural intracranial disease; however, one half to two thirds of patients with intracranial masses have headache symptoms. In human patients, tumor-based headaches have been described as dull worsening with posture changes and upon waking. In a review of pathologic headaches associated with brain tumors, the major clinical findings were nausea, vomiting, other neurologic abnormalities and a significant change in headache pattern. Mechanisms for headache associated with intracranial disease in humans are as follows:

- Traction by the mass causing direct or indirect displacement of pain-sensitive structures
- Direct pressure by the mass on cranial and cervical nerves
- Distortion of pain-sensitive areas caused by increased intracranial pressure secondary to obstruction of cerebrospinal fluid flow
- Inflammation in or around pain-sensitive structures
- Distention and dilation of intracranial arteries
- Referred pain
Diagnostic Approach

Signalment, history, and physical and neurologic examination findings will establish presence of a neurologic problem; provide neuroanatomic localization and consideration of differentials (Figures 1 and 2). Time of onset (peracute, acute or chonic), rate of progression (rapid or gradual), and temporal relation (intermittent and/or episodic, stable or chronic) can be established. A minimum database (CBC, biochemistry analysis, urinalysis and thoracic radiography) is recommended especially in animals 5 years of age and older or that have abnormalities on physical examination. Survey spinal radiographs can assist with recognition of obvious abnormalities such as discospondylitis, luxations, and bone neoplasia. If an abnormality is not visualized, advanced imaging and CSF analysis are indicated. Cerebrospinal fluid is collected from the cerebellomedullary cistern and analysis is performed immediately. Myelography is useful for detection, characterization of compressive spinal cord lesions (extradural, intradural and intramedullary) and determining extent of the compression and presence of a dynamic lesion. Computed tomography is used as a primary method to evaluate the spine or assist with determining lesion extent after myelography. Magnetic resonance imaging is becoming a more common diagnostic technique particularly useful in the detection of lesions within the spinal cord. Additional diagnostic procedures include electrodiagnostic evaluation (EMGs and nerve conduction studies), nerve and muscle biopsy, CSF protein electrophoresis, serology, and exploratory surgery.

In cases that involve concurrent cervical spinal pain and intracranial signs, brain imaging should be the primary diagnostic procedure used to establish the presence or absence of an intracranial mass lesion. Intracranial signs may be nonspecific and interpretation may depend on clinical assessment. Careful interpretation of the neurologic examination is essential to avoid diagnostic procedures that are inappropriate for lesion localization and potentially harmful to the patient.
<table>
<thead>
<tr>
<th>Differential Category</th>
<th>Nociceptive/Inflammatory Pain</th>
<th>Neuropathic Pain</th>
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<tbody>
<tr>
<td>Degenerative</td>
<td>Degenerative joint disease (axial and appendicular skeleton)</td>
<td>IVDD (Hansen type I and II), caudal cervical spondylomyelopathy, paraspinal cysts</td>
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<tr>
<td>Anomalous</td>
<td>Axial / appendicular skeletal malformation</td>
<td>Spinal malformation, caudal occipital malformation syndrome (Chiarilike malformation), syringohydromyelia, atlanto-axial instability</td>
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<td>Metabolic</td>
<td>Hyperparathyroidism</td>
<td>Hyperparathyroidism</td>
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<tr>
<td>Neoplastic</td>
<td>Primary and metastatic neoplasms of bone, joint, muscle, spine, meninges</td>
<td>Malignant nerve sheath tumor, Brain tumor, extradural, intradural/extramudullary, intramedullary (less likely) spinal cord tumors, vertebral/skull tumors, metastatic tumors, paraneoplastic</td>
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<tr>
<td>Nutritional</td>
<td>Hypervitaminosis A</td>
<td></td>
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<tr>
<td>Inflammatory (Infectious/Noninfectious)</td>
<td>Osteoarthritis, osteomyelitis, hypertrophic osteodystrophy, infectious and noninfectious meningitis, diskospondylitis, spinal empyema</td>
<td>Meningitis, spinal empyema</td>
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<tr>
<td>Immune</td>
<td>Osteoarthritis, myositis, systemic lupus erythema, rheumatoid disease</td>
<td>Chronic osteoarthritis</td>
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<td>Idiopathic</td>
<td></td>
<td>Spinal arachnoid diverticulum</td>
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<td>Traumatic</td>
<td>Fracture, type I intervertebral disc disease</td>
<td>Spinal fracture, type I IVD extrusion, neuroma, nerve avulsion, syrinx</td>
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<td>Vascular</td>
<td>Osteonecrosis</td>
<td>Ischemic neuromyopathy, extradural hemorrhage</td>
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Figure 1: Diagnostic approach in a patient with neck pain and no neurologic deficits

- **Neck Pain**
  - No Neurologic Deficits
    - Joint pain
    - Muscle pain
    - No muscle or joint pain
      - Survey spinal radiographs
      - Suspect spondylodiscitis
        - Urine and blood culture
        - Brucella titer
        - Echocardiography
        - Abdominal ultrasonography
      - Suspect polymyositis
        - Serum CK levels
        - Electrophysiology
        - Muscle biopsy
        - Antibody titers
        - Serum LE, ANA, RF
      - Suspect polyarthritis
        - Joint tap and analysis
        - Antibody titers
        - Blood and urine culture
        - Serum LE, ANA, RF
        - Joint capsule biopsy
        - R/O other systemic disease

Figure 2: Diagnostic approach in a patient with neck pain and neurologic deficits

- **Neck Pain**
  - Neurologic Deficits
    - Survey Spinal Radiographs
    - CSF Analysis
    - Advanced Imaging
      - CT/MRI Spine
      - CT/MRI Brain
      - Myelography
    - Suspect spondylodiscitis
      - Urine and blood culture
      - Brucella titer
      - Echocardiography
      - Abdominal ultrasonography
    - Suspect inflammatory disease
      - Infectious disease titers
      - Noninfectious causes
    - Degenerative Anomalous Inflammatory Trauma