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Review

Potential Sources of Neck and Back Pain in Clinical Conditions of Dogs and Cats: A Review

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SUMMARY

Pathological neck and back pain occurs in many medical conditions of dogs and cats. Pain may arise from a variety of structures including the intervertebral discs, facet joint capsules, dorsal root ganglia, vertebral ligaments, the vertebral periosteum, and the meninges. The source of this pain is dependent upon the type of disease process and its location within or surrounding the spinal column. Diseases can directly or indirectly stimulate pain sensors (nociceptors). Inflammatory diseases may hypersensitize these receptors or nociceptive pathways with inflammatory mediating substances such as serotonin, histamine and potassium. Diseases resulting in mechanical compression of nociceptors or nociceptive pathways may also result in neck or back pain. A thorough understanding of spinal pain occurring in dogs and cats will lead to more accurate diagnoses and treatments and may provide information regarding prognoses for various diseases. Evidence pointing to sources of spinal pain taken from scientific and clinical studies of a variety of species including humans is provided. Suspected or known sources of neck and back pain occurring in several clinical conditions of dogs and cats are discussed.

KEYWORDS: Anatomy; embryology; ligaments; meninges; spinal.

INTRODUCTION

Pain, as defined by the International Association for the Study of Pain, "is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP Subcommittee on Taxonomy, 1979). Pain is broadly categorized as being either physiological or pathological. Physiological pain is that pain which results from any noxious stimulus that does not result in tissue damage, whereas pathological pain results from noxious stimuli that cause damage or destruction of tissues. Back and neck pain occurring in the North American human population has a lifetime prevalence of 60–84% and 67%, respectively (Waddell, 1996; Cassidy *et al.*, 1998; Cote *et al.*, 1998). In the United Kingdom, neck and lower back pain occur at point prevalence rates of 34% and 37%, respectively (Papageorgiou *et al.*, 1995; Palmer *et al.*, 2001). It has been estimated that the direct medical and indirect costs (lost productivity, etc.) of back pain are approximately $50 (€57) billion annually, and could be as high as $100 (€135) billion annually (Frymoyer & Cats-Baril, 1991). More specifically, direct care cost of back pain in the United Kingdom was £1632 (€2660) million in 1998 (Maniadakis & Gray, 2000). This figure is small in comparison to indirect costs (including production losses) which total £10668 (€17389) million (Maniadakis & Gray, 2000). Although such epidemiological studies have not been performed in veterinary medicine, it seems reasonable that back pain could result in decreased performance of animals (race horses, competitive jumping horses, racing greyhounds, competitive

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sled dogs) which may result in decreased profit to owners and increased morbidity in these animals (Jeffcott, 1980; Jeffcott *et al.*, 1982).

The general public and recently graduated veterinary professionals have an increasing awareness of pain in veterinary medicine and are concerned about the treatment of pain (Dohoo & Dohoo, 1996a, b; 1998). This increasing awareness of pathological pain has prompted the publication of numerous papers dealing with pain and its management (Sackman, 1997; Abbott & Bonder, 1997; Raffe, 1997; Hellyer, 1997; Faggella, 1997; Machin, 1999; Hellyer, 1999a, 1999b; Marks & Haussler, 2000; Mathews, 2000; Lamont *et al.*, 2000; Muir & Woolf, 2001). Until recently, the diagnosis of back pain and its treatment have not been reviewed in veterinary medicine (Jeffcott, 1999). The present review is unique in that it attempts to describe potential sources of back/neck pain occurring in dogs and cats by drawing from the experimental, veterinary and human clinical literature. Identification of back pain in small animals and therapies for back pain will only be improved if one understands spinal anatomy, structures innervated by pain fibres and receptors, and the pathophysiological mechanisms (for review see Lamont *et al.*, 2000; Muir & Woolf, 2001) of pain. Consequently, a brief overview of spinal embryogenesis

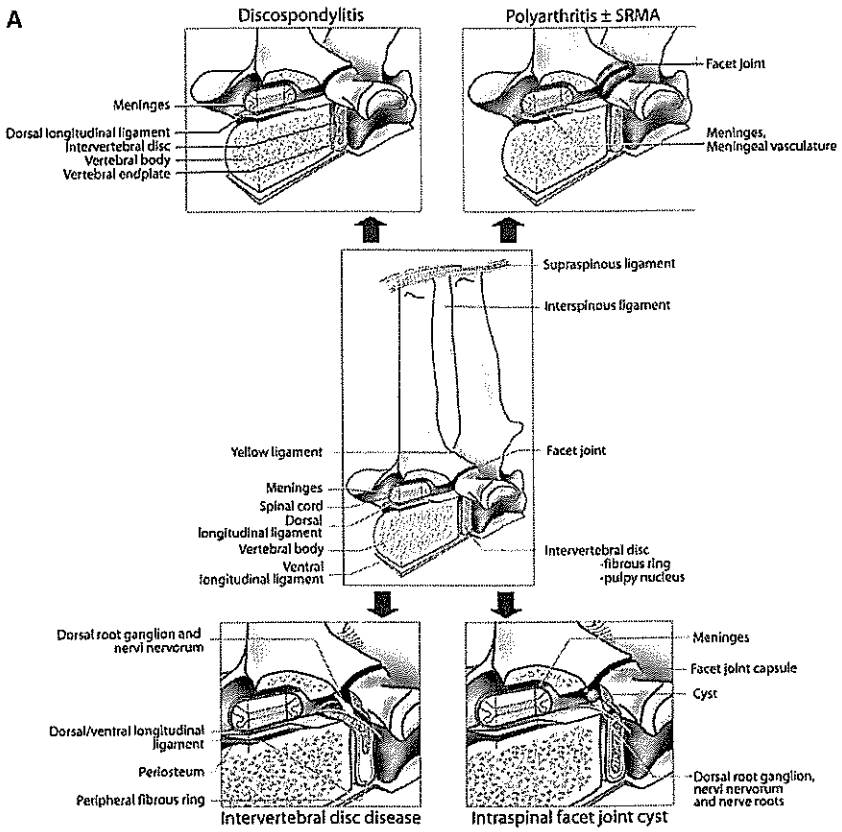

Fig. 1. (A) Knowledge of the basic anatomy of the spinal column is important for understanding potential sources of pain in clinical conditions affecting the neck or back in dogs and cats. A variety of inflammatory and non-inflammatory conditions can lead to neck or back pain. Note the potential structures that could be involved with producing pain in some clinical conditions of dogs and cats. (B) Diagrammatic representation of the spinal cord within a vertebra. The spinal cord, its blood supply (C), nerve roots, and dorsal root ganglia are protected by the bone of the vertebrae. The spinal cord is suspended within the fluid filled dural sac by the denticulate ligaments. The dura within the spinal canal is non-adherent to the bone of the vertebrae. (C) Schematic representation of the spinal cord and its arterial and venous blood supply. Blood enters the spinal cord through the dorsolateral longitudinal and ventral median longitudinal spinal arteries. Venous blood returns from the spinal cord and empties into the valveless internal vertebral venous sinuses which lie on the floor of the spinal canal.

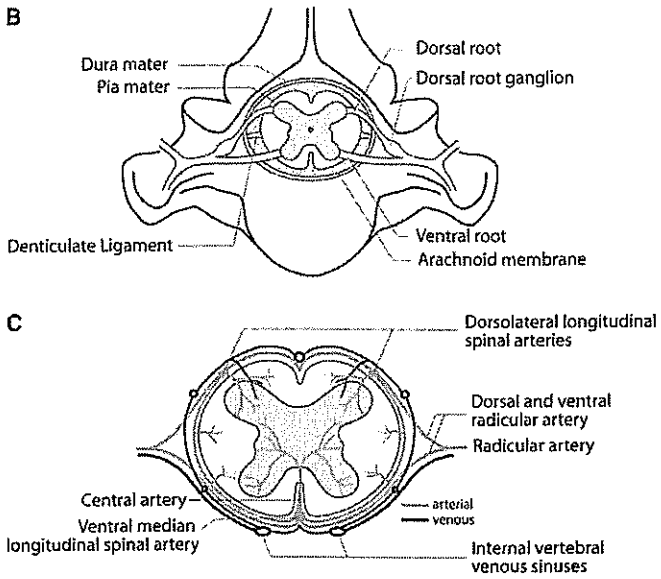

Fig. 1. *(Continued)*

(which may result in congenital anomalies that can lead to painful conditions) is provided and a list of anatomical structures implicated as potential sources of back pain are also provided. Several clinical examples of diseases resulting in spinal pain in dogs and cats are discussed.

THE VERTEBRAL COLUMN AND ITS SUPPORTING STRUCTURES ARE DERIVED FROM MESODERM

Understanding the basic embryogenesis, and the resulting mature anatomy, of the spine is essential for understanding its normal function along with congenital anomalies that might eventually lead to, conditions causing spinal pain.

The function of the vertebral column is to provide structural support (posture) and to protect the spinal cord from trauma. The vertebral column is made up of individual bones called vertebrae. Other components of the vertebral column act to support and provide/limit mobility of the vertebrae and include intervertebral discs, ligaments, and joints (Fig. 1A). Encased within the vertebral column are the spinal cord, its blood supply, nerve roots, and meninges (Figs. 1B and C). As will be discussed, the development of these structures does not occur arbitrarily and with abnormal development of these structures, painful conditions may result in the adult.

The vertebrate embryo is made of three germ layers: the ectoderm, mesoderm, and endoderm. The stage at which these germ layers form is known as gastrulation and occurs during the end of the second week of gestation in many domestic species of animals (Noden & DeLahunta, 1985). Each germ layer gives rise to different types of tissue in the developing embryo. The ectoderm gives rise to musculoskeletal structures of the head, epidermal, and nervous tissues. The mesoderm develops into other musculoskeletal structures, the urogenital, and cardiovascular systems. Endodermally derived tissues include the linings of the digestive and respiratory tracts and those organs directly involved with digestion (Noden & DeLahunta, 1985).

Vertebrae are derived from paraxial mesoderm that is located beside the neural tube (precursor of the spinal cord). The paraxial mesoderm is divided into segments called somites. The stage when somites form is called neurulation and occurs directly after gastrulation. Neurulation occurs at approximately 16 days of gestation for dogs, and 13 days of gestation for cats (Evans & Sack, 1973). Somites are divided into three components including the dermatome, myotome, and sclerotome. Vertebrae are derived from sclerotomes (Bagnall, 1992; Christ & Wilting, 1992; Brand *et al.*, 1996). During vertebral column development, the left and right sclerotomes for each vertebra join, and the medially located

notochord (mesodermally derived) is lost. However, between each vertebra the notochord persists and gives rise to the pulpy nucleus (*nucleus pulposus*) of the intervertebral disc (Trout *et al.*, 1982; Oda *et al.*, 1988; Rufai *et al.*, 1995). Ossification of the canine vertebral column begins during the sixth week of gestation. The differences in shape, the development of articulation between vertebrae, and the development of ribs depends upon the organizational structure of cells making up each sclerotome. The differentiation and organization of sclerotomal cells most likely results from predetermined genetic factors and the influence of the surrounding embryonic environment in which the vertebral column develops. It has been suggested that vascular ischaemic events of vertebral ossification centres may lead to abnormal development of vertebrae (Watson & Stewart, 1990). If vertebrae do not develop normally congenital malformations may lead to painful conditions in the adult and will be discussed later.

As previously mentioned, the vertebrae are distinct from each other and are separated ventrally from each other by an intervertebral disc. The intervertebral disc contains a centrally located pulpy nucleus contained within a fibrous ring (*annulus fibrosus*) (Bray & Burbidge, 1998b). The vertebral endplate is a cartilagenous structure located on either side of the disc. Its function is to provide nourishment to avascular disc tissue (Lammi *et al.*, 1998). If the endplate does not develop normally, it is plausible that painful conditions such as intravertebral disc herniations (Schmorl's nodules) may occur (Saluja *et al.*, 1986; Gaschen *et al.*, 1995).

Development of the intervertebral discs occurs during chondrification of the sclerotomes (Evans & Miller, 1993). The pulpy nucleus is thought to develop from the notochord (Trout *et al.*, 1982; Evans & Miller, 1993; Rufai *et al.*, 1995). The fibrous ring forms around the nucleus from sclerotomal mesenchymal tissue. Collagen types I, II, III, IV, IX, X, and XI have been identified in mature intervertebral discs (Lammi *et al.*, 1998). The function of the intervertebral disc is to absorb compressive shocks and withstand torsional forces (Johnson *et al.*, 1984).

Ligaments of the vertebral column provide mechanical support and assist in the maintenance of posture in both bipedal and quadrupedal species (Jiang *et al.*, 1995). Ligaments of the vertebral column develop at approximately the same time as the vertebrae (Borisevich & Komarova, 1989). Instability of vertebrae may result from, or be enhanced by, agenesis or malformations of spinal ligaments (Watson *et al.*, 1989). Vertebral instability may result in malarticulations between vertebrae which may lead to abnormal "wear and tear" of joints, or may contribute to nerve root and/or spinal cord impingement, both of which may lead to pain (Jiang *et al.*, 1995).

The term meninges is used when referring to the three protective membranous layers of the central nervous system. These layers include the *dura mater*, the arachnoid membrane and the *pia mater* (Evans & Miller, 1993). The arachnoid membrane and the *pia mater* are collectively known as the leptomeninges. The meninges of the spine are derived from somitic mesoderm (Catala, 1998). However, the meninges of the brainstem and cerebral hemispheres arise from cephalic mesoderm and neural crest respectively (Catala, 1998). Meningeal development occurs prior to cerebrospinal fluid (CSF) formation (Catala, 1998). Blood supply to the spinal cord occurs via vessels that traverse through the subarachnoid space and are enveloped by the leptomeninges prior to traveling into the spinal cord (Evans & Miller, 1993). The *pia mater* is thickened on either side of the spinal cord. These thickenings are known as the denticulate ligaments (Evans & Miller, 1993). Denticulate ligaments periodically traverse the arachnoid membrane and attach to the *dura mater* (Fig. 1B). The resulting function of the denticulate ligaments is to suspend the spinal cord within the surrounding cerebrospinal fluid. Conditions where congenital malformations of the meninges may occur include intracranial or spinal arachnoid cysts (Parker *et al.*, 1983; Grevel *et al.*, 1989; Bentley *et al.*, 1991; McKee & Renwick, 1994; Hardie *et al.*, 1996; Milner *et al.*, 1996; Cambridge *et al.*, 1997; Shamir *et al.*, 1997; Vernau *et al.*, 1997; Webb, 1999). Arachnoid cysts are not truly cystic but are dilations of the arachnoid membrane. Arachnoid cysts may cause compression of nerve roots, or adjacent structures which can result in back pain (Webb, 1999).

VARIOUS ANATOMICAL SPINAL STRUCTURES MAY CONTRIBUTE TO NECK OR BACK PAIN

There are various anatomical structures that may play important roles in the genesis of back pain. However, there is no model for studying back pain. Much of what has been hypothesized regarding the origin of back pain is based upon: (1) describing structures that are innervated and have nociceptors (Frymoyer & Gordon, 1989; Gronblad *et al.*, 1991b), (2) associations made between advanced imaging, clinical, and pathological findings (Adams *et al.*,

1995; Songer *et al.*, 1995; Fujiwara *et al.*, 1998; Beattie & Meyers, 1998; Frendin *et al.*, 1999; Ross, 1999; Atabaki & Ochsenschlager, 1999; Maroon *et al.*, 1999; Saifuddin *et al.*, 1999; Luoma *et al.*, 2000), and (3) the resolution of clinical signs following various treatments (Morgan *et al.*, 1993b; Ness, 1994; Sukhiani *et al.*, 1996; Webb, 1999). The following discussion identifies structures of the vertebral column itself, and does not include surrounding muscle or soft tissues (other than vertebral ligaments and discs, etc.), as sources of back or neck pain. Nevertheless, it should be noted that soft para-spinal tissues are important potential sources of back pain (Frendin *et al.*, 1999; Baum & Essfeld, 1999; Liu *et al.*, 1999; Valberg, 1999). Structures having been implicated as sources of back pain include intervertebral discs, facet joint capsules, dorsal root ganglion, vertebral ligaments, the vertebral periosteum, and the meninges.

Intervertebral discs

Intervertebral discs are found throughout the entire length of the vertebral column, except between the first and second cervical vertebrae and the fused sacral vertebrae. Their function is to absorb compressive forces exerted throughout the spine (Bray & Burbidge, 1998b). Intervertebral discs play a role in back pain as they are partially innervated. It is known that the outer third of the fibrous ring is innervated by branches of the sinuvertebral nerve in rats, rabbits, dogs, and humans (Yoshizawa *et al.*, 1980; Bogduk *et al.*, 1981; Forsythe & Ghoshal, 1984; Bogduk, 1988; Kojima *et al.*, 1990a, 1990b; Yamashita *et al.*, 1993b; Strasmann *et al.*, 1999). High threshold mechanoreceptors (type III mechanoreceptors) have been identified in the outer fibrous ring (Roberts *et al.*, 1995). It is thought that type III mechanoreceptors function as proprioceptors and polymodal nociceptors (Heppelmann *et al.*, 1990; Bove & Light, 1995). Electrophysiological evidence supports the possibility that these receptors may function in nociception in lumbar intervertebral discs of rabbits (Yamashita *et al.*, 1993b). Additional information implicating nerve fibres of the intervertebral disc as having a role in pain transmission includes positive immunohistochemical reactivity for neurotransmitters released by nociceptive neurons including substance P and vasoactive intestinal peptide (VIP) (Gronblad *et al.*, 1991b). Cloward (1959) demonstrated that direct stimulation of the fibrous ring could induce reproducible pain in predicted locations.

Facet joint capsule

Facet joint capsules are also potential sources of back pain as they are highly innervated by both mechanosensitive and other free nerve endings (Jackson *et al.*, 1966; Wyke, 1979; Cavanaugh *et al.*, 1989; Yamashita *et al.*, 1993a). Innervation of facet joint capsules serves both proprioceptive and nociceptive functions (Strasmann *et al.*, 1999). Substance P, calcitonin gene-related peptide (CGRP), and VIP immunoreactive nerves have been found in the joint capsule (Giles *et al.*, 1986; Giles & Harvey, 1987; El Bohy *et al.*, 1988; Gronblad *et al.*, 1991a; McLain, 1993; Strasmann *et al.*, 1999). Silver impregnation of the rat joint capsule revealed several individual axons which were thought to terminate as free nerve endings which may act as nociceptors (Cavanaugh *et al.*, 1989). Injection of hypertonic saline into the facet joint produced back pain in people (Mooney & Robertson, 1976). However, a variety of techniques have been attempted to reduce pain arising from the facet joint with varying rates of success in humans (Wall *et al.*, 1994).

Dorsal root ganglion and nerve roots

The dorsal root ganglion itself may be involved in producing pain in a variety of conditions observed in veterinary medicine. It has been shown that compression of the dorsal root ganglion can produce repetitive firing of afferent fibres lasting up to 20 min (Howe *et al.*, 1977). One theory suggests that mechanical compression results in altered blood flow to nerve roots (Rydevik, 1992) or dorsal root ganglion (Rydevik *et al.*, 1989; Yoshizawa *et al.*, 1991) with subsequent intraneural oedema and alterations in neural functioning which lead to pain (Rydevik *et al.*, 1984). An alternate hypothesis implicates nociceptors of the epineurium (the *nervi nervorum*) (Wall *et al.*, 1994; Sauer *et al.*, 1999). *Nervi nervorum* are mechanically sensitive (Wall and Devor, 1983) and may contribute to pain in any condition where local mechanical stimulation (compression or tension) results.

Spinal ligaments

Free nerve endings have been found in spinal ligaments of many species including rats, rabbits, cats, dogs, and humans (Forsythe & Ghoshal, 1984; El Bohy *et al.*, 1988; Konttinen *et al.*, 1990; Yahia & Newman, 1991; Gillette *et al.*, 1993). These ligaments include the posterior (dorsal) longitudinal ligaments, supraspinal ligaments, and interspinous ligaments but not the yellow ligament (*ligamentum flavum*) (Forsythe & Ghoshal, 1984; El Bohy *et al.*,

1988; Konttinen *et al.*, 1990; Yahia & Newman, 1991; Rhalmi *et al.*, 1993; Gillette *et al.*, 1993; Taylor & Finch, 1993). The nerve fibres innervating the posterior longitudinal ligament are approximately the size of type C and A-delta fibres, thus lending more support for their potential contributions in pain (Cavanaugh *et al.*, 1995). Electrophysiological studies implicate nerves of the supraspinous ligaments of rats as playing a role in nociception (Cavanaugh *et al.*, 1989). Immunoreactivity for substance P and CGRP of nerve fibres in the longitudinal ligaments in the rabbit has recently been described, thus providing more evidence supporting the implication of spinal ligaments as possible sources of back pain (Kallakuri *et al.*, 1998).

The vertebral body and its periosteum

Little information exists regarding the role of the vertebral periosteum, or of the vertebral body itself, as potential sources of back pain. Nevertheless, the authors of a recent study who described the innervation of the human vertebral body hypothesized that the osseous portion of the vertebral body may contribute to back pain (Antonacci *et al.*, 1998). Substance P immunoreactive nerves and nerve terminals have been described in human periosteum (Gronblad *et al.*, 1984). Substance P, CGRP and VIP immunoreactivity has been found in nerves of the periosteum of the tibia in rats (Hill & Elde, 1991). It seems probable that these patterns of innervation are not unique to non-vertebral bones. Consequently, with further investigation, the periosteum most likely represents a potential source of back pain.

Meninges

Finally, the meninges themselves may be involved in producing back pain. It has been postulated for some time that migraine headaches are the result of meningeal blood vessel dilation (Blau, 1978). Unmyelinated C-fibres, from the trigeminal nucleus, innervate meningeal blood vessels of cats and rats (Mayberg *et al.*, 1984; O'Connor & van der, 1986). Physiological evidence demonstrates activity in trigeminal neurons following mechanical, noxious chemical, thermal and electrical stimulation of the dura (Dostrovsky *et al.*, 1991; Bove & Moskowitz, 1997; Sauer *et al.*, 1999; Ellrich *et al.*, 1999; Schepelmann *et al.*, 1999). Interestingly, it has been found that acetylsalicylic acid (a non-steroidal anti-inflammatory drug (NSAID)) given systemically or administered topically to the dura in rats decreases activity of medullary dorsal horn neurons following mechanical stimulation of the dura (Ellrich *et al.*, 1999). The mechanisms by which this occurs is thought to be through peripheral mechanisms (decreased prostaglandin synthesis) and within the central nervous system (decreased central prostaglandin synthesis, opioid and/or serotoninergic mechanisms, or *N*-methyl-D-aspartate (NMDA) mechanisms) (Ellrich *et al.*, 1999). Recently CGRP and substance P immunopositive neurons have been found to be extensively distributed in the lumbar dura of rabbits (Kallakuri *et al.*, 1998). Stimulation of the lumbar dura in cats was found to activate lumbar dorsal horn neurons (Gillette *et al.*, 1993). These findings support the theory that the meninges contribute to neck and back pain in dogs and cats.

BACK PAIN CAN RESULT FROM INFLAMMATORY OR NON-INFLAMMATORY CONDITIONS

As previously mentioned, there are several conditions that cause back pain in dogs and cats. The mechanisms by which this pain is produced can be through inflammatory sensitization of nociceptors or through direct mechanical or chemical stimulation. Most often, veterinarians diagnose neck or back pain in dogs and cats by subjectively observing abnormalities in an animal's gait and/or changes in the animal's demeanor upon palpation and manipulation of the spine and para-spinal structures. Realizing that pain, let alone back pain, is often difficult to determine in dogs and cats, one should consider the pathophysiological mechanisms of a given disease and, if appropriate, assume that there is some component of pain involved and treat the animal accordingly. The following section attempts to group several (but not all-inclusive) potentially painful conditions in dogs and cats into one of two groups; inflammatory or non-inflammatory conditions (see Table I for summarized examples).

INFLAMMATORY CONDITIONS

Meningitis

Meningitis is an inflammation of the meninges. Causes of meningitis may be infectious, immune-mediated or idiopathic (Meric, 1988; Munana, 1996). Clinical signs of meningitis may vary depending on the severity and/or the chronicity of the disease. Meningitis may lead to depression, fever, anorexia, neck or back pain manifesting as cervical

or thoracolumbar stiffness, a variety of abnormal neurological and ophthalmologic signs, and even death (Meric, 1988; Munana, 1996). A variety of inflammatory mediators, produced by endothelial cells (Webb & Muir, 2000) and by inflammatory cells, can cause peripheral sensitization of nociceptors.

Treatment strategies for meningitis are usually focused on controlling the primary cause of the disease. Appropriate antimicrobials should be used for bacterial meningitis (Meric, 1988), and immunosuppressive dosages of corticosteroids should be used for steroid-responsive meningitis-arteritis or granulomatous meningoencephalitis (Munana & Luttgen, 1998). No information exists in the veterinary literature concerning treatment of pain in animals suffering from meningitis. Nevertheless, in cases where corticosteroids are administered, clinicians may be treating pain by decreasing the production of inflammatory mediators, thereby decreasing peripheral sensitization. As previously mentioned, administration of acetylsalicylic acid to rats decreased activity in dorsal horn neurons when the meninges were being mechanically stimulated (Ellrich *et al.*, 1999). With the increased availability of NSAIDs specific for cyclooxygenase-2, it may prove invaluable to investigate the efficacy of these drugs for the treatment of pain in canine and feline meningitis.

Discospondylitis

Discospondylitis is a microbial infection involving the intervertebral disc, endplate, and the adjacent vertebral bone (Fig. 1A) and may occur in dogs or cats (Norsworthy, 1979; Norton *et al.*, 1990; Malik *et al.*, 1990; Watson & Roberts, 1993; Thomas, 2000). Aetiological agents include a variety of bacteria and fungi (Kornegay & Barber, 1980; Kerwin *et al.*, 1992; Dallman *et al.*, 1992; Thomas, 2000). Infection may arise from haematogenous spread of the infectious agent (Kornegay & Barber, 1980; Calvert *et al.*, 1985; Thomas, 2000), foreign-bodies (Moore, 1992; Jacob *et al.*, 1996), or iatrogenic inoculation (Remedios *et al.*, 1996). Clinical signs may vary from fever, lethargy, and inappetance to paresis, paralysis, and anorexia (Hurov *et al.*, 1978; Thomas, 2000). Back pain is the most common clinical sign seen in canine discospondylitis and occurs in over 80% of affected dogs (Thomas, 2000). Several structures may contribute to neck or back pain in cases of discospondylitis. These may include the vertebral body, vertebral endplate, intervertebral disc, dorsal longitudinal ligament, and the meninges. The mechanism causing back pain in this disease is probably chemically mediated (prostaglandins, leukotrienes, histamine, serotonin, etc.) (Dray, 1995). It has been suggested that treatment of discospondylitis should not only be aimed at the aetiological agent (antimicrobials) but should also involve the control of back pain (Thomas, 2000). Suggested therapies for ameliorating pain involve excercise restriction and the use of NSAIDS or other analgesics (Thomas, 2000). It has been suggested that analgesics should be used for only 3–5 days following initiation of antimicrobial therapy, as analgesics may mask clinical signs of worsening discospondylitis (Thomas, 2000).

Non-infectious, non-erosive, idiopathic, immune-mediated polyarthritis

Non-infectious, non-erosive, idiopathic, immune-mediated polyarthritis (IMPA) has been reported to occur most commonly in dogs. Specifically, IMPA is diagnosed when there is evidence of a neutrophilic or mixed inflammatory response of synovial fluid obtained from appendicular joints without finding an orthopaedic, systemic or infectious cause and when there is no radiographic evidence of erosive joint disease (Webb *et al.*, 2002). Clinical manifestations of IMPA include neck or back pain, lameness, appendicular joint swelling, and pyrexia. Recently, a retrospective analysis of 62 cases of canine IMPA has demonstrated that approximately 29% of these dogs had concurrent spinal pain (Webb *et al.*, 2002). Spinal pain in affected animals is thought to result from inflammation and irritation of affected facet joints of the spine (Fig. 1A). Breed-specific arthritides, such as in the Akita, have demonstrated concurrent steroid responsive meningitis-arteritis (SRMA). It has been shown that approximately 46% of animals with IMPA and spinal pain also had concurrent SRMA (Webb *et al.*, 2002). Interestingly, there was no breed predilection for development of IMPA, SRMA, and spinal pain. Nevertheless the results of this study could not exclude the possibility that arthritis of the spinal facet joints was a contributing factor to the pain seen in these dogs. Regardless, it is clear that some dogs with IMPA and spinal pain may in fact have concurrent SRMA. Inflammation of the meninges and meningeal vasculature may thus account for neck or back pain observed in some cases of canine IMPA. Therapy for canine IMPA involves immunosuppressive dosages of corticosteroids and/or cytotoxic drugs for prolonged periods of time. The author believes that animals with IMPA and spinal pain may

Table I
Diseases that commonly produce neck and/or back pain in dogs and/or cats

Disease	*Potential anatomical structures involved in producing pain (structures involved depend on extent of the disease)*	*Diagnostic work-up* (some diagnostic tests are important for determining general health of the animal (important for pre-surgery/ anaesthesia screening), and important for ruling-out other differential diagnoses)*	*Signs associated with disease** (not all signs may be seen for a particular animal)*	*Treatments***
Meningitis	Meninges, meningeal vessels	Complete history, distant, physical (including deep palpation of vertebrae and flexion/extension of neck), neurological, and ophthalmological examinations	Fever, depression, anorexia, *signs of neck or back pain* (stiff gait, reluctance to jump, altered posture of neck and/ or back, pain on deep palpation of spinal column), various neurologic and ophthalmologic signs, death	*Infectious*: appropriate antimicrobial agent, +/− corticosteroids (dexamethasone: 0.15 mg/kg 20 min prior to antimicrobial therapy, then 0.15 mg/kg QID for up to a maximum of 4 days (Fenner, 1990)) corticosteroids must be given before the time of antimicrobial therapy-only used for bacterial meningitis althouth still controversial in veterinary patients, +/− supportive care
		Blood/body fluid analyses: complete blood cell count, serum chemistry profile, urine analysis, cerebrospinal fluid (CSF) analysis, paired serum and CSF IgA levels, blood/ urine/CSF aerobic and anaerobic cultures, appropriate serological testing for infectious diseases		
		Radigraphic imaging: spinal series (rule out discospondylitis or neoplasia)		
		Magnetic resonance imaging/computed tomographic imaging: region based on neuroanatomic diagnosis especially for diagnosis of granulomatous meningoencephalitis		*Steroid responsive meningitis-arteritis*: immunosuppressive doses of corticosteroids (prednisone: 2 mg/ kg PO BID for 1–2 weeks then taper over 6 months to 0.5 mg/kg PO every other day), +/− supportive care

		Central nervous system biopsy: for definitive diagnosis of cases of granulomatous meningoencephalitis		*Granulomatous meningoencephalitis*: immunosuppressive doses of corticosteroids, (prednisone: 1–2 mg/kg PO BID initially then taper gradually as signs improve OR cytosine arabinoside (see Nuhsbaum *et al.*, 2002), radiation therapy, +/− supportive care Pain is probably being treated inadvertantly by treating the underlying disease, may consider using non-steroidal anti-inflammatory drugs
Discospondylitis	Intervertebral disc, vertebral endplate, and body of vertebra	Complete history, distant, physical (including deep palpation of vertebrae and flexion/extension of neck), neurological examinations	Fever, general malaise, inappetance, anorexia, *signs of neck or back pain*, paresis, paralysis	Appropriate antimicrobials, alleviation of neck or back pain using non-steroidal anti-inflammatory drugs (meloxicam: dogs – 0.2 mg/kg SQ loading dose, then 0.1 mg/kg PO q 24 h for up to 5 days) or opioids (e.g. oxymorphone: dog – 0.05–0.2 mg/kg SQ every 4–6 h; cat – 0.025–0.05 mg/kg SQ every 4–6 h) during the acute course of the disease, exercise restriction
	May extend to involve dorsal and ventral longitudinal ligaments and meninges	*Blood/body fluid analyses*: complete blood cell count, serum chemistry profile, urine analysis, cerebrospinal fluid (CSF) analysis, blood/urine/CSF aerobic and anaerobic cultures *Radiographic imaging*: spinal series *Magnetic resonance imaging/computed tomographic imaging*: spinal series *Biopsy/culture of affected tissue*: attempt to provide etiological diagnosis to aid in appropriate antimicrobial therapy		

Table I (*Continued*)

Disease	*Potential anatomical structures involved in producing pain (structures involved depend on extent of the disease)*	*Diagnostic work-up* (some diagnostic tests are important for determining general health of the animal (important for pre-surgery/ anaesthesia screening), and important for ruling-out other differential diagnoses)*	*Signs associated with disease** (not all signs may be seen for a particular animal)*	*Treatments***
Idiopathic Immune-mediated polyarthritis (+/− concurrent steroid responsive meningitis-arteritis)	Facet joints of the spine, +/− meninges, and meningeal vessels	Complete history, distant and physical (including deep palpation of vertebrae and flexion/extension of neck), and orthopaedic and neurological examinations *Blood/body fluid analyses*: complete blood cell count, serum chemistry profile, urine analysis, cerebrospinal fluid (CSF) analysis, cytological analysis of synovial fluid taken from multiple appendicular joints, blood/ urine/CSF/synovial fluid aerobic and anaerobic cultures *Radiographic imaging*: spinal series	Fever, general malaise, appendicular joint swelling, *signs of neck or back pain*, lameness	Immunosuppressive dosages of corticosteroids (prednisone: 2 mg/kg BID PO for 1–2 weeks; then taper to 0.5 mg/kg/day PO over 6 months) or cytotoxic agents (azathioprine: 2 mg/kg PO SID for 2–3 weeks then every other day), +/− supportive care, pain is probably treated inadvertently by treating the disease
Intervertebral disc disease	Intervertebral disc, dorsal or ventral longitudinal ligaments, meninges or meningeal vessels, vertebral endplate, dorsal root ganglion, nerve roots	Complete history, distant, physical (including deep palpation of vertebrae and flexion/extension of neck), and neurological examinations	*Signs of neck or back pain*, varying degrees of neurological dysfunction depending on the neuroanatomical location and severity of spinal cord injury	*Corticosteroids*: Methylprednisolone sodium succinate (MPSS) administered intravenously as a 30 mg/kg IV bolus then as constant rate IV infusion (5.4 mg/kg/h) for 23 h (Bracken *et al.*, 1990) (alternate therapy for MPSS: 30 mg/kg IV bolus then 15 mg/kg IV 2 h later, then 15 mg/kg 6 h after the initial dose (Bagley, 2000))

Blood/body fluid analyses: complete blood cell count, serum chemistry profile, urine analysis, +/− cerebrospinal fluid (CSF) analysis

Radiographic/magnetic resonance/computed tomographic imaging: spinal series (especially neuroanatomically diagnosed region), spinal series with contrast enhancement (i.e., myelography)

Used for acute traumatic spinal cord injury cases only if animal admitted within 8 h of spinal cord injury (use is controversial in both human and veterinary medicine), combine with appropriate decompressive surgery

Surgical decompression: perform appropriate decompressive surgery (i.e. dorsal *vs.* hemilaminectomy *vs.* ventral slot), remove remaining intervertebral disc material, consider fenestration of known degenerated discs

Supportive care: including manual urinary bladder evacuation, hygiene, physical therapy, consider pain management using opioids (e.g. oxymorphone: dog, 0.05–0.2 mg/kg SQ every 4–6 h; cat, 0.025–0.05 mg/kg SQ every 4–6 h), or NSAIDs (meloxicam: dogs – 0.2 mg/kg SQ loading dose, then 0.1 mg/kg PO q 24 h for up to 5 days) *following* surgery (avoid using NSAIDs concurrently with corticosteroids)

Table I (*Continued*)

Disease	*Potential anatomical structures involved in producing pain (structures involved depend on extent of the disease)*	*Diagnostic work-up* (some diagnostic tests are important for determining general health of the animal (important for pre-surgery/ anaesthesia screening), and important for ruling-out other differential diagnoses)*	*Signs associated with disease** (not all signs may be seen for a particular animal)*	*Treatments***
Intraspinal arachnoid cysts	Nerve roots, meninges, meningeal vessels	Complete history, distant, physical (including deep palpation of vertebrae and flexion/extension of neck), neurological examinations *Blood/body fluid analyses*: complete blood cell count, serum chemistry profile, urine analysis, +/− cerebrospinal fluid (CSF) analysis *Radiographic/magnetic resonance/computed tomographic imaging*: spinal series (especially neuroanatomically diagnosed region), spinal series with contrast enhancement (i.e. myelography) *Histopathology*: submit excised "cyst" for histopathological diagnosis (necessary for definitive diagnosis)	*Signs of neck or back pain*, varying degrees of neurological dysfunction depending on the neuroanatomical location and severity of spinal cord injury	*Surgical decompression*: excise cyst +/− marsupialization of meninges, perform appropriate decompressive surgery (i.e. dorsal *vs.* hemilaminectomy *vs.* ventral slot) *Supportive care*: including manual urinary bladder evacuation, hygiene, physical therapy, consider pain management using opioids (e.g., oxymorphone: dog – 0.05–0.2 mg/kg SQ every 4–6 h; cat – 0.025-0.05 mg/kg SQ every 4–6 h), or NSAIDs (e.g., meloxicam: dogs – 0.2 mg/kg SQ loading dose, then 0.1 mg/kg PO q 24 h for up to 5 days) *following* surgery (avoid using NSAIDs concurrently with corticosteroids)

Intraspinal facet joint cysts (synovial or ganglion cysts)	Synovium of facet joints, meninges, meningeal vessels, nerve roots	Complete history, distant and physical (including deep palpation of vertebrae and flexion/extension of neck), neurological examinations *Blood/body fluid analyses*: complete blood cell count, serum chemistry profile, urine analysis, +/− cerebrospinal fluid (CSF) analysis *Magnetic resonance/computed tomographic imaging*: spinal series (especially region neuroanatomically diagnosed), with and without contrast (possible to see rim enhancement and/or delayed filling of cyst) *Histopathology*: submit excised tissue for histopathological diagnosis (necessary for definitive diagnosis)	*Signs of neck or back pain*, varying degrees of neurological dysfunction depending on the neuroanatomical location and severity of spinal cord injury	*Surgical decompression*: excise cyst, perform decompressive surgery +/− arthrodesis of facet joint

Drug dosages were compiled from *Handbook of Small Animal Practice* (1997) 3rd Edition, Ed. R.V. Morgan and references included in the table or recommended on the drug label. Therapy must be tailored accordingly on a case-by-case basis.

*Diagnostic work-up – only perform diagnostic tests that are necessary and useful on a case-by-case basis.

**Clinical signs and treatments vary depending on the severity of the disease and/or the type/degree of neurologic dysfunction.

also likely have concurrent SRMA. Cerebrospinal fluid should be collected and examined from these cases to confirm a diagnosis of SRMA. In addition, it is possible that dogs with SRMA may have concurrent IMPA. Considering that IMPA is easier to monitor than SRMA, it may prove invaluable to determine if dogs with SRMA indeed have concurrent IMPA.

NON-INFLAMMATORY CONDITIONS

Intervertebral disc disease

Intervertebral disc disease (IVDD) occurs commonly in the dog (Gage, 1975; Priester, 1976; Adams *et al.*, 1995) and less commonly in the cat (Heavner, 1971; Littlewood *et al.*, 1984; Sparkes & Skerry, 1990; Kathmann *et al.*, 2000; Munana *et al.*, 2001; Knipe *et al.*, 2001). As previously mentioned, the pulpy nucleus is a remnant of the notochord. The nucleus generally remains jelly-like in approximately 75% of the total number of discs in dogs under the age of four years. However, the pulpy nucleus in chondrodystrophic breeds (Beagle, Dachshund, etc.) loses this jelly-like nature by one year of age (Hansen, 1952). The abnormal development of the nucleus is the result of decreased amounts of glycosaminoglycans, decreased water content, and an increase in the collagen content (Ghosh *et al.*, 1976a; Ghosh *et al.*, 1976b; Cole *et al.*, 1986). These changes are probably genetically related and consequently, certain dogs are predisposed to developing IVDD (Ghosh *et al.*, 1975).

IVDD can be classified as one of two types; Hansen type I or type II (Hansen, 1952). Type I disc disease involves an explosive release of nucleus through the fibrous ring resulting from chondroid degeneration. Type II disc disease occurs when the fibrous ring protrudes from its normal anatomical location, yet is not released from the disc, and is seen with fibroid degeneration of the disc. Type II disc disease is most like the disc disease that occurs in humans (Bray & Burbidge, 1998a). Until recently type II disc disease was thought to be the most likely disc disease occurring in cats. A retrospective analysis of 10 cats with intervertebral disc disease revealed that eight of the cats had Hansen's type I intervertebral disc prolapse (Munana *et al.*, 2001). Clinical signs may include neck or back pain (Morgan *et al.*, 1993b; Sukhiani *et al.*, 1996) which may manifest as altered posture of the neck and/or back, a stiff gait, and reluctance to jump. Other clinical signs may include loss of proprioception, para- or tetraparesis (depending on site of disc prolapse), paralysis, and loss of deep pain (Schulz *et al.*, 1998).

Back pain resulting from intervertebral disc protrusion could be caused by direct mechanical stimulation or chemically mediated sensitization of nociceptors. Direct compression of the dorsal root ganglion, from dorsolateral disc protrusion, may lead to intraneural oedema and altered blood supply to the dorsal root ganglion resulting in abnormal neuronal activity and pain (Weinstein, 1991). Pain may also arise from direct mechanical stimulation of the *nervi nervorum* of the dorsal root ganglion (Weinstein, 1991). Mechanical stimulation of the dorsal root ganglion and nerve roots resulted in elevations of substance P in the dorsal root ganglion in one study (Badalamente *et al.*, 1987). Other potential sources of back pain in animals with intervertebral disc disease include the dorsal or ventral longitudinal ligament, peripheral fibrous ring, meninges, and the periosteum (Fig. 1A). Compression of any of these structures could potentially cause pain. In cases where the meninges have adhered to the spinal cord (adhesive arachnoiditis) or other spinal tissue following myelography or laminectomy, the dura and nerve roots may become bound by fibrotic tissue which subsequently applies traction to these structures resulting in pain (Robertson, 1996).

A variety of substances are released from degenerated discs. These substances include glycosaminoglycans, the pulpy nucleus itself, and lactic acid (Marshall *et al.*, 1977; McCarron *et al.*, 1987; Nachemson, 1969; Diamont *et al.*, 1968). Material from the pulpy nucleus has been shown to cause adhesive arachnoiditis (Haughton *et al.*, 1993). The authors of this study suggested that leakage of pulpy nucleus into the epidural space could cause an inflammatory response in the arachnoid and epidural spaces. The resulting inflammatory response could cause the production of many chemical mediators of inflammation that can result in hyperalgesia through peripheral sensitization. Levels of phospholipase A_2, the rate limiting enzyme of the arachidonic acid cascade, are elevated near the site of intervertebral disc prolapse (Saal *et al.*, 1990), providing further evidence of inflammation resulting from disc extrusion. Inflammatory cytokines including interleukin-1α and β, interleukin-6, and tumour necrosis factor alpha are elevated in prolapsed human lumbar intervertebral discs and are associated with elevations in prostaglandin-E (Takahashi *et al.*, 1996). Cytokines cause increased expression of adhesion molecules on endothelium which results in increased numbers of white blood cells in the

area of inflammation, resulting in further stimulation of the inflammatory cascade (Webb & Muir, 2000). Local accumulation of inflammatory mediators could lead to hyperalgesia. Consequently, it is probably best to remove as much of the remaining prolapsed disc during surgery, not only to reduce spinal cord compression but to limit the degree of inflammation.

Criteria used to treat a dog or cat with intervertebral disc disease medically or surgically vary depending on whether the prolapse has occurred in the cervical or in the thoracolumbar spinal cord (Coates, 2000). However, medical treatments usually include some form of antiinflammatory drug and excercise restrition (Coates, 2000). Recurrence of clinical signs following laminectomy and fenestration may result from incomplete removal of prolapsed disc material, dural adhesions, extrusion of another disc, or surgical manipulation (Coates, 2000). Recurrences of clinical signs vary from 34-40% for medical treatment of thoracolumbar intervertebral disease (Coates, 2000). The majority of animals that go on to develop recurrence of clinical signs do so within two years following commencement of treatment (Coates, 2000). In contrast, surgical therapy for prolapsed thoracolumbar intervertebral discs has a recurrence of clinical signs varying from 2.7% to 41.7%. The recurrence rate appears to depend on the type of surgical methods employed for surgical decompression (Coates, 2000). In light of these reported recurrence rates, and the fact that pet owners may be unwilling to put their pet through a second surgery, it seems reasonable to consider whether back pain can be alleviated medically in those cases where back pain is the predominant recurring clinical sign.

Developmental and degenerative anomalies

A wide variety of conditions that are suspected or proven to result from abnormal development of vertebral structures may cause neck or back pain in dogs and cats. These conditions include intravertebral disc herniations (Saluja *et al.*, 1986; Gaschen *et al.*, 1995; Chandraraj *et al.*, 1998), arachnoid cysts (Grevel *et al.*, 1989; Bentley *et al.*, 1991; McKee & Renwick, 1994; Hardie *et al.*, 1996; Milner *et al.*, 1996; Cambridge *et al.*, 1997; Shamir *et al.*, 1997; Webb, 1999), lumbosacral vertebral malformations (Morgan *et al.*, 1993a), synovial and ganglion cysts arising from the vertebral joint capsules (Levitski *et al.*, 1999a; Levitski *et al.*, 1999b; Perez *et al.*, 2000; Dickinson *et al.*, 2001; Webb *et al.*, 2001), and cervical vertebral malformations (Ng, 1987; Watson *et al.*, 1988; Watson *et al.*, 1989; Watson & Stewart, 1990; Jaggy *et al.*, 1991; Gibson *et al.*, 1995). The mechanism by which each of these diseases may produce pain probably varies. For example, intravertebral disc herniation may cause pain by causing a local inflammatory response in the vertebral body resulting in sensitization of nociceptors. Intraspinal arachnoid cysts, however, cause compression on the dura and may cause traction on nerve roots that may mechanically cause pain. Pain occurring in dogs with intraspinal facet joint cysts may be due to compression of the meninges or from tension and expansion on the synovial lining of the joint (Fig. 1A).

It should be noted that pain might not be manifested at a young age in those animals with developmental anomalies. For example, lumbosacral vertebral malformations are present since birth yet pain may not manifest for several years. Vertebral malformations can lead to osteoarthrosis or intervertebral disc protrusion, leading to narrowing of the vertebral canal and compression of nerve roots (De Risio *et al.*, 2000).

CONCLUSIONS AND RELEVANCE

Diagnosis of neck or back pain in dogs and cats is generally the result of a veterinarian's subjective evaluation of the animal's behaviour. It is probable that many dogs and cats are treated for neck/back pain inappropriately or not at all if they do not show overt clinical signs. One should not be content simply to identify neck and back pain, but should also attempt to determine the aetiology of the pain so treatments and prognoses can be provided to the animal's owner. Although there is no model for studying neck or back pain, knowledge pertaining to potential sources of neck or back pain, and the mechanisms by which a disease may result in pain, is invaluable when considering the cause of pain. For example, abnormal findings on imaging studies (myelography, CT, MRI) may be overlooked, considered insignificant, or may be overinterpreted if the observer is unaware of the anatomical structures that may play potential roles in producing pain. This may lead to inappropriate surgical or medical management that could put the animal's well-being at risk. A thorough understanding of the origin of neck and back pain will lead to improvements in the diagnosis and management of spinal pain in dogs and cats.

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