Back Pain in the Canine a matter of structure or function.

Lowri Davies BVSc MRCVS Cert Vet Acup CCRP
The SMART Veterinary Clinic Ltd, Weighbridge Referral Centre,
Kemys Way, Swansea, SA6 8QF. 01792 799006. www.smartvetwales.co.uk

The incidence of back pain within the canine population is unknown and likely to be vastly underdiagnosed. A significant proportion (80% or more) of canine patients presenting at the SMART Clinic with various orthopedic complaints will demonstrate symptoms consistent with back pain. Presenting signs may vary from a stilted gait and poor balance and co-ordination to behavioral changes, stiffness, inability to jump, shifting lameness or a lameness which localises to one limb without any abnormalities being detected in the limb itself. Poor performance in the canine athlete may also be due to pain in the cervical, thoracic and lumbar region.

The exact aetiology of canine back pain is largely unknown with the majority of cases remaining undiagnosed until a persistent lameness develops or severe spinal cord compression result in paresis or paralysis. An information black hole exists with respect to why back pain develops and as to why there is comparatively such a high rate of disc degeneration in the dog.

In the human field, the issue of low back pain in particular is under discussion at present. Traditionally, researchers and clinicians have assumed that the symptoms of patients with low back pain reflect **structural abnormalities** in the lumbar spine due to some combination of injuries and degenerative changes - **The End Organ Dysfunction Model**. The fundamental premise of this model is that the patient feels back pain due to a nociceptive focus in the spine. Thus, the pain experiences of patients represents normal functioning of the nervous system in the context of tissue injury or dysfunction.

An alternative approach to this model is broadly termed the "**Altered Nervous System Processing Mode**l". In this model the fundamental premise is that patients with low back pain suffer from alterations in nervous system encoding or processing of sensory information, rather than from ongoing injury or dysfunction in some structures of the lumbar spine i.e. it is a **functional problem**. Whether this change is due to physiological changes in the CNS precipitated by nociceptive input or whether it is due to heightened susceptibility to pain either due to genetic factors, depression/anxiety or psychological traits is unclear.

Although many obvious differences exist between the human and the canine model many similarities are also present and the question can again be posed as to whether pain is due to structure or function or a combination of both.

When considering the biomechanics of the canine spine; what forces be they compressive, torsional, shear or rotational could potentially contribute over time to the degenerative structural changes that can be viewed with the aid of present day imaging technology? Once we can answer this, the next question to pose is whether through our clinical examination can we correlate the presenting clinical signs with those changes or are they merely red herrings reflecting structural wear and tear but of no direct clinical consequence.

Biomechanics of the Spine.

The function of the spine is to provide support, flexibility in motion and to protect the spinal cord from injury. Activities of daily living must facilitate twisting and turning, and

jumping while retaining stability. Motions of the spine are never "relaxed" but are the result of highly sophisticated bone and soft tissue interactions in concert with active muscle contraction.
The spine is composed of a series of bone segments connected by discs and ligaments. Flexibility in this rod-like system is provided by small displacements of its multiple linkages. The advantage of this configuration is that only slight movement of each unit is required to facilitate large excursions of the structure as a whole. The net result is an inherently stable structure composed of multiple, relatively immobile segments rather than a few highly mobile articulations. Conversely however, loss of mobility at one segment can have significant and wide ranging implications to the spine as a whole which despite it's structure is prone to failure.
In the Dog, the contours of the vertebral column do not reproduce the dorsal standing profile of the animal. The caudal end of the cervical segment is the most flexible part and allows the dog to access almost any part of the body with it's mouth. Ventral flexion in the cranial thoracic vertebrae facilitate lowering of the head to the ground with very little motion in the cervical vertebrae. The large stable configuration of the cervical vertebra is essential in order to provide a strong anchor point for the cervical musculature. The cervical musculature in turn have to be immensely strong to function as a cantilever to oppose the weight of the head. The dramatic change in vertebral anatomy in the thoracic and lumbar region however reflect the need for increased mobility and function. Considerable mobility of the caudal thoracic and lumbar joints is necessary for the alternating sagittal flexion and extension of the back in the gallop. This enables the hindlimbs to be hurled in front of the forelimbs while bounding forwards. In general the canine spine is far less flexible than that of the human. Apart from the obvious differences in stance this reduction in flexibility is partly attributable to the fact that 16% of the length of the vertebral column is accounted for by the intervertebral discs, compared to 25% in humans.
During normal activities of daily living the Spine is subjected to a combination of Compressive, Shear, Bending and Torsional forces. At the walk, the spine is continuously subjected to compression - tension cycles as the back legs push and the forelimbs pull to facilitate forward motion. This should result in a sinuous snaking or fishing motion along the length of the spinal column. Pain and subsequent muscle spasm will impede this smooth motion and lead to either a reduction in motion through the spinal column as a whole or an increase in rotational motion at various points along the length of the back.
The exact etiology of why the incidence of intervertebral disc disease is so high in dogs remains unclear. We accept that certain breeds are prone to disc degeneration and have attempted to classify degeneration as a disease process i.e. Type 1 and 2 disc degeneration. However is it possible that much of the disease process can be attributed to biomechanical forces acting on the vertebral column?
With the spine in neutral, active contraction of the extensor muscles oppose the load placed on the spine by body weight and serves to prevent kyphosis or lordosis of the spine. If the extensor muscles are weak then body weight can not be opposed. The importance of the abdominal cavity and structure of the abdominal wall must also be considered. If the abdominal cavity is likened to a balloon placed within a can, compression of the balloon from any direction generates pressure which is equal in all directions. This hydrostatic pressure can support loads. The lateral wall resists the tendency for the balloon to bulge or bend outwards. Thus the constrained balloon can support the weight at a given height without further deformation. The abdominal cavity,

if it's walls do not deform (i.e. when the muscles firmly contract) can support some of the weight from above. A weak abdominal wall will not be able to function effectively in this way however and will provide little support for the spine. Often the muscles of the abdominal wall will be in severe bracing mode or in spasm and this may well represent the individuals attempt to try and stabilise the trunk and support the spine. A similar model can be applied to the anulus fibrosus as it contains the nucleus pulposus to support the load across the disk space **(1)**.
Each disc acts as an articulation and minimizes the bending stresses of the bony portions of the spinal column by allowing the spine to flex at much lower stresses. If a disc degenerates, little motion within that segment can occur. Instability results until the deformation creates sufficient stresses to resist further motion. Loss of disc function means greater deformation and greater bending stresses at adjacent disks and vertebrae during a given flexion. A stress concentration is thereby produced because of disc degeneration. The greater the number of rigid segments the more the remaining segments must deform to achieve the same motion.
In the canine, during motion, each spinal unit should demonstrate small excursions and through working as a system of units should facilitate smooth, fairly uniform sagittal and ventrodorsal motion. Motion to either side of the midline should be linear with minimal rotational or shear forces being applied to the column. This scenario is rarely seen within most canine patients and from clinical observation patients can be divided into the following groups.

Hyper-mobile
Increased motion to the left or right of the midline
Increased torsional forces at specific points in the vertebral column (mainly upper thoracic, thoracolumbar and lumbosacral junction).
Splinters and bracers - minimal spinal motion.

Motion occurs at the discs as they deform more for given stress (because of lower Young's modulus) than either vertebral body above or below. As torsional forces continue to be applied, the relative motion of one vertebra about another creates tensile and shear stresses in the annulus fibrosus. The greatest stresses occur in areas furthest from the centre of rotation (neutral axis). Rotation produces asymmetric stresses at the joints. Thus one facet joint closes up and one opens out. Thus compressive and shear stresses are concentrated at one joint and tensile stresses are concentrated in the capsular and ligamentous structures of the contralateral one. Additional resistance to torsional stresses are provided by the rib cage and the costovertebral ligaments.
That rotational forces are detrimental to the disc has been demonstrated in animal models. Hadjipavlou et al. (2) and (3) described a rabbit model involving a torsional injury that leads to accelerated disc degeneration. Sixty-five New Zealand rabbits underwent a surgical facetectomy and a 30-degree torsional lumbar injury. The researchers noted that within 60 to 90 days the rabbits that received the torsional strain exhibited clear signs of disc changes, including thinning, increased phospholipase A2 and decreased NP volume. The control group (surgical facetectomy without the torsional strain) did not exhibit these findings, which suggests the role of torsional strain as a possible mechanism of disc degeneration.

Extensive muscle atrophy and loss of function leading to subsequent mechanical instability in many canine patients may also contribute to disc degeneration and spondylopathy

Miyamoto et al. (4) described an easily reproducible cervical spondylosis model in mice. The researchers noted that cervical disc degeneration was accelerated with detachment of the posterior paravertebral muscles from the vertebrae and resection of the spinous processes along with the supraspinous and interspinous ligaments. Mechanical instability in the cervical spine was elicited by the surgical intervention, thereby accelerating the degenerative process over a 6- to 12-month period. Pathologic changes that occurred as a result of the instability included proliferation of cartilaginous tissue and fissures in the AF, shrinkage of the NP, disc herniation and osteophytic formation (4). It is feasible that long term instability in patients could lead to similar changes as those seen in this experimental model.

In the "ideal" animal, the thoracolumbar spine should form a positive arch. From an engineering perspective this reflects the strongest configuration for load bearing. In this configuration, the epaxial muscles can function optimally and provide maximum stability under dynamic loading. Very few back pain cases will present with this postural outline, rather a negative arch (lordotic back) is often seen. Again from an engineering perspective this now reflects a weak configuration for load bearing and one where the epaxial muscles can no longer function effectively and poor stability is seen under dynamic loading.

It is also important to distinguish between stiffness or splinting of the spine and dynamic stability. The stiffer the spine, the less is it's ability to maintain it's trajectory in the face of external forces and therefore the less stable it is. Though an effective short term strategy for coping with pain, splinting and limiting motion soon becomes a potentiating factor within the back pain complex.

Function and Pain

A straightforward biomechanical model goes a long way to account for why abnormal motion and loading leads to altered radiographic and MRI/Cat Scan findings in a population. However in reality it has proved difficult to find strong correlations between the symptoms reported by human patients and indices of biological pathology in the lumbar spine. Imaging studies have been particularly disappointing - evidence of disk pathology on MRI scans is often seen in asymptomatic patients.(5,6) and longitudinal studies have failed to demonstrate that disk pathology at one point in time predicts later LBP (7,8). Reasonable conclusions from the abundant evidence now available are that: degenerative changes in lumbar intervertebral discs and facet joints are highly prevalent in individuals with and without LBP, these changes increase as a function of age and association between abnormalities of these structures shown on imaging studies and symptoms are modest. Similar changes in the absence of symptoms have been found within the canine population.

Abnormal functioning in the Nervous System.

There is ample evidence that peripheral sustained injury, be it inflammation or neuropathic causes local reorganisation of nociceptive and non nociceptive afferents.

These changes lead to alterations in excitability of the afferents to external stimuli (painful and non painful) and also to changes in resting membrane properties, such that sensory neurones that are usually silent in healthy tissue can now generate spontaneous action potentials (9,10).
The dorsal horn of the spinal cord is the first relay and central processing site for nociception and basic science studies on animals provide ample evidence for plasticity of afferent input processing in various experimental models of persistent or chronic pain.(9,10).Thus the animal studies point to increased gain both in the periphery and the spinal cord in chronic pain.
Given that descending modulatory circuits integrate supraspial, cortical and subcortical information, changes in properties of descending modulation point to the role of cortical influences on the spinal cord processing of nociception. Studies in rodents show that manipulating local circuitry in the anterior cingulate and amygdala, insula and medial prefrontal cortex modulates pain behavior and also changes the response properties of spinal cord nociceptors. moreover there is evidence that in various neuropathic or inflammatory conditions, response properties in multiple supraspinal segments are modified. This circuitry must play a role in the mechanism by which learned behavior can modify response to painful stimuli and reciprocally pain experiences induce changes in behavior, learning and memory.

Brain Function in low back pain patients.

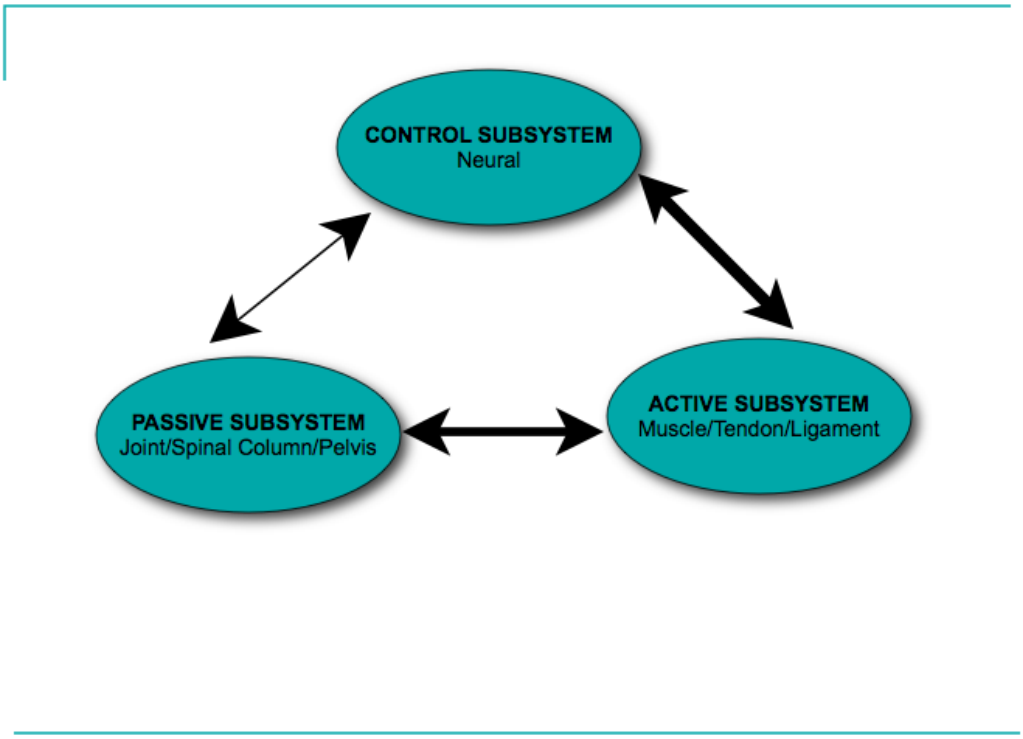
Based on the abundant evidence of brain and spinal cord plasticity in animal studies, one would expect enhanced nociceptive transmission from the periphery to supraspinal targets in patients with LBP. As the spinothalamic pathway is commonly assumed to be the main nociceptive signaling system in the CNS the cortical regions it subserves should indicate enhanced activity either for spontaneous pain or for various external painful and nonpainful stimuli in LBP. Animal models have also demonstrated “fudging” within the cortex of individuals subjected to chronic pain signaling.

What conclusions can we make and how can they be applied clinically?

In summary, research shows that chronic LBP is associated with characteristic functional and anatomical changes within the CNS. Important questions regarding the significance of these changes remain to be explored. In particular it is not known whether the changes are the cause or the consequence of long term pain and whether CNS function and structure return to normal after noxious input form the end organ ceases.
When a dog moves forward, if there is no impediment to movement then no force develops and the body moves forward freely. This however is never the case in reality with gravity, inertia, air and ground forces interacting to oppose motion. To some extent these forces are minimised when a dog is placed on a ground treadmill with no incline.

Once the dog has accommodated through visual and proprioceptive pathways, most of the impediment to free, unencumbered motion should reflect pathology and not external factors. Once abnormal motion has been recognised then it becomes easier to target treatment appropriately.

Accurate visualization of motion coupled with detailed palpation should enable us to deduce what part or parts of the system is malfunctioning. We should be able to identify which muscle groups are functioning and which are not. This in turn should allow us to enhance activity within specific muscle groups which should lead to an improvement in posture over all. Finally our observation should also have provided some information regarding neuromotor control of motion and what strategies can be implemented to enhance this.



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The aim of any treatment regime should be to restore dynamic stability and it is essential to remember that static stiffness does not equate to function. It is essential that we correct for posture, correct for movement and promote functional muscle activation.

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