In large-breed dogs, the lumbosacral (LS) region of the spine is most commonly affected by pathologies (1, 13, 16, 31, 48). The most prevalent disorders involving this region include degenerative lumbosacral stenosis, lumbosacral transitional vertebrae and spondylosis deformans. Large-breed dogs are more prone to develop spine pathologies due to their size and anatomy. The lumbosacral region of the spine is the most commonly affected area because of its long range of motion and the weight it has to bear. Pathologies in this region can lead to decreased mobility and even paralysis if left untreated for too long. Early diagnosis is key for successful treatment and prevention of further complications related to spinal pathologies.

Degenerative lumbosacral stenosis (cauda equina syndrome)

Cauda equina syndrome (CES), also known as cauda equina compression or degenerative lumbosacral stenosis (DLSS), is a condition that usually occurs in older working dogs of large or medium breeds, more commonly in males (26, 47). Working dogs are much more likely to develop acquired disorders. Young dogs, in turn, are more likely to present with congenital disorders leading to CES (11). Dog breeds that are particularly predisposed to CES include German Shepherds, Great Danes, Airedale Terriers, Irish Setters, English Springer Spaniels, Boxers, Labrador Retrievers and Golden Retrievers (4, 37, 47). CES develops as a result of simultaneous compression and damage of numerous nerve roots in the lumbosacral (LS) region of the spine, which resemble a horse’s tail, i.e. cauda equina (CE) (10). Consequently, the compressed spinal cord ganglia with sensory neurons become painful, which manifests with clinical symptoms. The compression is caused by a single or multiple congenital disorders of the spine, e.g. sacral canal stenosis, transitional vertebra or developmental damage, such as aseptic necrosis affecting joint surfaces of the sacrum (22). Dogs with lumbosacral transitional vertebrae, especially those with asymmetric transitional vertebrae, are affected by CES at a much younger age (11). The altered force distribution and mobility in the lumbosacral joint cause premature degeneration of intervertebral discs and lesions in the lumbosacral junction, which lead to CES, sometimes with predominance of unilateral symptoms (24). CES might also be caused by congenital defects, e.g. type II intervertebral disc herniation, lumbosacral instability with secondary thickening of connective tissue structures (yellow ligament or dorsal longitudinal ligament), vertebral body osteophytes, intervertebral foraminal stenosis, consequences of traumas and neoplasms. The clinical signs differ depending on aetiology, compression degree and individual differences (22).

The clinical signs of CES in dogs are most commonly caused by damage to the nerve roots in the L7-Ca5 segment of the spine (9, 47). The resulting pain is known as lumbar pain (27, 47). It may be caused by radiculopathy, annulus fibrosus damage, degenerative lesions of the articular processes or direct irritation of the dura mater. When the above-listed changes occur, the pressure on the LS region in a given animal produces symptoms of pain and discomfort (7, 8, 28). The clinical signs of CES in dogs include pelvic limb...
lameness, bilateral pelvic limb paresis (paraparesis) and a characteristic crouched position with overflexed hip, stifle and hock joints. This results in a shortened pelvic limb stride (hypometria) and additional vocalisation during movement (27, 35, 36). Pelvic limb lameness is usually more prominent when the dog rears up or gets up from a prone position. Furthermore, animals may be reluctant to wag their tail or get up from a prone position. They often avoid physical activity (mainly jumping), as it generates pain (8). Motor function impairment also results in the weakness or paralysis of muscles in the tail. This usually stems from the damage to the dorsal or ventral rami of the caudal nerves and the ventral rami of the sacral nerves innervating the intertransverse muscles, which results in an impaired function of the coccygeus muscle, medial ventral sacrocaudal muscle, ventral lateral sacrocaudal muscle, medial dorsal sacrocaudal muscle, lateral dorsal sacrocaudal muscle and intertransverse caudal muscles. This manifests with the inability to wag the tail or raise it while urinating or defecating. The condition is sometimes accompanied by analgesia (loss of sensation) or dysesthesia or hypeaesthesia (abnormal sensation) and hyperpathia (strong pain), which may result in chewing, licking or mutilating the back of the thighs, metatarsal area, toes, groin, genitalia and tail (28). The most advanced cases of CES may result in full paralysis of the pelvic limbs over time (pelvic limb paraplegia). Furthermore, compression and damage to the spinal cord ganglia with sensory neurons of this region are often associated with difficulties in urinating and defecating and with impaired sexual function (27, 29).

Normal bladder voiding is initiated in the pelvic splanchnic nerve, which transmits an impulse to the sacral segment of the spinal cord. Next, the signal travels along the spinal cord to the pontine reticular formation. The activation of neural fields leads to the release of a motor impulse travelling to the sacral spinal cord segments. As a result, parasympathetic preganglionic motor neurons are activated in the lateral horns of the sacral spinal cord grey matter. Then, the input is transmitted via the pelvic splanchnic nerves. This results in detrusor muscle contraction. At the same time, the sensory input from the pelvic splanchnic nerve is transmitted to the lumbar segments and pudendal motor neurons in the ventral horns of the sacral segment, resulting in sphincter relaxation (27). Damage to the nerves in the L1-L4 spinal cord segments leads to bladder contractions, whereas damage to sacral spinal cord nerves S1-S3 results in an atonic bladder and, consequently, urinary retention. In the latter case, the bladder is flaccid and distended because of the weakened detrusor muscle, while the sphincter tone is preserved, as it is innervated by the intact pudendal nerve (12, 29). The degree of urinary incontinence in dogs varies from sporadic overflow during sleep to constant incontinence. Other dysfunctions found in dogs with CES include defecation abnormalities, such as faecal incontinence, inability to maintain/change position or straining during defecation. The anal sphincter may be atonic, and the perineal reflex absent or weak (27). Sympathetic and parasympathetic innervation of these muscles arises in the lumbar and sacral spinal cord segments, respectively. Dogs affected by CES present two types of sexual problems. Firstly, lumbosacral pain hinders copulation. Secondly, significant CES lesions hinder the reflex-mediated blood flow increase in the external genitals (29).

Another frequent lumbosacral pathological condition in dogs are transitional vertebrae. These are abnormally formed vertebrae which resemble those from an adjacent segment. The lesions may affect the entire vertebra or its part. Transitional vertebrae are most commonly found in the lumbosacral junction. The term lumbosacral transitional vertebra (LTV) denotes an abnormally formed vertebra located between the last healthy lumbar vertebra and the first healthy sacral vertebra (S1), which consequently appears to be part of the lumbar segment, is called lumbarisation of S1.

If the lesion affects the seventh lumbar vertebra (L7), which becomes anatomically similar to the sacral vertebrae, the condition is called sacralisation of L7. The presence of a transitional vertebra results in the development of joint abnormalities and degenerative stenosis in various regions of the spine, which may consequently lead to neurological disorders (3). In a healthy intervertebral joint, adjacent vertebral bodies connect via intervertebral discs. Each intervertebral disc consists of a centrally located nucleus pulposus and the surrounding annulus fibrosus. The bodies of individual vertebrae articulate not only by means of intervertebral discs, but also longitudinal ligaments (dorsal and ventral), whereas the arches of adjacent vertebrae are connected by cranial and caudal articular processes. Interarch spinal spaces are filled by ligamenta flava, which strengthen connections between the articular processes. Spinal ligaments also include interspinous and intertransverse ligaments. The apices of the spinous processes are overlaid by a single supraspinous ligament, which in dogs extends from Fig. 1. German Shepherd, 3-year-old male. The figure shows the lumbar region. Transitional vertebrae are marked with arrows
the spinous process of the first thoracic vertebra to the caudal vertebrae. LTV in dogs predisposes them to intervertebral disc degeneration and CES (24). Furthermore, transitional vertebra may hinder the assessment of hip dysplasia in the dog and thus promote the progression of this condition (21).

The accompanying symptoms of LTV are caused mainly by secondary intervertebral disc degeneration and CES, which follow LTV. The most common symptoms include lumbosacral pain, hyperesthesia, and reluctance to get up and sit down. Hyperesthesia may result from the compression or inflammation of the spinal meninges and nerve roots in the CE region caused by the bony elements of the lumbosacral region, spinal cord compression caused by intervertebral disc L7-S1 or compression to joint capsules of the cranial and caudal articular processes of the L7-S1 intervertebral area. Dogs with lesions in this spinal region often react with pain when extending hip joints, which is most probably associated with the fact that extension of these joints causes spinal cord nerve pinching. Other signs are related to impaired signalling in the afferent proprioceptive fibres in the LS region of the spine. When minor, it can slow down proprioceptive positioning reactions. Severe impairment, on the other hand, may lead to ataxia and numbness in the pelvic limbs. In some cases, when the end part of the spinal cord is damaged, tail movements become weakened due to the decreased muscle tone in the LS region. Damage to the lumbar segments or sacral nerves may result in urinary and faecal incontinence (32). Depending on the severity of clinical signs and the clinical condition of the dog, animals with LTV can receive either conservative or surgical treatment. The indication for conservative treatment is one episode of clinical signs or recurrent moderately severe pain. Conservative methods mainly involve movement limitation for 8-14 weeks, physical therapy, body weight reduction in obese animals, and administration of anti-inflammatory and analgesic agents (41). In dogs with clinical signs, such as severe pain, significant neurological deficits, and especially urinary incontinence, the following surgical procedures are performed: laminectomy, foraminotomy, spinal cord decompression and vertebral stabilisation with plates, bone cement and bone rods (32). Dorsal laminectomy makes it possible to decompress the CE region and expose degenerative lesions in this segment. It involves removing the vertebral arch lamina. During the procedure, the surgeon usually removes some or all of the spinous process of L7. Alternatively, the nerve roots can be moved aside to expose degenerative lesions in the intervertebral disc area. Dorsal or lateral access foraminotomy can be performed using a pneumatic drill or a bone surgical curette. During the procedure, the surgeon removes fragments of articular processes at L7-S1 to widen the L7-S1 intervertebral foramen. Surgical stabilisation techniques include distraction-fusion, fusion and stabilisation with Kirschner wires or pins. The distraction-fusion method is used to widen the intervertebral space and intervertebral foramen, which consequently relieves pressure on the CE nerves (41).

The prevalence of LTV diagnosed in dogs ranges from 2.3% do 40.4% depending on the classification criteria and sample size (6, 24, 46). LTV prevalence in the general canine population is 3.5%, with males being more predisposed to the development of lumbosacral disease secondary to LTV (42). Some authors suggest that LTV may be hereditary (6, 30, 31). Furthermore, asymmetric types of LTV can lead to a pelvic tilt beyond the vertical axis, resulting in a unilateral increase in hip joint loading, causing an abnormal development of the joint (6, 11).

Spondylosis deforms is the third most prevalent pathology of the LS region in dogs. It is a degenerative disorder due to the development of osteophytes in the form of spurs or bridges connecting two adjacent vertebrae (5, 23, 25). Spondylosis most often affects the ventral and lateral aspects of the vertebrae.

The dorsal aspect can also be involved, but less often. Spondylosis has been detected on CT images in 62% of dogs diagnosed with degenerative lumbosacral stenosis (43). Despite the suggestion that spondylosis is associated with Hansen type II herniated nucleus pulposus (25), the disorder has also been observed in combination with normal intervertebral discs. Spondylosis is more commonly found in old dogs, mainly in German Shepherds and Boxers. Definite diagnosis can be made based on radiologic or histopathological tests (5, 23, 25, 30, 38). Spondylosis is often regarded as clinically irrelevant. The exception are working dogs, whose activity may be limited by reduced spinal flexibility (30). There are numerous hypotheses on the aetiology and pathogenesis of spondylosis. One of them points to increased stress in the ligaments and periosteum, which is most often observed in active and working dogs. The stress might also be caused by disc degeneration, trauma or repeated pregnancies. According to another theory, changes in vertebrae are always preceded by the softening of intervertebral discs due to a trauma or the weakening of ligaments or muscles.
This results in motion and disc herniation which exert pressure on the periosteum (17, 18). The clinical signs are caused by osteophytes that extend dorsolaterally, thus compressing the spinal cord nerve roots at the intervertebral foramen level (30). The signs of severe spondylosis include back stiffness, lameness, gait changes and pain (5).

Diffuse idiopathic skeletal hyperostosis (DISH) is a systemic disease which affects structures surrounding the axial skeleton. It causes ossification of soft tissues, particularly the ventral longitudinal ligament, which follows the bodies of the vertebrae from the 7th thoracic vertebra to the sacrum on their ventral surfaces, as well as sites where tendons and articular capsules attach to the bone (15, 20). DISH is characterised by progressive calcification along the spinal vertebrae. The most common lesions do not cause any clinical signs in dogs. The progression of the changes is slow, and the disease is chronic in nature, but the long-term prognosis is generally good. Importantly, the condition is not associated with any risk to internal organs. Problems related to the course of DISH are due to the gradually diminishing flexibility of the spinal fusion, resulting in a reduced motion range of the spine. This results in a decreased agility and a reduced motion range of this axial skeleton region. In very rare cases, DISH may cause painful compression of the spinal cord nerve. Treatment may then involve administering NSAIDs or steroids to reduce inflammation and pain, as well as other analgesics. If pharmacological treatment proves ineffective, it is recommended that surgery be performed to reduce nerve compression (33, 34). DISH may occur alone or in combination with spinal spondylosis, and the prevalence of both these conditions increases with age. In the literature, these two disorders are usually discussed together. However, veterinarians should be aware of the occurrence and potential clinical relevance of DISH, which might cause spinal stiffness and pain, mainly in the lumbar region (20).

Hemivertebra (wedge vertebra) is a congenital disorder occurring during embryogenesis, in which the left and right halves of the vertebral body do not develop in a uniform way. Although the exact aetiology of hemivertebrae is unclear, it is assumed that the condition is hereditary (40). Due to the malformation, the vertebral body parts do not fuse into a uniform structure. The wider part wedges between the adjacent vertebrae (2). This may cause compression to the spinal cord and the resulting neurological symptoms. The most commonly reported clinical signs associated with hemivertebrae include reluctance to move, spinal pain, deep sensory impairment, increased back muscle tone, limb paresis as well as urination and defecation disorders (2, 44). It is suggested that hemivertebrae are more often associated with neurological deficits in Pugs as compared to other brachycephalic breeds, such as French and English Bulldogs (39).

Hemivertebrae are most frequently found in French Bulldogs, English Bulldogs, Pugs and Boston Terriers (2, 14, 39). Although the condition is often accidentally detected during imaging tests, it seems to have greater clinical relevance in Pugs as compared to other brachycephalic breeds (2, 19, 39). It is suspected that a long-term selection of dogs aimed at preserving the so-called screw tail increases the risk of hemivertebra formation in the thoracic and lumbar regions of the spine. Screw tail, which is a desired exterior feature in numerous brachycephalic breeds, is paradoxically caused by abnormal vertebral development in the caudal region of the spine. Therefore, the prevalence of hemivertebrae may increase if dogs with shorter tails are preferentially selected for breeding (40, 45).

References
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