Osseous and Soft Tissue Pathology of the Thoracolumbar Spine and Pelvic Region

Kevin K. Haussler, DVM, DC, PhD, DACVSMR

Author's address—Gail Holmes Equine Orthopaedic Research Center, Department of Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523; e-mail: Kevin.Haussler@ColoState.edu

Take Home Message—Spinal disorders and sacroiliac joint injuries have been identified as significant causes of reduced performance in horses.

I. INTRODUCTION

Clinical conditions affecting the axial skeleton can be categorized as: (1) osseous disorders of the vertebral body, vertebral arch, or vertebral processes; (2) soft tissue disorders involving musculotendinous or ligamentous structures; and (3) neurologic disorders that compromise the spinal cord or spinal nerves. Osseous lesions known to occur in the equine axial skeleton include spinous process impingement,[1,2] degenerative joint disease,[3,4] ankylosis,[1,5] and vertebral fractures.[2] In humans, the most common known sources of back pain are related to the intervertebral disks, zygapophyseal joints, and sacroiliac articulations.[6] Intervertebral disk disease is uncommon in horses, but significant and widespread degenerative changes of the thoracolumbar zygapophyseal joints and sacroiliac articulations have been reported, even in active racehorses.[7] Increased knowledge of osseous pathology inherent in equine athletes will enhance our understanding of the etiopathogenesis of spinal disorders and back pain in horses.

The antemortem diagnosis of axial skeleton injuries and pathology are frequently limited by technological constraints of applied diagnostic imaging modalities due to the large body size or thickness of the trunk and pelvic region of horses. Digital radiographs can provide consistent images of the dorsal spinous processes and with special techniques the thoracolumbar articular processes.[8-10] Depending on trunk size and shape, diagnostic radiographs may also be taken of the vertebral bodies. Radiographic imaging of the pelvic region can be done in both standing and anesthetized horses under special circumstances.[11] Diagnostic ultrasound has been a very useful tool to localize changes in the supraspinous ligament, thoracolumbar articular processes, dorsal sacroiliac ligaments, and the ventral margins of the sacroiliac joint when applied transrectally.[12] Ultrasound-guided injections of the thoracolumbar articular processes, lumbar intertransverse joints, and the sacroiliac region are commonly used in performance horses with back or pelvic pain.[13,14] Nuclear scintigraphy can provide useful insights into areas of inflammation or horses with poorly localized back or pelvic pain.[15,16] Advanced diagnostic imaging (CT, MRI) can be applied to the trunk and pelvic region of foals and some small ponies or horses.[17] Unfortunately, small gantry size has prevented full spine imaging in adult horses.

II. CONGENITAL SPINAL MALFORMATIONS

Developmental variations in the morphology of thoracolumbar vertebral bodies, processes, and joints in horses are known to occur.[18-23] Knowledge of normal spinal morphology and vertebral anomalies is important for distinction of pathologic change from normal anatomic variations. Congenital alterations in the normal spinal curvature include variable degrees of scoliosis, lordosis and kyphosis. Scoliosis (i.e., lateral deviation) is the most common congenital spinal condition. Lordosis (i.e., ventral deviation) may be due to congenitally malformed articular processes but can also be an acquired condition in older horses. Kyphosis (i.e., dorsal deviation) may be either congenital or acquired. Structural spinal curvature abnormalities are usually non-painful and difficult to correct. Congenital vertebral defects (e.g., hemivertebrae) are usually uncommon but can produce structural alterations in the normal spinal curvature, depending on the location and severity of the defect. Prolonged *in-utero* malpositioning of the axial skeleton may cause alterations in the vertebral development that are diagnosed clinically as congenital forms of scoliosis or kyphosis. In adult horses, localized or regional muscle spasms and subsequent soft tissue contractures may also produce functional scoliosis, lordosis, or kyphosis, depending on the injury location.

Transitional vertebrae, by definition, are located between two adjacent spinal regions and have morphologic characteristics of both adjacent spinal regions.[19] Transitional vertebrae can have right-to-left asymmetry or altered cranial-to-caudal gradation in vertebral morphology. Alterations in vertebral morphology often affect the vertebral arches and transverse processes, but rarely the vertebral bodies.[19] In a necropsy survey of 36 Thoroughbred racehorses, 22% of specimens had

thoracolumbar transitional vertebrae (Fig. 1) and 36% had sacrocaudal transitional vertebrae, characterized by sacral fusion with the first caudal vertebra (Cd1).[23] Older horses tend to have the first caudal vertebra fused with the sacrum (Fig. 2). Thoracolumbar transitional vertebrae are probably not clinically significant developmental variations. Although, lumbosacral transitional vertebrae have been reported in two horses with clinical signs of hind limb lameness and possible sacroiliac joint injury.[24] Both horses also had evidence of contralateral sacroiliac joint arthrosis or ankylosis. In dogs, lumbosacral transitional vertebrae have been associated with cauda equina syndrome and possible secondary degenerative joint disease, altered sacropelvic biomechanics, and hip dysplasia.[25-27] Similar compensatory musculoskeletal abnormalities might be expected in horses with appreciably malformed lumbosacral transitional vertebrae.

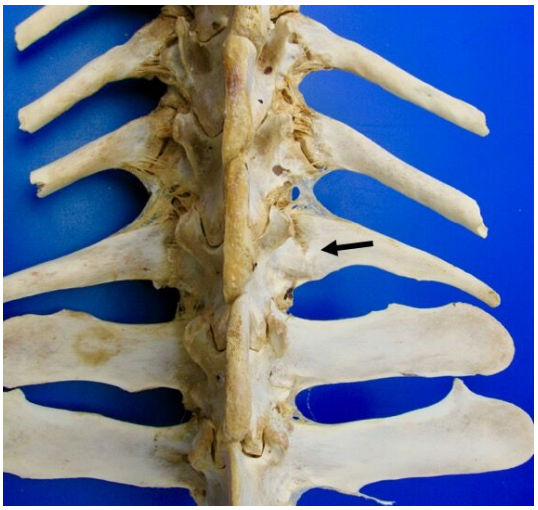


Fig. 1. Dorsal view of thoracolumbar transitional vertebra. Note that the right costal process is rib-shaped and not wide and flattened like a typical lumbar transverse process; however, the costotransverse joint is absent (arrow).

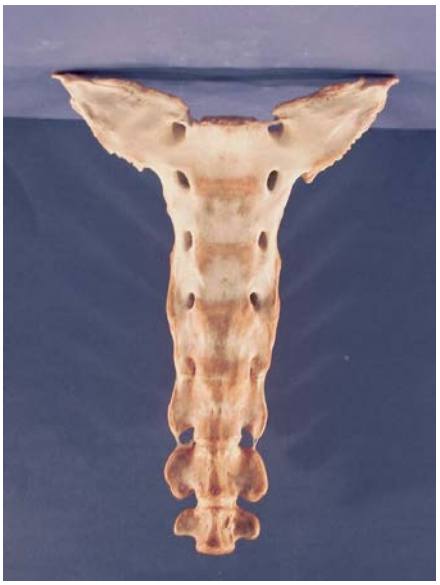


Fig. 2. Ventral view of sacrocaudal transitional vertebra. Note that there are six fused sacral segments, instead of the typical five sacral segments that form the sacrum.

III. VERTEBRAL BODY DISORDERS

Congenital malformations of the vertebral body can produce significant spinal deformities. Block vertebrae are a developmental disorder of the vertebral body characterized by the fusion of two adjacent vertebrae, whereas hemivertebrae are wedge-shaped vertebrae caused by the abnormal development of vertebral body ossification centers. Vertebral body fractures at open vertebral body physes need to be ruled out in foals that experience significant spinal trauma.

Spondylosis is described as an ankylosing or degenerative disease of the vertebral joints that produces large osteophytes that span the intervertebral disk between vertebral bodies (Fig. 3). Spondylosis is initially insidious and subclinical unless inflammation, impingement or fracture of the osseous proliferations occur. In the later stages of development, spondylosis is non-painful due to formation of a complete osseous bridge and eventual ankylosis of affected vertebrae. More commonly, spondylosis is a non-painful osseous bridge formed between two or more vertebral bodies that results in restricted spinal mobility in older horses. In older horses, the thoracolumbar spine is commonly predisposed to a chronic, progressive development of osseous proliferation at the ventrolateral vertebral body margins due to tearing of the outer annular fibers with subsequent osteophyte formation.[1,2] The bony proliferation is continuous with the cortex of the vertebral body and bridges apparently normal vertebral end plates and intervertebral disks. The exact cause of vertebral body osteophytes is unknown but biomechanical and biochemical mechanisms have been proposed.[28] Abnormal joint loading produces microtrauma and degeneration of the annulus fibrosis and periarticular tissues. Portions of the annulus fibrosis and ventral longitudinal ligaments become ossified and produce partial bridging of the involved joints. As the osteophytes increase in size, nerve roots may be compressed at the intervertebral foramen or spinal cord compression occurs if the

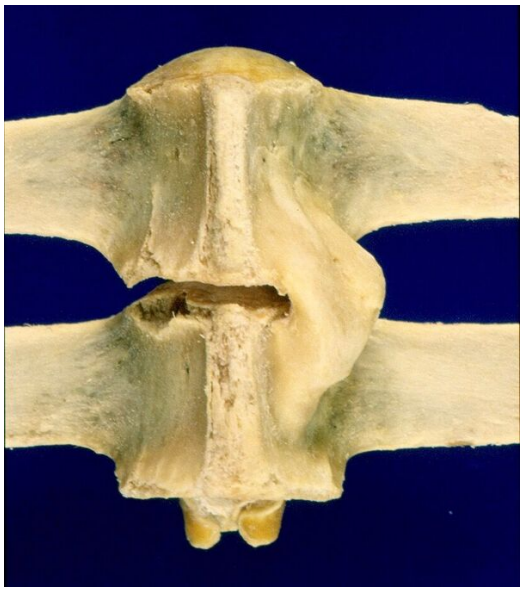


Fig. 3. Ventral view of lumbar spondylosis. A large ventrolateral spondylophyte forms a complete osseous bridge that causes ankylosis between the two lumbar vertebral bodies.

proliferation extends dorsally into the vertebral canal. The cycle of altered joint biomechanics and inflammatory mediators may continue until complete ankylosis and obliteration of the joint occurs. The forming vertebral osteophytes and the ankylosed vertebral bodies are susceptible to fracture due to the reduced ability to absorb or transfer normal locomotor forces through the ossified ligaments and fused vertebral joints. Several vertebral bodies are usually involved, especially in advanced stages. In a radiographic study, Jeffcott found vertebral body osteophytes in only 3% (14 of 443) of horses presenting for thoracolumbar problems.[2] Although, radiographic diagnosis may be limited by variable degrees of ossification, ventrolateral vertebral body location, and inability to obtain consistent quality radiographs of the thoracolumbar spine in horses.

IV. VERTEBRAL ARCH DISORDERS

Stress fractures are usually characterized by several factors which include bone-specific predilection sites, periosteal and endosteal callus, an incomplete fracture line that may progress to complete fracture, tendency for occurring bilaterally, a predominance in athletes undergoing strenuous or repetitive activities, and a patient history of periodic recurrent low grade lameness.[29-31] In a necropsy sample of Thoroughbred racehorses, 50% (18 of 36) of specimens had incomplete fractures and focal periosteal proliferation of the vertebral lamina noted at post-mortem, which were characterized as vertebral stress fractures.[32] Incomplete fractures of the vertebral lamina occurred consistently at the cranial aspect near the junction of a cranial articular process and the spinous process (Fig. 4). Most vertebral stress fractures were continuous with vertical articular clefts of the cranial articular processes, which may provide a site for stress concentration in the etiopathogenesis of equine vertebral stress fractures.[23] Most specimens had one vertebra affected unilaterally, but several specimens had multiple vertebral lamina stress fractures and some were bilateral. Incomplete fracture progression is likely to result in a complete laminar fracture, and unilateral fractures are thought to have a predilection for developing bilateral laminar fractures if excessive mechanical stress or other etiologic factors continued to be present. Bilateral complete fractures of the vertebral lamina were noted in one specimen, so that the dorsal L6 vertebral arch and spinous process could be removed. In horses, articular processes of the caudal thoracic and lumbar portions of the spine (T16-L6) are deeply interlocking and restrict axial rotation.[1,22] Acute articular process fractures in this vertebral region are thought to result from excessive axial rotation.[1] Thoracolumbar articular process morphology may also contribute to the development of vertebral lamina stress fractures. Unfortunately, the complete clinical histories of these Thoroughbred racehorses were unknown. Although, it is possible that vertebral stress fractures may contribute to poor performance and back pain of undiagnosed etiology.

V. ARTICULAR DISORDERS

Osteoarthritis of the thoracolumbar articular processes is common but there is an inconsistent relationship with the presence of back pain.[33] In a necropsy survey of 36 horses, were found various types of articular process degenerative changes in young Thoroughbred racehorses (Fig. 5).[7] Variable degrees of articular process degenerative changes were observed in 35 (97%) specimens and affected 38% (153 of 407) of intervertebral articulations. Degenerative changes were detected at an average of four vertebrae per specimen. Articular surface lipping, osteophytes, periarticular erosions, intra-articular erosions, and ankylosis were noted in the specimens. Articular surface lipping and periarticular erosions of the articular processes were common at the thoracolumbar junction and cranial lumbar spine. Intra-articular erosions and ankylosis were restricted to the caudal lumbar vertebral segments, where vertebral mobility is normally limited.[34] Additional studies are needed to confirm the relationship between radiographic, clinical, and pathologic signs of articular degeneration and back pain in horses.

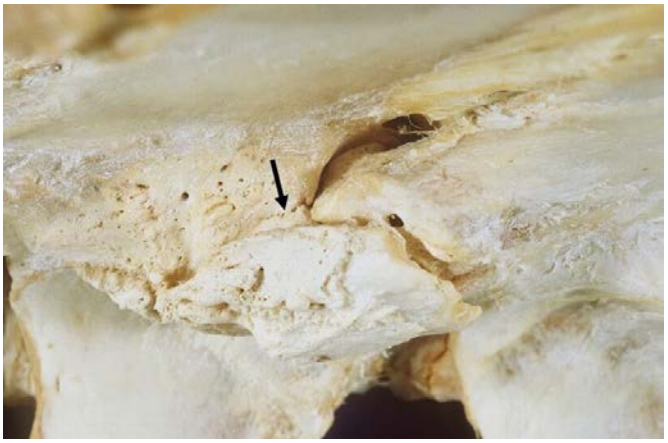


Fig. 4. Dorsolateral view of a vertebral lamina stress fracture. Note the large amount of periosteal callus and the incomplete fracture line (arrow) present over the laminar portion of a lumbar vertebra.

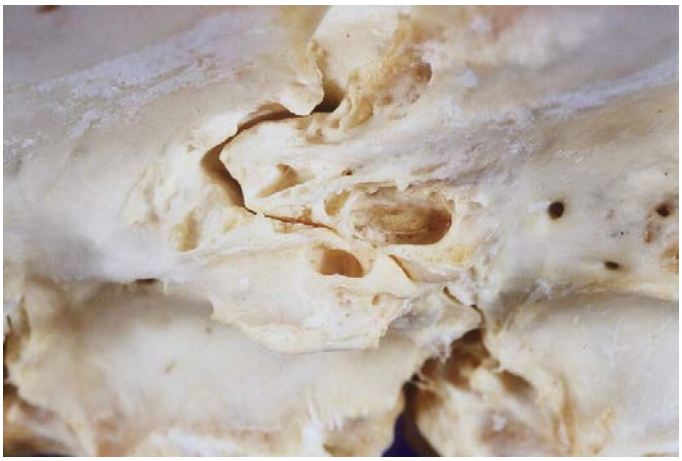


Fig. 5. Dorsolateral view of articular process osteoarthritis and severe periarticular lysis affecting a thoracic intervertebral articulation.

Variable degenerative changes were found at the lumbar intertransverse joints in all specimens of a necropsy survey of 36 Thoroughbred racehorses (Fig. 6).[7] Articular surface lipping, cortical buttressing, osteophytes, intra-articular erosions, and

ankylosis were the types of intertransverse joint degenerative changes noted. Cortical buttressing and the increasing size of the caudad intertransverse joints cause an increased surface area that may contribute to regional vertebral stability and subsequent force transfer between the hind limbs and vertebral column. Intertransverse joint ankylosis was observed in 28% of Thoroughbred racehorses compared with a reported 39% prevalence of ankylosis in a population of mixed breed horses.[35] Intertransverse joint ankylosis is thought to be a developmental condition and not a cause of low back pain.[1,5,35] However, intra-articular erosions were detected in specimens that appeared, if allowed to progress, would likely result in intertransverse joint ankylosis. Therefore, both developmental and acquired intertransverse joint ankylosis probably occurs in horses.[4] Ankylosis of the lumbosacral joint was not detected in any of 245 specimens of mixed equine species evaluated.[36] Lumbosacral joint degenerative changes were milder and less frequent compared with L5-L6 intertransverse joints.[7] Degenerative changes were observed commonly at the ventromedial (i.e., intervertebral foramina) aspect of the lumbosacral intertransverse joints and it is possible that nerve compression could occur with severe, proliferative changes.

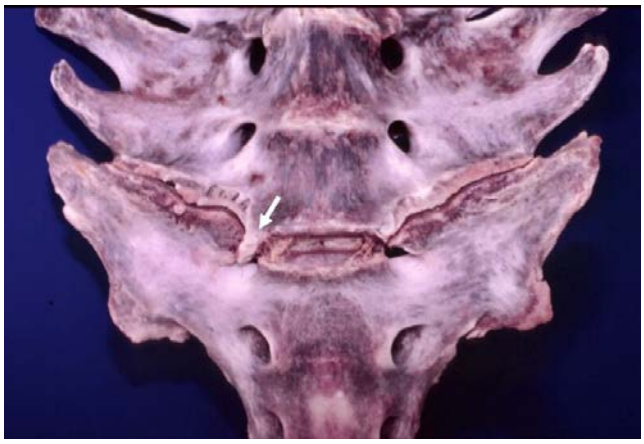
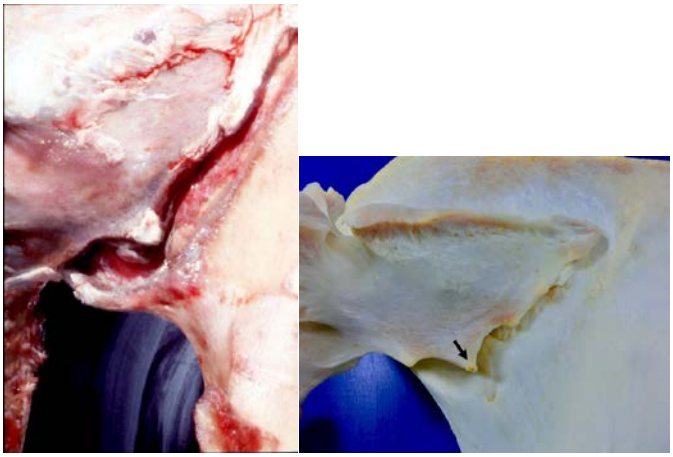


Fig. 6. Ventral view of periarticular lumbar intertransverse joint osteophytes. Note the large osteophyte that is occluding the right ventral L6-S1 intervertebral foramen (arrow). The horse had a chronic right hind limb lameness that was unresponsive to numerous therapies.

Jeffcott, et al reported that the prevalence of sacroiliac damage in performance horses is probably quite high and many cases may go undiagnosed.[3] In a necropsy survey of Thoroughbred racehorses, sacroiliac joint degenerative changes were observed at the sacral and ilial articular surfaces in 100% and 72% of specimens, respectively (Fig. 7).[7] Proliferative changes observed at the sacroiliac articulation included lipping of the articular surface, cortical buttressing, and osteophytes. These lesions tend to be bilaterally symmetrical and localized to the medial aspect of the sacroiliac joint. The pathogenesis of these proliferative changes is uncertain but thought to be related to chronic instability resulting in gradual remodeling and subsequent enlargement of the ventromedial joint surfaces.[3] Osseous changes have been reported at the ventromedial sacroiliac joint margins in horses suspected of having chronic sacroiliac injury; however, no obvious sacroiliac ligament laxity has been observed at necropsy.[2,3,37] Similar expansions of the ventromedial sacroiliac joint surfaces have been reported in a few clinically normal horses.[4] The osseous expansions may develop in response to altered mechanical stresses and assist in the distribution of biomechanical forces. Sacroiliac articular cartilage erosion presumably leads to an eventual sacroiliac joint ankylosis but, ankylosis of the sacroiliac joint has not been reported.[7,38]



7A **7B**

Fig. 7. Cranioventral views of the sacroiliac joint. A, Severe osteophyte proliferation and synovitis. B, Mild osteophytes with a large ventral enthesis (arrow).

Clinical signs associated with degenerative joint disease of the sacroiliac joints need to be differentiated from chronic sacroiliac joint subluxation, which is probably not as prevalent of a clinical condition as has been suggested.[39] Tuber sacralia height asymmetries are often mistaken and interpreted as "subluxation" of the sacroiliac joint; however, only five documented cases of chronic sacroiliac joint subluxation or luxation have been reported in the literature.[40,41] Clinical and radiographic evidence of chronic sacroiliac joint injury has been reported in horses presented for chronic pelvic pain and thoracolumbar disorders.[2,37] Radiographically, some of these cases had an apparent increase in the sacroiliac joint space. A few of these horses were evaluated at necropsy and none of them had reported evidence of visible sacroiliac ligament changes, joint laxity or subluxation.[2,3] Although, most of these specimens displayed varying degrees of sacroiliac joint degeneration. Unless severe acute pelvic trauma has occurred, pelvic asymmetry should be considered an adaptive response to training in a consistent direction on a racetrack of due to chronic muscular imbalances or hindlimb lameness.[42]

VI. SPINOUS PROCESS DISORDERS

Spinous process overlap, without any evidence of osseous impingement, has been noted in horses (Fig. 8).[32] It is thought that localized vertebral process overlap or malalignment may be related to asymmetric ligamentous or musculotendinous forces that induce osseous remodeling and subsequent vertebral process deviation (Fig. 9). In humans, isolated spinous process deviation is a common finding and can be related to vertebral rotation (i.e., scoliosis) or developmental asymmetries in the

neural arch, but rarely is it associated with spinous process fracture or malposition of the entire vertebra.[43,44] Fractures of the spinous process of the withers are often reported following flipping over backwards and landing on the highest point of their back. Conservative care is usually recommended, and proper saddle fit is difficult. Impinged spinous processes (i.e., kissing spines) is characterized by spinous processes that closely appose each other and subsequently induce soft tissue inflammation and osseous proliferation (Fig. 10). The final stage of spinous process impingement is believed to produce a non-painful osseous union of the involved spinous processes.

Fig. 8. Lateral view of overlap of the L4-L5 dorsal spinous processes.

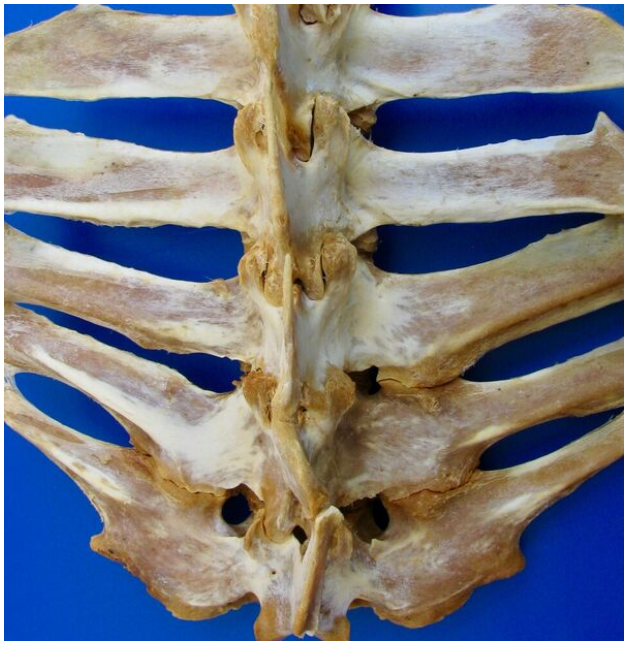
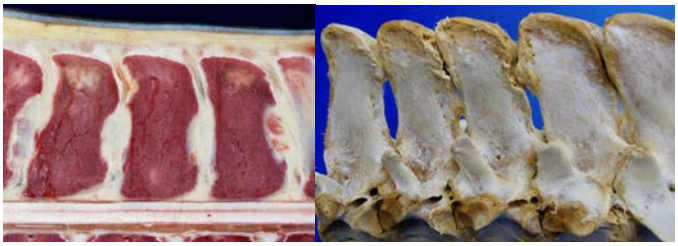


Fig. 9. Dorsal view of misaligned L5-L6 dorsal spinous processes due to impingement.

Impingement of the spinous processes is reported to be the most common osseous cause of back pain in horses.[2] Radiographic evidence of spinous process impingement has been reported in 34% of functionally normal thoracolumbar specimens[21] and 33% of horses with a history of thoracolumbar problems.[2] The horses with functionally normal backs had fewer severe radiographic changes. Gross anatomic evidence of spinous process impingement has been reported in 86% of functionally normal thoracolumbar specimens.[1] The discrepancy between gross and radiographic evidence of spinous process impingement is most likely related to inherent differences in the evaluation techniques. In a necropsy survey of 36 Thoroughbred racehorses, thoracolumbar spinous process impingement or overlap was found in 92% of specimens and was localized to the dorsal ½ or summit (apex) of the thoracolumbar spinous processes (Fig. 10).[7] An average of four impinged thoracolumbar spinous processes was observed per specimen. In some horses, spinous process impingement may also occur at the ventral portion of the spinous processes.



10A **10B**

Fig. 10. Lateral views of impinged dorsal spinous processes. A, Mid sagittal view of pseudarthrosis formation between impinged spinous processes. Note the normal interspinous ligaments and spaces at the cranial and caudal intervertebral sites. B, Osseous specimen showing multiple levels of bony proliferation associated with impinged spinous processes in the caudal thoracic region (T15-L1).

Spinous process impingement in the thoracic spine occurs most commonly between T13-T18, reportedly related to altered spinous process morphology.[1,21] Thoroughbreds have been reported to have a higher prevalence of spinous process impingement compared with other breeds because of misshaped dorsal apices and narrower interspinous spaces.[2,21] Competitive jumping or dressage horses reportedly have a higher prevalence of thoracolumbar spinous process impingement believed related to induced extension or highly demanding spinal maneuvers.[2] Although, dorsoventral vertebral mobility in the caudal thoracic spine has not been reported to differ from other adjacent vertebral regions, therefore increased dorsoventral movement may not account for the vertebral distribution of spinous process impingement.[1,34] Additional weight carrying requirements have also been implicated because this vertebral region is covered by the saddle when rode.[29] Although thoracolumbar spinous process impingement has been reported in Standardbreds and an extinct equine species, *Equus occidentalis*, which presumably have not had extraneous weight placed on their backs.[45] Aging has not been found to be a factor in the pathogenesis of thoracolumbar spinous process impingement.[1,2,46]

VII. TRAUMATIC SPINAL DISORDERS

Dorsal spinous process fractures are typically localized to the wither region (T2-T9) as the spinous processes that form the withers are the first point of contact with the ground when a horse flips over backwards.[2] Conservative care is usually

recommended; however, proper saddle fit is difficult due to lateral displacement of the multiple fracture fragments and widening of the dorsal wither contour. Spinous process fractures are not reported to typically cause spinal cord compromise.[47] Vertebral end plate fractures occur more commonly in foals and are usually reported in association with falls or significant trauma. Vertebral body compression fractures in adult horses have been reported in the thoracolumbar vertebral region (T1-T3, T9-T16 and T18-L6) and are frequently due to physical trauma, electric shock or lightning strike.[47-50] Depending on the severity of the vertebral fracture, minimal fracture displacement is usually found due to surrounding osseous and soft tissue stabilization. A complete neurologic examination will help to localize the fracture site if spinal cord compromise is present.[51,52] Isolated hind limb paresis or paralysis with normal forelimb function suggests a spinal cord lesion caudal to T2. Sacral fractures are a common cause of cauda equina injury. Horses that forcefully back into a solid object or fall backwards can fracture the sacrocaudal vertebral region and produce abnormal perineal neurologic signs. Careful palpation may help to localize a site of pain or vertebral asymmetry.

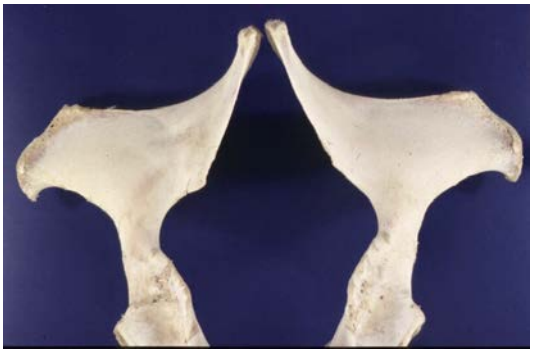
VIII. PELVIC ASYMMETRY AND INJURIES

Variable degrees of osseous and muscular asymmetry are common within the pelvic region of horses.[53] Osseous asymmetries are most commonly noted with left-right differences in tuber sacrale height (Fig. 11). Bilateral prominence of the tuber sacrale (i.e., hunter or jumper bumps) can also occur and may reflect a conformational issue or localized epaxial muscle atrophy in the lumbosacral region that make the bony prominence more apparent. Bony pelvic asymmetries are thought to contribute to reduced performance. Croup asymmetries due to middle gluteal or biceps femoris muscle imbalance may reflect the presence of a chronic hind limb lameness issue.

Pelvic stress fractures have been reported in Thoroughbred racehorses.[29,31,32] Pelvic stress fractures and associated complete fractures occur spontaneously in horses in athletic training without history of acute trauma.[29,54] Most pelvic stress fractures occur unilaterally; however, bilateral fractures have been reported (Fig. 12).[32,55] In contrast to previous reports where complete pelvic fractures occurred predominantly in 4-year-old or younger and female horses,[29,54,56] pelvic stress fractures appear to occur predominantly in older geldings.[32] Pelvic stress fractures occur in consistent locations on the caudal border of the ilium adjacent to the caudomedial aspect of the sacroiliac joint.[29,31,55] Pelvic stress fractures appear to originate at the caudal border of the ilium directly over the region of the sacroiliac joint and course in a craniodorsal or craniolateral direction toward the tuber sacrale or tuber coxae.[32] Fracture pattern and proximity to the sacroiliac joint have possible etiopathogenic implications. The consistent orientation of the incomplete fracture line and the tendency for forking with cranial progression is consistent with tensile forces on the caudal ilial border associated with bending at the sacroiliac articulation. Observations of complete ilial wing fractures also suggest a similar mechanism of fracture.[55] It is likely that pelvic stress fractures affect performance during some portion of their development and are one cause of undiagnosed poor performance and lameness.[29] The proximity of pelvic stress fractures to the sacroiliac joint may be related to sacroiliac joint degeneration.[29,32]

Pelvic fractures are associated with gross physical trauma and are typically localized to the bony prominences of the pelvis which include the tuber coxa, tuber sacralia and ischial tuberosities.[57] Depending on the size of the fracture fragment and the fracture configuration, many pelvic fractures can be managed conservatively. Pelvic fractures that are severe or involve the acetabulum carry a poorer prognosis.[58]

11A



11B

Fig. 11. Caudal view of left-right tuber sacrale height asymmetries. A, In a live horse. B, in a pelvic specimen. Note the additional asymmetry along the caudal aspect of the ilial wings and the left-right differences in tuber coxae heights.

IX. SOFT TISSUE INJURIES

Soft tissue injuries have been noted in 39% of horses with back pain.[2] The most commonly reported injuries included longissimus muscle strain and supraspinous and sacroiliac ligament sprains. Jeffcott reported a complete recovery rate of 73% and recurrence of 25% in horses with soft tissue injuries.[59] Sprains are periarticular injuries caused by overstretching or tearing of a ligament (desmitis) or its bony insertions (enthesitis). Sprains differ from strains, which are injuries to

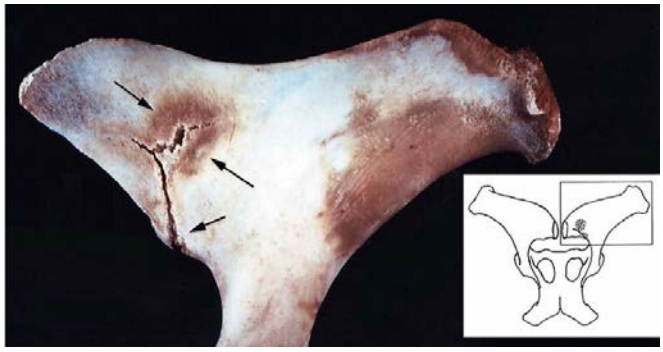


Fig. 12. Dorsolateral view of a severe pelvic stress fracture of the right ilial wing (arrows).

musculotendinous units, although they often occur simultaneously.[60] Ligaments function to maintain joint stability while allowing for joint motion. Partial ligamentous tearing to complete rupture produce disorders varying from subluxation (altered bony alignment) to luxation (complete separation of articular surfaces). Sprains are initiated by overloading of collagen fibers beyond the limits of normal deformation, caused by direct trauma, a fall, or an abnormally restrained movement. Sprains are classified as mild (collagen fiber stretching, intraligamentous hemorrhage, inflammatory exudate) or moderate (significant fiber damage, prominent hemorrhage, and inflammation). Severe sprains are characterized by complete ligamentous disruption, extensive hemorrhage and inflammation, and joint instability (i.e., joint luxation).[60] Clinical signs include localized pain, ligament laxity, localized muscle guarding, heat, swelling, and altered gait. Mild sprains with localized swelling or pain may be difficult to detect by palpation and joint manipulation. Moderate sprains are associated with noticeable lameness, obvious pain and swelling, and ligamentous laxity. Complete ligamentous rupture may result in non-weight-bearing lameness, edema, and extraligamentous hemorrhage, joint crepitus, and joint instability. Signs of inflammation characterize acute sprains, but lameness may vary from none to non-weight bearing. Chronic sprains have minimal inflammatory signs but are characterized by joint capsule thickening and joint instability. Subluxation and luxation result in loss of joint function and mobility and produce obvious postural deformities. In horses, common sites of subluxation or luxation include the pastern, shoulder, coxofemoral, and upper cervical vertebral articulations.

Supraspinous ligament injury or inflammation (desmitis) often produces a palpable localized thickening within the ligament that can be visualized on ultrasonography.[61] The extract clinical relevance of supraspinous ligament desmitis is yet to be fully understood.[62] Diagnostic local anesthesia and diagnostic ultrasound evaluation is recommended to localize and characterize the extent of the desmitis relative to the clinical signs present.

Muscle strain is often due to overexertion or trauma and is characterized by pain, swelling and reduced muscle function. Primary back injuries usually produce a generalized trunk muscle hypertonicity and pain response. Palpable areas of localized pain, muscle hypertonicity and restricted segmental motion are often noted in patients that present with back problems. Strained sub-lumbar muscles will result in back stiffness and reduced hind limb retraction.[63] In horses, abnormal psoas major or psoas minor muscle hypertonicity or injury may be palpated during a rectal examination. Clinical considerations of the fore and hind limb girdle muscles include muscle injury or fatigue that produces an altered gait with a shortened cranial or caudal swing phase of the affected limb.

X. SUMMARY

Pain and dysfunction localized to the thoracolumbar spine and sacroiliac region are common causes of poor performance in horses. An in-depth understanding of the specific structural features and their functional contributions to overall spinal mobility and stability provide a foundation of knowledge necessary to address current sports medicine and rehabilitation issues in athletic horses.

ACKNOWLEDGMENTS

Declaration of Ethics: The author has adhered to the Principles of Veterinary Medical Ethics as stipulated by the AVMA.

Conflicts of Interest: The author has no conflict of interest.

REFERENCES

1. Townsend HG, Leach DH, Doige CE, et al. Relationship between spinal biomechanics and pathological changes in the equine thoracolumbar spine. *Equine Vet J* 1986;18:107-112.
2. Jeffcott LB. Disorders of the thoracolumbar spine of the horse--a survey of 443 cases. *Equine Vet J* 1980;12:197-210.
3. Jeffcott LB, Dalin G, Ekman S, et al. Sacroiliac lesions as a cause of chronic poor performance in competitive horses. *Equine Vet J* 1985;17:111-118.
4. Dalin G, Jeffcott LB. Sacroiliac joint of the horse. 1. Gross morphology. *Anat Histol Embryol* 1986;15:80-94.
5. Rooney JR. A controversy on mechanics of sacroiliac arthrosis in the horse. *Can Vet Journal* 1982;23:144-145.
6. Bogduk N. The anatomical basis for spinal pain syndromes. *J Manipulative Physiol Ther* 1995;18:603-605.
7. Haussler KK, Stover SM, Willits NH. Pathologic changes in the lumbosacral vertebrae and pelvis in Thoroughbred racehorses. *Amer J Vet Res* 1999;60:143-153.
8. Zimmerman M, Dyson S, Murray R. Close, impinging and overriding spinous processes in the thoracolumbar spine: The relationship between radiological and scintigraphic findings and clinical signs. *Equine Vet J* 2012;44:178-184.
9. Zimmerman M, Dyson S, Murray R. Comparison of radiographic and scintigraphic findings of the spinous processes in the equine thoracolumbar region. *Vet Radiol Ultrasound* 2011;52:661-671.

10. Cousty M, Retureau C, Tricaud C, et al. Location of radiological lesions of the thoracolumbar column in French trotters with and without signs of back pain. *Vet Rec* 2010;166:41-45.
11. Gorgas D, Kircher P, Doherr MG, et al. Radiographic technique and anatomy of the equine sacroiliac region. *Vet Radiol Ultrasound* 2007;48:501-506.
12. Nagy A, Dyson S, Barr A. Ultrasonographic findings in the lumbosacral joint of 43 horses with no clinical signs of back pain or hindlimb lameness. *Vet Radiol Ultrasound* 2010;51:533-539.
13. Cousty M, Firidolfi C, Geffroy O, et al. Comparison of medial and lateral ultrasound-guided approaches for periarticular injection of the thoracolumbar intervertebral facet joints in horses. *Vet Surg* 2011;40:494-499.
14. Cousty M, Rossier Y, David F. Ultrasound-guided periarticular injections of the sacroiliac region in horses: A cadaveric study. *Equine Vet J* 2008;40:160-166.
15. Sporn A, Berner D, Winter K, et al. Quantitative evaluation of bone scintigraphy of the spinous processes of the equine thoracic spine at different times after administering (9)(9)mTc-hydroxymethylene-diphosphonate. *Vet Rec* 2014;174:505.
16. Meehan L, Dyson S, Murray R. Radiographic and scintigraphic evaluation of spondylosis in the equine thoracolumbar spine: a retrospective study. *Equine Vet J* 2009;41:800-807.
17. Trump M, Kircher PR, Furst A. The use of computed tomography in the diagnosis of pelvic fractures involving the acetabulum in two fillies. *Vet Comp Orthop Traumatol* 2011;24:68-71.
18. Stecher RM. Lateral facets and lateral joints in the lumbar spine of the horse--a descriptive and statistical study. *Amer J Vet Res* 1962;23:939-947.
19. Getty R. *Sisson and Grossman's The Anatomy of the Domestic Animals*. 5th ed. Philadelphia: W.B. Saunders & Co., 1975.
20. Rooney JR. Congenital equine scoliosis and lordosis. *Clin Orthop Relat Res* 1969;62:25-30.
21. Jeffcott LB. Radiographic features of the normal equine thoracolumbar spine. *Vet Radiol* 1979;20:140-147.
22. Townsend HG, Leach DH. Relationship between intervertebral joint morphology and mobility in the equine thoracolumbar spine. *Equine Vet J* 1984;16:461-465.
23. Haussler KK, Stover SM, Willits NH. Developmental variation in lumbosacropelvic anatomy of Thoroughbred racehorses. *Amer J Vet Res* 1997;58:1083-1091.
24. Jeffcott LB. Linear tomography as an aid to diagnosis of pelvic damage in the horse. *SvVetTidn* 1983;35.
25. Larsen JS. Lumbosacral transitional vertebrae in the dog. *J Am Vet Radiol Soc* 1977;18:76-79.
26. Morgan JP. Congenital anomalies of the vertebral column of the dog: A study of the incidence and significance based on a radiographic and morphologic study. *J Am Vet Radiol Soc* 1968;9:21-29.
27. Morgan JP, Bahr A, Franti CE, et al. Lumbosacral transitional vertebrae as a predisposing cause of cauda equina syndrome in German Shepherd dogs: 161 cases (1987-1990). *J Amer Vet Med Assoc* 1993;202:1877-1882.
28. Clyne MJ. Pathogenesis of degenerative joint disease. *Equine Vet J* 1987;19:15-18.
29. Pilsworth RC, Shepherd MC, Herinckx BM, et al. Fracture of the wing of the ilium, adjacent to the sacroiliac joint, in thoroughbred racehorses. *Equine Vet J* 1994;26:94-99.
30. Shepherd MC, Pilsworth RC, Hopes R, et al. Clinical signs, diagnosis, management and outcome of complete and incomplete fracture to the ilium, in *Proceedings.* Am Assoc Equine Pract1994;40:177-180.
31. Stover SM, Ardans AA, Read DH, et al. Patterns of stress fractures associated with complete bone fractures in racehorses, in *Proceedings.* Am Assoc Equine Pract 1993;39:131-132.
32. Haussler KK, Stover SM. Stress fractures of the vertebral lamina and pelvis in Thoroughbred racehorses. *Equine Vet J* 1998;30:374-381.
33. Denoix J-M. Diagnosis of the cause of back pain in horses. CESMAS - Conference on Equine Sports Medicine and Science 1998;97-110.
34. Townsend HG, Leach DH, Fretz PB. Kinematics of the equine thoracolumbar spine. *Equine Vet J* 1983;15:117-122.
35. Stecher RM. Lateral facets and lateral joints in the lumbar spine of the horse: A descriptive and statistical study. *Amer J Vet Res* 1962;23:939-947.
36. Stecher RM. Anatomical variations of the spine in the horse. *J Mammal* 1962;43:205-219.
37. Jeffcott LB. Pelvic lameness in the horse. *Equine Pract* 1982;4:21-47.
38. Ekman S, Dalin G, Olsson SE, et al. Sacroiliac joint of the horse. 3. Histological appearance. *Anat Histol Embryol* 1986;15:108-121.
39. Townsend HGG. Pathogenesis of back pain in the horse. *Equine Sports Med* 1987;6:29-32.
40. Rooney JR, Delaney FM, Mayo JA. Sacroiliac luxation in the horse. *Equine Vet J* 1969;1:287-289.
41. Rooney JR. Sacroiliac luxation. *Mod Vet Pract* 1979;60:44-46.
42. Jeffcott LB, Dalin G. The sacroiliac joint of the horse and chronic changes associated with poor competitive performance in *Proceedings.* Am Assoc Equine Pract 1986;32:335-351.
43. Van Schaik JPJ, Verbiest H, Van Schaik FDJ. Isolated spinous process deviation: A pitfall in the interpretation of AP radiographs of the lumbar spine. *Spine* 1989;14:970-976.
44. Cramer GD, Darby SA. *Basic and clinical anatomy of the spine, spinal cord and ANS*. St. Louis: Mosby-Year Book, Inc., 1995.
45. Klide AM. Overriding vertebral spinous processes in the extinct horse, Equus occidentalis. *Amer J Vet Res* 1989;50:592-593.
46. Jeffcott LB. A clinical approach to examination of horses with a potential back problem. *In Practice* 1979;1:4.
47. Jeffcott LB, Whitwell KE. Fractures of the thoracolumbar spine of the horse in *Proceedings.* Am Assoc Equine Pract 1976;22:91-102.

48. Wagner PC. Surgical treatment of traumatic disease of the spinal column In: Auer JA, ed. *Equine surgery*. Philadelphia, PA: WB Saunders Co., 1992;1093-1098.
49. Rhoads WS, Cox JH. What is your diagnosis? Compression fracture of the 12th thoracic vertebra. *J Amer Vet Med Assoc* 1997;210:755-756.
50. DeBowes RM, Wagner PC, Gavin PR, et al. Vertebral compression fracture in a foal following electric shock. *J Vet Orthop* 1981;2:14-19.
51. Moyer WA, Rooney JR. Vertebral fracture in a horse. *J Amer Vet Med Assoc* 1971;159:1022-1024.
52. Chiapetta JR, Baker JC, Feeney DA. Vertebral fracture, extensor hypertonia of thoracic limbs, and paralysis of pelvic limbs (Schiff-Sherrington syndrome) in an Arabian foal. *J Amer Vet Med Assoc* 1985;186:387-388.
53. Dalin G, Magnusson L-E, Thafvelin BC. Retrospective study of hindquarter asymmetry in Standardbred Trotters and its correlation with performance. *Equine Vet J* 1985;17:292-296.
54. Jeffcott LB, Rossdale PD, Freestone J, et al. An assessment of wastage in Thoroughbred racing from conception to four years of age. *Equine Vet J* 1982;14:185-198.
55. Hornof WJ, Stover SM, Koblik PD, et al. Oblique views of the ilium and the scintigraphic appearance of stress fractures of the ilium. *Equine Vet J* 1996;28:355-358.
56. Heinze CD, Lewis RE. Radiographic examination of the equine pelvis: case reports. *J Amer Vet Med Assoc* 1971;159:1328-1334.
57. Rutkowski JA, Richardson DW. A retrospective study of 100 pelvic fractures in horses. *Equine Vet J* 1989;21:256-259.
58. Rutkowski JA, Richardson DW. The diagnosis and treatment of pelvic fractures in horses in *Proceedings.* Am Assoc Equine Pract 1987;33:701-705.
59. Jeffcott LB. Back problems in the horse - a look at past, present and future progress. *Equine Vet J* 1979;11:129-136.
60. Farrow CS. Sprain, strain, and contusion. *Vet Clin North Am Small Anim Pract* 1978;8:169-182.
61. Lamas LP. Supraspinous ligament and dorsal sacroiliac ligament desmitis In: Henson FMD, ed. *Equine back pathology: Diagnosis and Treatment*. UK: Wiley-Blackwell, 2009;179-188.
62. Henson FM, Lamas L, Knezevic S, et al. Ultrasonographic evaluation of the supraspinous ligament in a series of ridden and unridden horses and horses with unrelated back pathology. *BMC Vet Res* 2007;3:3.
63. Stashak TS. Adams' Lameness in Horses In: Stashak TS, ed. *Adams' Lameness in Horses*. 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2002.