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Stress fractures of the vertebral lamina and pelvis in Thoroughbred racehorses

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Summary

Thirty-six Thoroughbred racehorses that died at California racetracks between October 1993 and July 1994 were evaluated for stress fractures in the caudal portion of the thoracic and lumbosacral regions of the spine and the pelvis. The lumbosacral spine and pelvis were collected, debrided of soft tissues and examined visually for the presence of an incomplete fracture line and focal periosteal proliferation, characteristic of a stress fracture.

Sixty-one per cent of specimens had evidence of stress fracture in the caudal portion of the thoracic and lumbosacral regions of the spine and the pelvis. Vertebral lamina stress fractures were found in 50% of specimens and were positively associated with the severity of dorsal spinous process impingement and overall severity of articular process degenerative changes. Pelvic stress fractures affected 28% of specimens and occurred more frequently in older horses. Pelvic stress fractures were positively associated with the severity of lumbar transverse process impingement and several ilial articular surface degenerative changes.

A high prevalence of vertebral and pelvic stress fractures was noted in this sample of Thoroughbred racehorses that died because of unrelated injuries. Vertebral and pelvic stress fractures need to be considered in the clinical evaluation of horses with back problems or hindlimb lameness. Undiagnosed stress fractures of the vertebrae or pelvis could be a significant cause of poor performance and lameness in Thoroughbred racehorses.

Introduction

Athletic demands may place excessive strain on musculoskeletal structures of performance horses resulting in injuries related to mechanical failure. Bones subjected to repetitive loading are susceptible to overuse injuries which elicit a stress fracture response, characterised radiographically by an incomplete fracture line, periosteal callus and/or endosteal callus. Evidence of stress fracture has been reported primarily in Thoroughbred (Koblik *et al.* 1988; Mackey *et al.* 1987) and Standardbred (Pleasant *et al.* 1992; Ruggles *et al.* 1996) racehorses exposed to intense levels of exercise. Common long bones affected include the metacarpus (Koblik *et al.* 1988; Pleasant *et al.* 1992), radius (Mackey *et al.* 1987), humerus (Mackey *et al.* 1987; Stover *et al.* 1992), and tibia (Mackey *et al.* 1987; Ruggles *et al.* 1996). Stress fractures in the pelvis and scapula of Thoroughbred racehorses have also been reported (Stover *et al.* 1993; Shepherd *et al.* 1994; Pilsworth *et al.* 1994; Hornof *et al.* 1996). However, the prevalence of these overuse injuries has not been reported for Thoroughbred racehorses, nor have stress fractures been reported in the vertebral column of horses.

The high prevalence of back pain and lumbosacral spinal lesions in performance horses (Jeffcott 1980; Smythe 1962; Townsend *et al.* 1986) led us to evaluate the caudal portion of the thoracic and lumbosacral regions of the spine and the pelvis for stress fractures; and our purpose here is to describe their prevalence and severity in a sample of Thoroughbred racehorses.

Materials and methods

Specimens

A sample of Thoroughbred racehorses that died at a California racetrack and were necropsied through the Davis Branch of the California Veterinary Diagnostic Laboratory System (CVDLS) for the California Horse Racing Board Post Mortem Programme between October 1993 and July 1994 were studied. These same horses were previously evaluated for developmental variations (Haussler *et al.* 1997) and pathological changes (Haussler 1997) in the caudal portion of the thoracic and lumbosacral regions of the spine and the pelvis. During this time a total of 161 Thoroughbred racehorses died at California racetracks and were necropsied. Age, sex (female, sexually intact male, gelding) and reason for euthanasia were recorded for each horse. The lumbosacral spine and pelvis were collected via manual dissection and dissolution in 0.5% potassium hydroxide at 43°C for 4–7 days. The osseous specimens were rinsed in tap water, air dried for 2–3 days and then visually examined. Modified vertebral reference systems were used to allow designation of vertebral segments when the total number of vertebrae in a spinal region was unknown (e.g. the thoracic region in this study) and to facilitate comparison of anatomically related or biomechanically relevant regions between specimens (Haussler *et al.* 1997).

Stress fractures

Specimens were evaluated for the presence of an incomplete fracture line and focal periosteal proliferation, indicative of stress fracture. The location and severity of stress fractures of the

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TABLE 1: Vertebral lamina stress fracture descriptions and locations

Specimen No.	Fracture length (cm)*	Periosteal reaction*	Vertebral location
812	0.7	Mild	Left L(3) lamina
814	0.5/0.5	Mild/Mild	Bilateral L(3) lamina
818	0.5	Absent	Left L(3) lamina
	0.5	Absent	Right L(2) lamina
	1.0	Mild	Left L(1) lamina
	1.2/1.0	Moderate/Mild	Bilateral S1 lamina
819	1.0	Moderate	Left L(2) lamina
837	0.5	Moderate	Right T(1) lamina
839	0.5	Mild	Right L(3) lamina
843	0.1/0.1	Absent/Absent	Bilateral T(1) lamina
	0.5	Absent	Right L(4) lamina
	2.0	Moderate	Left L(1) lamina
850	0.5	Mild	Right L(2) lamina
858	2.0	Absent	Left S1 lamina
866	0.5	Mild	Left L(4) lamina
867	1.0	Moderate	Right L(5) lamina
	0.5	Absent	Left L(4) lamina
883	1.0/1.0	Mild/Mild	Bilateral L(3) lamina
	0.5	Mild	Right S1 lamina
892	0.3/0.3	Mild/Mild	Bilateral T(2) lamina
	1.0	Moderate	Right S1 lamina
919	0.2	Absent	Right L(4) lamina
921	0.2	Mild	Left L(6) lamina
	0.2	Moderate	Left L(4) lamina
	1.0	Severe	Right L(3) lamina
	3.0/3.0	Severe/Severe	Bilateral L(1) lamina
925	0.5	Mild	Left L(3) lamina
926	0.1	Mild	Right L(3) lamina
965	0.5	Mild	Left L(3) lamina

*Left/right descriptions of bilateral vertebral stress fractures.

caudal portion of the thoracic and lumbosacral regions of the spine and the pelvis were recorded. Incomplete fractures were categorised by fracture line length. Mild incomplete fractures were <0.5 cm in length in the vertebrae or <2 cm in length in the pelvic region. Moderate incomplete fractures were 0.6–1 cm (vertebra) or 2–5 cm (pelvis) in length. Severe incomplete fractures were >1 cm (vertebra) or >5 cm (pelvis) in length. Focal periosteal proliferation was categorised by the size and thickness of the callus. Periosteal proliferation was mild if <1 cm^2 and < 1 mm thick; moderate if 1–3 cm^2 and 1–2 mm thick; and severe if >3 cm^2 and >2 mm thick. If the incomplete fracture and focal periosteal proliferation were of differing severity, the stress fracture was classified as the more severe category.

Statistical analysis

Associations were examined between stress fractures and previously reported developmental (Haussler *et al.* 1997) and pathological (Haussler 1997) variables in the same specimens. A 2-tailed Fisher's exact test (FET) was used for categorical variables in 2 x 2 contingency tables with an expected value ≤ 5 in at least one cell. X^2 analysis was used for larger contingency tables. The level for statistical significance was set at $P<0.05$, and for statistical trends $0.05 \leq P \leq 0.10$.

Results

Sample population

Intact lumbosacral spines and pelves were acquired from 36 Thoroughbred racehorses age 2–9 years (mean ± s.d. 4.5 ± 1.5 years); consisting of 12 females, 5 sexually intact males, and 19 geldings. Our sample was derived from 161 racehorses that died and were necropsied during the study period; aged 2–9 years (3.8 ± 1.4 years); consisting of 61 females, 45 sexually intact males, and 55 geldings. No significant differences in age ($X^2_3 = 0.4$, $P = 0.807$) or sex (female or male; $X^2 = 0.6$, $P = 0.430$) were found between the 36 racehorses sampled and the available population. Although, a significant difference was found between our sample and the available population when sex was categorised as female, sexually intact male, or gelding ($X^2_2 = 9.6$, $P = 0.008$). Musculoskeletal related injuries were the cause of euthanasia in 29 (81%) horses, and included spontaneous bone fractures or tendon and/or ligament injuries (spontaneous musculoskeletal injury, 21 horses), trauma (6), and laminitis (2). Nonmusculoskeletal causes of death occurred in the other 7 (19%) horses. No significant differences in the musculoskeletal or nonmusculoskeletal causes of death ($X^2 = 0.3$, $P = 0.858$) were found between the 36 racehorses sampled and the available population.

TABLE 2: Pelvic stress fracture descriptions and locations

Specimen No.	Fracture length (cm)	Periosteal reaction	Pelvic location
791	6.0	Moderate	Left ilium
	1.0	Mild	Right ilium
816	4.0	Severe	Right ilium
819	1.5	Severe	Left ilium
857	1.0	Mild	Left ilium
	3.0	Mild	Right ilium
866	4.5	Mild	Right ilium
892	8.0 (forked)	Severe	Right ilium
919	1.0	Mild	Right ilium
925	2.0	Mild	Left ilium
926	3.0	Mild	Right ilium
927	6.0	Mild	Right ilium

Stress fractures

Twenty-two (61%) specimens had at least one stress fracture in the caudal portion of the thoracic or lumbosacral regions of the spine or the pelvis, characterised by an incomplete fracture and focal periosteal proliferation. Eight (22%) specimens had one stress fracture, 9 (25%) had 2, one (3%) had 3, 2 (6%) had 4, and 2 (6%) specimens had 5 stress fractures. Additionally, 9 (25%) specimens had focal periosteal proliferation without a visible fracture line in the caudal portion of the thoracic or lumbosacral regions of the spine or the pelvis. Six of these 9 specimens had concurrent vertebral or pelvic stress fractures.

Vertebral stress fractures

Eighteen (50%) specimens had a vertebral lamina stress fracture (Fig 1) in a total of 29 vertebrae and 35 vertebral lamina sites (Table 1). Twelve (33%) specimens had one vertebrae affected, 3 (8%) had 2, one (3%) had 3, and 2 (6%) specimens had 4 vertebrae affected. The distribution of affected vertebrae included one at T(2), 2 at T(1), one at L(6), one at L(5), 5 at L(4), 9 at L(3), 3 at L(2), 3 at L(1) and 4 at S1. Vertebral lamina stress fractures were unilateral in 23 vertebrae and bilateral in 6 vertebrae (Fig 2). The left and right vertebral lamina were equally affected (18 left, 17 right). Additionally, focal periosteal

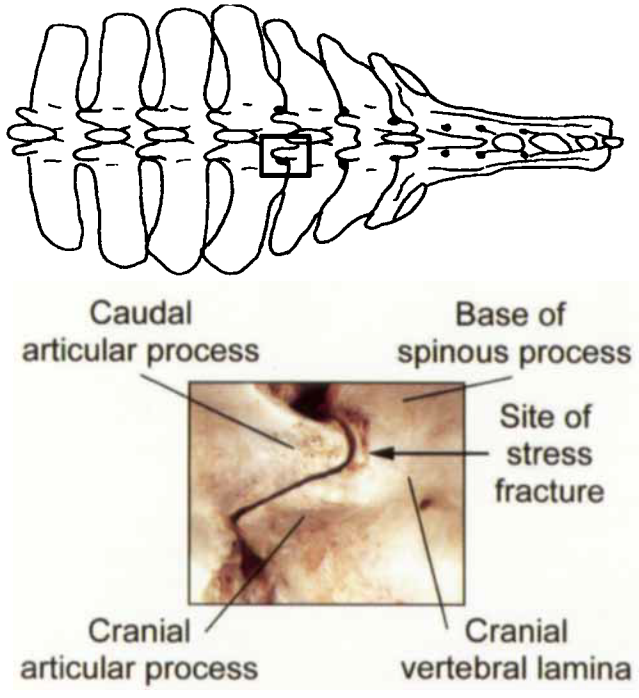

Fig 1: Dorsal view diagram and photographs of articular processes and cranial vertebral lamina illustrating normal structures (labelled) and mild, moderate and severe (top to bottom) incomplete fracture lines extending caudally into the lamina (left column) and focal periosteal proliferation in the same location, affecting the dorsal surface of the lamina (right column). Box on the dorsal view diagram designates region representative of those illustrated in photographs.

proliferation without a visible fracture line was found at the cranial aspect of the vertebral lamina in 6 (17%) specimens, cranial articular process in one (3%), and dorsal aspect of a lumbar transverse process in one (3%) specimen.

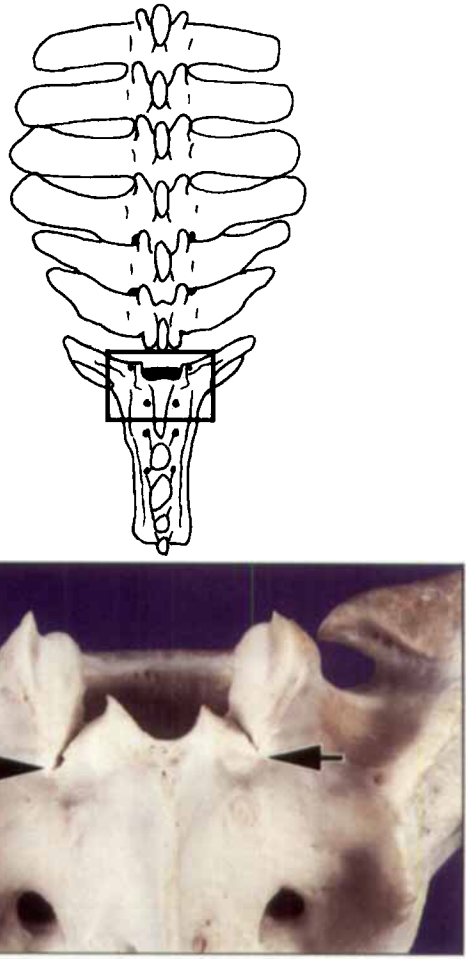

Fig 2: Dorsal view photograph of the sacrum illustrating bilateral vertebral lamina stress fractures (arrows) continuous with articular clefts. Box on the dorsal view diagram represents region illustrated in photograph.

Incomplete fractures of the vertebral lamina occurred consistently at the cranial aspect near the junction of a cranial articular process and the dorsal spinous process in all specimens except one, where the caudal aspect of the vertebral lamina was affected. All vertebral stress fractures were continuous with vertical articular clefts (Haussler *et al.* 1997) of the cranial articular facets (Fig 3), except the caudal vertebral lamina fracture and 2 laminar fractures adjacent ankylosed articular processes. Twenty-seven of 35 (77%) incomplete fractures of the vertebral lamina had surrounding focal periosteal proliferation. Vertebral fracture length (0.8 ± 0.7 cm) was positively associated with the grade of focal periosteal proliferation (X^2_3 = 12.0, P = 0.007). Vertebral stress fractures were graded mild in 16 vertebrae, moderate in 8 and severe in 5 (Fig 1). Bilateral complete fractures of the vertebral lamina were noted in one specimen, so that the dorsal L6 vertebral arch and dorsal spinous process could be removed (Fig 4).

The presence of vertebral stress fractures had a positive trend with age (FET, P = 0.092) (Haussler 1997). Specimens with vertebral stress fractures tended to be from older horses. No association was found between vertebral stress fractures and spontaneous musculoskeletal injuries that resulted in euthanasia (FET, P = 1.000). A trend was noted between vertebral stress

fractures and a diverging L(2)-L(1) interspinous space (FET, P = 0.083). Vertebral stress fractures were not associated with the number of impinged thoracolumbar dorsal spinous processes (FET, P = 0.146); but were positively associated with the overall severity of thoracolumbar dorsal spinous process impingement (X^2_2 = 7.3, P = 0.026).

A positive trend or association was found between vertebral stress fractures and articular process osteophytes (FET, P = 0.060), periarticular erosions (FET, P = 0.095), ankylosis (FET, P = 0.019), and the overall number (FET, P = 0.044) and overall severity of degenerative changes of the articular processes (X^2_2 = 18.4, P = 0.001). Vertebral stress fractures were positively associated with the overall number (FET, P = 0.041), but not with the overall severity of degenerative changes of the intertransverse joints (X^2_2 = 3.2, P = 0.198). A positive trend was noted between vertebral stress fractures and lipping of the ilial articular surface (FET, P = 0.060). Vertebral stress fractures were not associated with the presence of spinal or pelvic enthesophytes.

Pelvic stress fractures

Ten (28%) specimens had a pelvic stress fracture (Fig 5). Pelvic stress fractures occurred in only the left ilial wing in 2 (6%) specimens, only the right ilial wing in 6 (17%), and bilaterally in 2 (6%, Table 2). All pelvic stress fractures originated at the caudal border of the ilium directly over the region of the sacroiliac joint (Fig 6) and coursed in a craniodorsal or craniolateral direction toward the tuber sacrale or tuber coxae. The fracture lines were single, linear defects in all specimens, except for the largest stress fracture which was forked (i.e. Y-shaped) at the cranial aspect of the fracture. Focal periosteal proliferation was present adjacent all pelvic incomplete fractures. However, fracture length (3.4 ± 2.3 cm) was not associated with the amount of focal periosteal proliferation (X^2_2 = 3.1, P = 0.214). Pelvic stress fractures were graded mild in 2 (6%) specimens, moderate in 3 (8%), and severe in 5 (14%, Fig 7). Focal haemorrhagic areas were noted in the periosteum adjacent most moderate and severe, but not mild, pelvic stress fractures. Focal periosteal proliferation without a visible fracture line was found in 2 additional specimens at pelvic sites distant to the sacroiliac articulations (i.e. bilateral iliac crest and caudal branch of pubis).

Pelvic stress fractures were positively associated with age (FET, P = 0.022) but not sex (FET, P = 0.115). Nine of 10 specimens with pelvic stress fractures were 4-years-old or older, however, the largest stress fracture was found in a 3-year-old. Eight geldings, one sexually intact male and one female had pelvic stress fractures. No associations were noted between pelvic stress fractures and spontaneous musculoskeletal injuries (FET, P = 0.716) or any developmental anatomic variations (Haussler 1997). Pelvic stress fractures had a positive association with the severity of impinged lumbar transverse processes (X^2_2 = 7.5, P = 0.024). No associations were noted between pelvic stress fractures and the presence of degenerative changes at the articular processes or intertransverse joints.

A positive association was found between pelvic stress fractures and the overall presence of sacroiliac joint osteophytes (FET, P = 0.001). Pelvic stress fractures were also positively associated with overall severity of ilial articular surface degenerative changes (X^2_2 = 13.5, P = 0.001) and the presence of enthesophytes near the ilial articular surfaces (FET, P = 0.076). Left and right ilial wing stress fractures had a negative trend or association with the presence of ipsilateral left (FET, P = 0.058) and right sacral articular surface lipping (FET, P = 0.013). Positive associations were found between left and right ilial wing stress fractures and ipsilateral left (FET, P = 0.030) and right ilial articular surface osteophytes (FET, P = 0.015). Left and right ilial wing stress fractures were also positively associated with the overall severity of ipsilateral left (X^2_2 = 11.8, P = 0.003) and right ilial articular surface degenerative changes (X^2_2 = 14.4, P = 0.001).

Pelvic stress fractures were not associated with vertebral stress fractures (FET, P = 0.711) although, 6 (17%) specimens had both vertebral and pelvic stress fractures. No association was noted between pelvic stress fractures and ipsilateral or contralateral catastrophic forelimb fractures related to the cause of euthanasia.

Discussion

Vertebral stress fractures

To our knowledge, vertebral stress fractures have not been previously reported in horses. In our necropsy sample of Thoroughbred racehorses, 50% of specimens had incomplete fractures and focal periosteal proliferation of the vertebral lamina, categorised as vertebral stress fractures. The prevalence is probably less in the entire California Thoroughbred racehorse population since 58% of our sample died from probable 'overuse' musculoskeletal injuries of the appendicular skeleton. In these cases, more than one skeletal site, including vertebral sites, could reasonably be affected. However, vertebral stress fractures were not associated with spontaneous musculoskeletal injuries that resulted in euthanasia. Our findings indicate that vertebral stress fractures may be a relatively common entity in Thoroughbred racehorses and it is possible that they may contribute to poor performance and/or back pain of undiagnosed aetiology.

In general, stress fractures are characterised by bone specific predilection sites, an incomplete fracture that may progress to complete fracture, periosteal and/or endosteal callus, tendency for bilaterality, predominance in athletes undergoing strenuous or repetitive activities, and a patient history of periodic recurrent low grade lameness (Stover *et al.* 1993; Pilsworth *et al.* 1994; Shepherd and Pilsworth 1994). Equine vertebral stress fractures characteristically affected the cranial aspect of the lamina near the junction of the vertebral pedicle, cranial articular process and dorsal spinous process. All affected vertebrae had incomplete fractures, except one with complete fractures bilaterally. Periosteal callus was associated with 77% (27 of 35) of incomplete fractures, and was positively associated with fracture length. Six of 29 (21%) affected vertebrae had bilateral stress fractures. The prevalence is high in our necropsy sample of young elite racehorses. Unfortunately the complete clinical histories of our sample of Thoroughbred racehorses are unknown. The consistent location, incomplete fracture line configuration, evidence of periosteal proliferation and probable biomechanical etiology in athletic animals are characteristic of stress fractures.

In man, the last lumbar vertebra (i.e. L[1]) is the most common skeletal location for stress fracture (Yochum and Rowe 1987) with upright posture (i.e. bipedal locomotion) and related high mechanical loads being significant contributors to vertebral stress fracture development and subsequent spondylolysis (Alexander 1985). Spondylolysis is a complete osseous separation at the *pars interarticularis* (i.e. lamina-pedicle

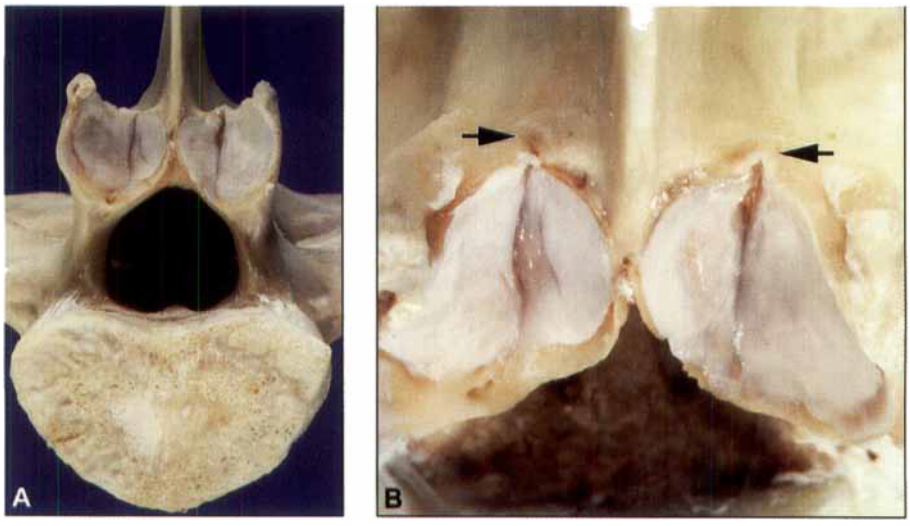

Fig 3: Cranial (A) and close-up (B) view photographs of a lumbar vertebrae illustrating vertical clefts affecting the cranial articular facets. Bilateral vertebral lamina stress fractures (arrows) at the dorsocaudal aspect of the clefts are characterised by an incomplete fracture extending caudally into the lamina and surrounding focal periosteal proliferation.

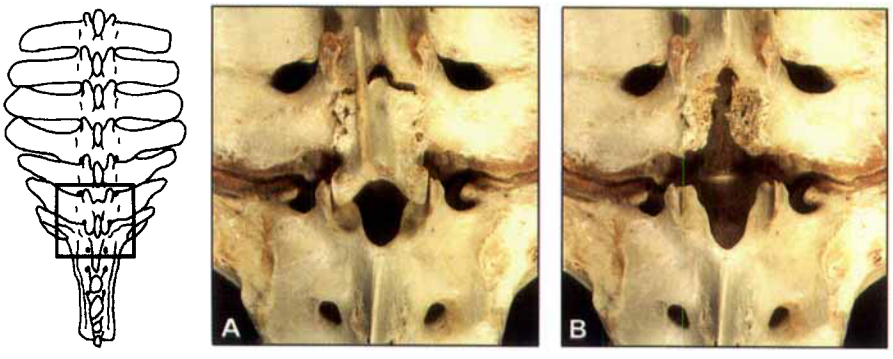

Fig 4: Dorsal view photographs illustrating (A) bilateral complete stress fractures of L(1) which resulted in (B) complete separation of the dorsal neural arch and spinous process from the remaining ventral portion of the affected vertebrae. Box on the dorsal view diagram represents region illustrated in photographs.

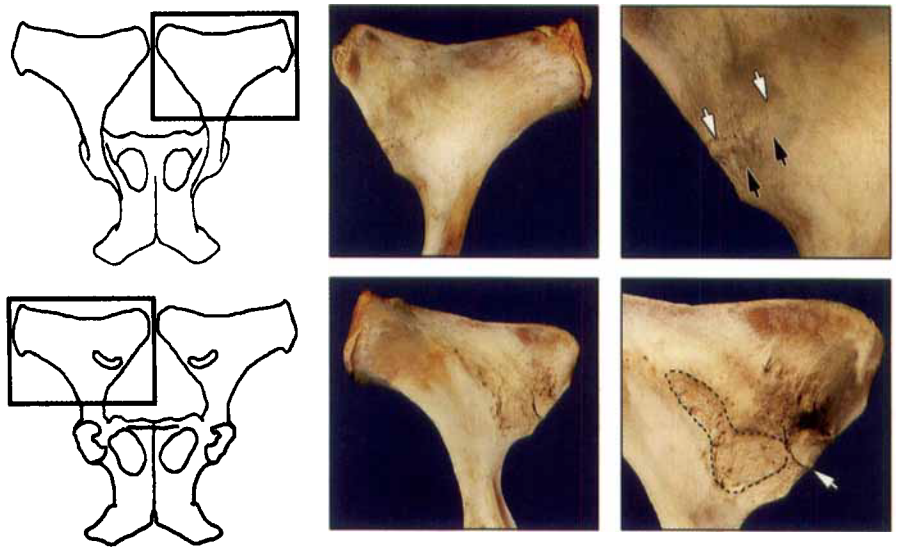

Fig 5: Dorsal (top row) and ventral (bottom row) view photographs of the ilial wing illustrating the typical site of pelvic stress fracture, characterised by incomplete fracture (white arrows) and focal periosteal proliferation (black arrows). Boxes on the dorsal and ventral view diagrams represent regions illustrated in photographs. The ventral view photograph illustrates the spatial relationship of the sacroiliac joint (dotted line) and the incomplete fracture.

junction), which occurs unilaterally or bilaterally. It is hypothesised that spondylolysis results primarily from stress fracture, although congenital and developmental factors have also been implicated in aetiology (Yochum and Rowe 1987). *In vitro* studies of the lumbosacral portion of the human spine, support the role of recurrent mechanical stress in the aetiopathogenesis of spondylolysis, particularly repetitive flexion and extension (Cyron *et al.* 1978; Dietrich and Kurowski 1985; Green *et al.* 1994).

Variations in the prevalence of spondylolysis occur in different human populations (range 2.4–40.3%), average 5–7% in the general population (Yochum and Rowe 1987). Spondylolysis can occur in children shortly after walking begins; presumably due to the development of lumbar lordosis and repetitive mechanical spinal trauma. However, clinical and radiographic detection often does not occur until age 10–15 years. In man, spondylolysis is not usually painful, unless associated with recent spinal trauma (Yochum and Rowe 1987). However, back pain may go unrecognised because of the young age (2–10 years) of the patient and failure to acknowledge the

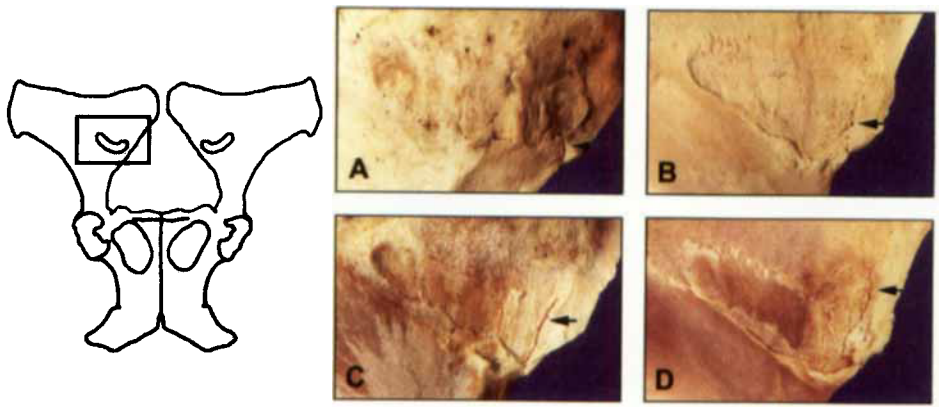

Fig 6: Ventral view photographs of the ilial wing illustrating progression (A-D) of incomplete fractures adjacent the sacroiliac joint. Box on the ventral view diagram represents region illustrated in photographs. The incomplete fracture (arrows) follows the caudomedial joint margin of the sacroiliac joint as the fracture line becomes more severe.

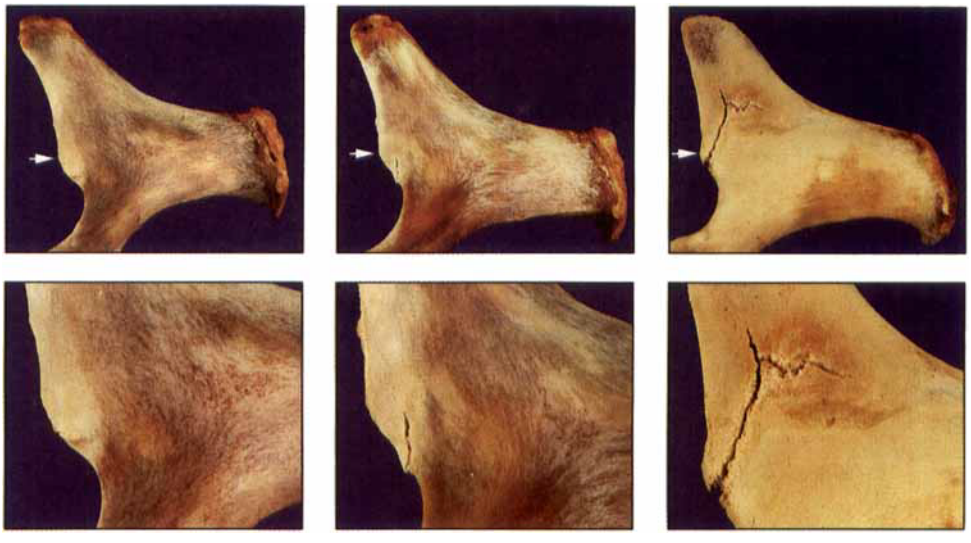

Fig 7: Dorsal (top row) and close-up (bottom row) view photographs of the ilial wing illustrating mild, moderate and severe (left to right) pelvic stress fractures. Note osseous proliferation (arrows) at the caudomedial aspect of the ilial wing, adjacent the pelvic stress fractures.

child's complaints which may be considered 'growing pains'. A similar scenario could occur in equine athletes if veterinarians are not aware of potential spinal pathological changes, including vertebral stress fractures. A 2:1 male predominance of spondylolysis has been reported in man (Yochum and Rowe 1987). We did not find a sex predilection for vertebral stress fractures in our Thoroughbred racehorses.

In young human athletes, repetitive lumbar hyperextension and spinal trauma associated with diving, gymnastics, weightlifting, and pole vaulting have been implicated as aetiological factors in vertebral stress fracture development and subsequent spondylolysis (Yochum and Rowe 1987). The clinical management of young human athletes with symptomatic spondylolysis often includes a restriction or reduction in athletic activities (Blanda *et al.* 1993; Morita *et al.* 1995). However, spondylolysis in adults potentially occurs much earlier in their life, and is usually asymptomatic, if unrelated to a recent history of spinal trauma (Yochum and Rowe 1987). Human subjects often have undiagnosed or untreated spondylolysis, which produces an osseous nonunion that only becomes clinical with excessive mechanical stress. Therefore, prescribed rest and supportive care are best reserved for individuals with biomechanical instability and persistent symptoms. Similar guidelines may be appropriate for equine athletes with vertebral stress fracture and spondylolysis; since the majority of our equine specimens that had incomplete vertebral stress fracture had the potential to progress into complete fractures if excessive mechanical stress or other aetiological factors continued to be present.

Equine vertebral stress fractures, while similar in laminar location to those of man, progress in a caudal direction as opposed to the caudolateral direction (Merbs 1995; Yochum and Rowe 1987). Presumably, anatomical differences in bone morphology and the mechanics of quadrupedal locomotion produce a variation in the vertebral loading environment characteristic of man. In horses, articular processes of the caudal thoracic and lumbar portions of the spine (T16–L6) are deeply interlocking and restrict axial rotation (Townsend and Leach 1984; Townsend *et al.* 1986). Acute articular process fractures in this spinal region are thought to result from excessive axial rotation (Townsend *et al.* 1986). Vertical clefts of the cranial articular facets have been reported in this same spinal region (T16–L6) (Haussler *et al.* 1997). In almost all specimens, incomplete vertebral lamina fractures were continuous with these articular clefts. The articular clefts may provide a site for stress concentration in the aetiopathogenesis of equine vertebral stress fractures. Equine articular process morphology and articular clefts may also contribute to the development of multi-level vertebral stress fractures found in 6 of 18 affected specimens, compared to single vertebral levels commonly reported in man (Yochum and Rowe 1987). Incomplete fracture progression would result in complete laminar fracture, and unilateral fractures would have a predilection for developing bilateral laminar fractures, as noted in one equine specimen. This is consistent with vertebral stress fractures and spondylolysis, as seen in man.

Pelvic stress fractures

Pelvic stress fractures have been previously reported in Thoroughbred racehorses (Stover *et al.* 1993; Pilsworth *et al.* 1994; Shepherd *et al.* 1994) and associated complete fractures occur spontaneously in training without history of acute trauma (Jeffcott 1982; Rutkowski and Richardson 1989; Pilsworth *et al.* 1994; Shepherd *et al.* 1994). They occur unilaterally and bilaterally and may progress to complete fracture (Hornof *et al.* 1996). Whereas the majority of fractures were unilateral, 2 of our specimens had bilateral fractures. Right sided ilial wing stress fractures were 3 times more frequent than left sided fractures in our specimens. However, a side prevalence has not been apparent from other reports (Shepherd *et al.* 1994) and our numbers are too low to draw any conclusions other than continued observation.

In contrast to previous reports where complete pelvic fractures occurred predominantly in 4-year-old or younger and female horses (Heinze and Lewis 1971; Jeffcott 1982; Little and Hilbert 1987; Rutkowski and Richardson 1989), pelvic stress fractures in our specimens occurred predominantly in older geldings. Our sample of Thoroughbred racehorses did contain proportionally more geldings and fewer sexually intact males than the available population, but females were equally represented. The pelvic morphology in female horses compared to male horses provides larger lever arms for muscles that might generate larger muscle moments and precipitate complete fracture when an incomplete fracture is present. Therefore, male horses may have a higher tolerance for pelvic stress fracture, whereas female horses would be more likely to propagate a complete fracture from a stress fracture. Reasons for horse age differences between studies is not readily apparent.

Ultrasonography, scintigraphy and radiography are utilised for *antemortem* diagnosis of pelvic stress fractures (Pilsworth *et al.* 1994; Shepherd and Pilsworth 1994; Shepherd *et al.* 1994; Hornof *et al.* 1996). Nine of 20 horses with a pelvic incomplete or complete fracture had ultrasonographic evidence of roughened and irregular bone contour adjacent the fracture line, suggestive of periosteal callus formation (Shepherd *et al.* 1994). In a *post mortem* study, the pelvis of all 8 racehorses with complete pelvic fracture had periosteal callus adjacent a portion of the complete fracture line, indicative of a pre-existing stress fracture (Stover *et al.* 1993). All pelvic stress fractures in our specimens had periosteal callus in association with the incomplete fracture.

The pelvic stress fractures in this study were consistent in location with previous reports (Stover *et al.* 1993; Shepherd and Pilsworth 1994; Hornof *et al.* 1996). All fractures were located on the caudal border of the ilium adjacent the caudomedial aspect of the sacroiliac joint. However, one specimen with bilateral pelvic stress fractures also had focal periosteal proliferation without a grossly visible fracture line in the less commonly reported location on the pubis (Stover *et al.* 1993). Pelvic stress fractures progressed in size and length craniolaterally. The longest fracture forked at the cranial aspect. Fracture configuration and proximity to the sacroiliac joint have possible aetiopathogenic 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 (Hornof *et al.* 1996).

The pattern of fracture line propagation in the ilial wing may be related to the sacrum and the sacroiliac joint's influence on bone strain. Pelvic fractures that originate within the ilial wing, either dorsal or ventral to the sacroiliac joint, appear to propagate perpendicular (i.e. craniolaterally) to the caudomedial border of the ilium. However, in some specimens, fractures originating directly adjacent the sacroiliac joint appear to propagate perpendicular to the ilial border until the sacroiliac joint margin is reached. The fracture line then is directed craniodorsally along the sacroiliac joint margin, parallel to the caudomedial border of the ilium. When the fracture line progresses beyond the dorsal sacroiliac joint margin, it then appears to be redirected again craniolaterally, perpendicular to the caudomedial ilial border.

It is probable that pelvic stress fractures affect performance during some portion of their development and are one cause of undiagnosed poor performance and/or lameness (Pilsworth *et al.* 1994; Shepherd *et al.* 1994). Stress fractures in long bone counterparts (e.g. metacarpus), when allowed to heal, usually do so without permanent sequella (i.e. persistent lameness) because bone tissue is capable of regeneration and remodelling. Therefore, the culmination of bone repair is a structure that is, for the most part, indistinguishable morphologically and functionally from the parent structure. However, in physiological circumstances, articular cartilage does not exhibit regeneration. The proximity of pelvic stress fractures to the sacroiliac joint may be related to sacroiliac joint degeneration (Shepherd *et al.* 1994). This is supported by the positive association or trend between pelvic stress fractures and the severity of degenerative changes of the ilial articular surface and the overall severity of sacroiliac joint degenerative changes.

In conclusion, stress fractures of the vertebrae and/or pelvis were present in 61% of our necropsy sample of Thoroughbred racehorses. Vertebral stress fractures, which have not been previously reported in horses, were found in 50% of specimens. Positive associations were found between vertebral stress fractures and the severity of dorsal spinous process impingement and the overall severity of vertebral articular process degenerative changes. Pelvic stress fractures were found in 28% of specimens, particularly specimens from older geldings. Positive associations were found between pelvic stress fractures and the severity of lumbar transverse process impingement and the overall severity of ilial articular surface degenerative changes. Whereas the clinical significance of these findings is unknown, it is probable that vertebral and pelvic stress fractures may be a cause of lameness and/or poor performance during at least some portions of their development.

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