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Objective—To determine clinical characteristics of and outcome in Thoroughbred racehorses with tibial or humeral stress fractures.

Design—Retrospective study.

Animals—99 Thoroughbreds with tibial or humeral stress fractures.

Procedure—Information obtained from the medical records included history, signalment, and clinical, radiographic, and scintigraphic findings. Outcome was determined by interviewing trainers, performing follow-up examinations, and analyzing race records.

Results—Seventy-four tibial stress fractures were identified in 61 Thoroughbreds, and 48 humeral stress fractures were identified in 39 Thoroughbreds (1 horse was included in both groups). Tibial stress fractures occurred most commonly in 2-year-old or unraced horses. Fractures were located in 1 of 3 sites in the tibia (most commonly, the caudolateral cortex of the mid-diaphysis) and 1 of 4 sites in the humerus (most commonly, the caudodistal cortex). Forty-four of 58 (76%) tibial stress fractures and 18 of 32 (56%) humeral stress fractures were identified radiographically. Humeral stress fractures involving the caudodistal cortex were not detected radiographically. Treatment consisted of rest and exercise restriction, and 49 of 61 (80%) horses with tibial stress fractures and 30 of 39 (77%) horses with humeral stress fractures returned to racing. Humeral stress fractures recurred in 6 horses.

Conclusions and Clinical Relevance—Results suggested that in Thoroughbred racehorses, tibial stress fractures occurred most commonly in unraced 2-year-olds, whereas humeral stress fractures occurred most commonly in older horses that had raced previously. The prognosis for racing following treatment was good. (J Am Vet Med Assoc 2003;222:491–498)

Stress (fatigue) fractures of the third metacarpal bone, tibia, humerus, radius, and pelvis are well-recognized causes of lameness in racehorses. However, diagnosis of stress fractures of the bones that make up the upper portions of the limbs poses a particular clinical challenge, as the site of pain is rarely amenable to identification through palpation or regional anesthesia, and radiographic examination is limited, typically resulting in inconclusive findings. Fine-detail radiography may demonstrate stress fractures and their associated callus, but radiographic evidence of callus does not necessarily correlate with active bone remodeling or signs of pain. These reasons, combined with the high sensitivity of nuclear scintigraphy in the detection of increased bone metabolic activity, suggest that nuclear scintigraphy is the imaging modality of choice for the diagnosis of stress fractures of the bones of the upper portions of the limbs.

The use of nuclear scintigraphy has resulted in identification of tibial and humeral stress fractures in substantial numbers of Thoroughbred racehorses. Furthermore, stress and incomplete fractures of the tibia and humerus have been implicated as precursors to complete catastrophic fractures. Early and accurate diagnosis of tibial and humeral stress fractures is, therefore, highly desirable. However, despite the relatively high incidence and clinical importance of tibial and humeral stress fractures, there is limited published information on clinical, radiographic, and scintigraphic findings in and outcome of affected racehorses. Therefore, the purpose of the study reported here was to determine clinical characteristics and outcome in a large group of Thoroughbred racehorses with tibial or humeral stress fractures.

Criteria for Selection of Cases

Medical records of all Thoroughbred racehorses examined at the Randwick Equine Centre between January 1992 and March 2000 because of a stress fracture of the tibia or humerus were reviewed. Horses were included in the study if a diagnosis of stress fracture had been made on the basis of history and results of clinical and lameness examinations and nuclear scintigraphy. Radiography of the fracture site was not a criterion for inclusion in the study.

Procedures

Data collection—Information obtained from the medical records included history, breed, age, sex, and clinical, scintigraphic, and radiographic findings. Lameness examination findings included affected limb, severity of lameness as graded on a scale from 1 to 5, and response to manipulative tests and diagnostic anesthesia.

Bone-phase nuclear scintigraphic images were obtained with a gamma camera and integrated nuclear imaging system 3 hours after IV administration of 7

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GBq of technetium Tc 99m oxidronate. A diagnosis of a stress fracture was made if a focal area of intense increased radiopharmaceutical uptake (IRU) was seen in the cortex of the tibia or humerus. Areas of mild IRU consistent with exercise-induced bone remodeling were not considered to be stress fractures; these areas were alternatively termed stress reactions.8,14

Radiography was typically performed after scintigraphic identification of the lesion; in this instance, only the most appropriate radiographic projections were obtained. If radiography was performed prior to scintigraphy, 4 standard projections of the tibia were obtained: a caudoproximal-craniodistal oblique projection made at 10° proximal to the supporting surface (Ca10Pr–CrDiO projection), a craniodistal-lateral-medial oblique projection made at 45° lateral to the dorsoplantar line (Ca45L–CrMO projection), a craniodistal-lateral-medial oblique projection made at 45° lateral to the dorsoplantar line (Cr45L–CaMO projection), and a lateromedial (LM) projection. Radiographic projections of the humerus consisted of a flexed mediolateral (ML) projection of the shoulder and proximal portion of the humerus or a flexed ML projection of the distal portion of the humerus and the elbow. Radiographic evidence of a focal periosteal and endosteal reaction or an intracortical fracture line corresponding with the site identified scintigraphically was considered diagnostic of a stress fracture.

Outcome was determined by interviewing trainers of affected horses, performing follow-up examinations, and analyzing race records supplied by the Australian Jockey Club. Information obtained from the race records included time to return to racing after fracture and number of starts and earnings per start before and after the stress fracture was identified.

Data analysis—Summary statistics were calculated. A χ² test was used to compare proportions of males and females in the tibial and humeral stress fracture groups with proportions of males and females in an age-matched sample of the Australian racing population. A χ²-test of proportions with the Yates correction was used to determine, for horses with tibial stress fractures and for horses with humeral stress fractures, whether proportions of left and right limbs affected were similar. Kruskal-Wallis 1-way ANOVA on ranks was used to examine whether severity of lameness was associated with fracture site. For horses with humeral stress fractures, χ² analysis was used to compare the proportions of cranial versus caudal cortex fractures.

A Wilcoxon sign rank test was used to compare earnings per start before and after fracture for horses that raced before and after fracture within each group. Mann-Whitney U tests were used to compare age and number of starts prior to fracture between horses with tibial fractures and horses with humeral fractures. Proportions of horses with fracture recurrence were compared between groups with a Fisher exact test. For all analyses, values of P < 0.05 were considered significant.

Results

Ninety-nine Thoroughbred racehorses with a total of 122 stress fractures of the tibia or humerus were eligible for inclusion in the study. One horse was examined on separate occasions for stress fractures of the tibia and humerus. All horses trained and raced in a clockwise direction, and all horses raced exclusively on turf. Horses were trained on a variety of surfaces including turf, dirt, and sand. During the period of this study, 711 nuclear scintigraphic imaging studies were performed at Randwick Equine Centre on Thoroughbred horses in race training with musculoskeletal problems.

Horses with tibial stress fractures—Sixty-one of the horses included in the study had tibial stress fractures. Ten horses had simultaneous bilateral fractures, and 1 horse had 3 stress fractures at separate sites in the same tibia. One horse was examined twice—once because of a tibial stress fracture of 1 limb and again at a later date because of a tibial stress fracture involving the contralateral limb. The 2 fractures in this horse were considered separate cases of tibial stress fracture; therefore, each episode of tibial stress fracture in this horse was considered a separate case, and clinical data were analyzed for 62 cases of tibial stress fracture.

Therefore, a total of 74 tibial stress fractures were identified. Horses with tibial stress fractures represented 9% of the total number of Thoroughbred racehorses undergoing scintigraphy for a musculoskeletal problem during the period of the study.

Affected cases included 44 (71%) 2 year olds, 14 (22%) 3 year olds, 2 (3%) 4 year olds, 1 (2%) 5 year old, and 1 (2%) 6 year old. Thirty were females, and 32 were males, of which 18 had been castrated. This sex distribution was not significantly (P = 0.12) different from sex distribution of an age-matched sample of the Australian racing population. Forty-three of 61 (70%) horses had not raced prior to diagnosis of a tibial stress fracture. All horses were examined because of lameness; at the time of initial examination, 3 were classified as grade-1 lame, 21 were classified as grade-2 lame, 33 were classified as grade-3 lame, and 4 were classified as grade-4 lame. Results of an upper limb flexion (spavin) test were recorded for 46 cases and were positive (ie, lameness was exacerbated) in 35 (76%). Only 8 of 27 (30%) cases had a positive response to firm palpation and percussion of the medial diaphysis of the tibia.

Diagnostic anesthesia of sites remote from the stress fracture site was performed as part of the lameness examination in 33 (57%) cases, all of which had lameness less severe than grade-4 at the time of initial examination. Severity of the lameness did not improve in any of these cases after diagnostic anesthesia, and in many cases, the lameness worsened with trotting.

Scintigraphic and radiographic findings in horses with tibial stress fractures—Of the 74 tibial stress fractures identified, 34 (46%) involved the left hind limb, and 40 (54%) involved the right hind limb. For horses with a single tibial stress fracture, the proportion of fractures involving the left hind limb was not significantly (P = 0.48) different from the proportion involving the right hind limb. All fractures identified were confined to 3 distinct locations, with 8 fractures (11%) involving the proximal third of the bone, 57 fractures (77%) involving the middle third (Fig 1), and 9 fractures (12%) involv-
ing the distal third. All of the proximal and mid-diaphyseal fractures were located in the caudolateral aspect of the cortex and were apparent scintigraphically as a focal area of intense IRU. Scintigraphic findings for the 9 distal fractures consisted of a more diffuse area of intense IRU that extended beyond the caudal cortex. Areas of mild local IRU that were defined as stress reactions rather than stress fractures were seen in 8 of 62 (13%) cases, 4 in the same limb as the fracture and 4 in the contralateral limb (Fig 2). These stress reactions were seen to occur at the locations identified as being predisposed to tibial stress fracture formation.

Fifty-six of the 71 (79%) tibias with stress fractures were radiographed; this represented 58 of the 74 fracture sites. Forty-four of the 58 (76%) fracture sites had radiographic evidence of a stress fracture. Radiographic findings consisted of a callus and fracture line in 17 sites (39%), a callus alone in 25 (57%) sites, and a fracture line alone in 2 sites (4%). Radiographic evidence of a stress fracture was seen for 6 of the 7 (86%) proximal fractures that were radiographed, 36 of the 45 (80%) mid-diaphyseal fractures that were radiographed, and 2 of the 6 (33%) distal fractures that were radiographed. For proximal and mid-diaphyseal fractures, the callus and fracture line were best seen on the Cr45L–CaMO projection (Fig 3). Likelihoods of radiographically identifying proximal and mid-diaphyseal fractures were significantly \( P = 0.03 \) greater than the likelihood of radiographically identifying a distal fracture.

Median lameness grades in horses with proximal, mid-diaphyseal, and distal fractures were 2.5 (range, 2 to 4), 3 (range, 1 to 4), and 3 (range, 2 to 3), respectively. There was no significant \( P = 0.427 \) association between fracture site and severity of lameness.

**Treatment of horses with tibial stress fractures**—For all horses with tibial stress fractures, treatment consisted of exercise restriction for 3 to 4
months. This consisted of a minimum of 4 weeks of stall rest, with limited hand walking commencing when the horse was no longer lame at the walk. If the horse was no longer lame at a trot in a straight line after 4 weeks of limited hand walking, access to a small yard or walkout area (approx 20 X 20 ft) was allowed for a further 4 weeks. If the horse was still lame, additional stall rest until no longer lame at the trot was advised prior to access to small yard or walkout area. After the exercise restriction period, they were allowed access to pasture turnout for 4 to 8 weeks before training was resumed.

Outcome of horses with tibial stress fractures—Follow-up time ranged from 0.6 to 9 years (mean, 3.7 years). None of the horses with tibial stress fractures in this study developed complete fractures during the follow-up period. Race records were available from the Australian Jockey Club for 59 horses. The remaining 2 horses raced in Malaysia (twice) and Singapore (once) after diagnosis and treatment for tibial stress fractures. These 2 horses were included in analyses of the percentage of horses that returned to racing but were excluded from analyses of other race results. In addition, Australian Jockey Club records for 1 horse were archived, and earnings per start before and after tibial stress fracture could not be calculated.

Eighteen of the 61 (30%) horses raced prior to fracture (median number of starts, 2; range, 1 to 43), and 49 (80%) raced after fracture (median number of starts, 8; range, 1 to 53). Thirty-nine horses had ≥ 3 starts after treatment for a tibial stress fracture. Median time to return to racing after fracture diagnosis could be calculated for 47 horses and was 7 months (range, 5 to 18 months).

The horse that was examined on 2 occasions because of separate tibial stress fractures had not raced prior to diagnosis of the first fracture. This horse raced once after treatment for the first fracture and prior to identification of the second fracture and raced 17 times after treatment for the second fracture.

Of the 12 horses that did not race after treatment of a tibial stress fracture, 7 were voluntarily retired to stud or because of a lack of ability, 3 were retired for unrelated reasons (superficial digital flexor tendonitis in 2 and left laryngeal hemiplegia in 1), and 2 were lost to follow-up.

Comparison of race records before and after fracture was possible for 12 horses, excluding the horse that developed a second tibial stress fracture. No significant (P = 0.733) difference was found between mean earnings per start before fracture and mean earnings per start after fracture. For these horses, median numbers of starts before and after fracture were 1 (range, 1 to 43) and 10 (range, 3 to 42), respectively. Mean earnings per start before and after fracture were $8,692 (range, $0 to $71,725) and $5,310 (range, $0 to $39,000), respectively. Seven of these 12 horses had increased mean earnings per start after treatment for tibial stress fracture, and 5 had decreased earnings per start.

Horses with humeral stress fractures—Thirty-nine of the horses included in the study had humeral stress fractures. Two horses had simultaneous bilateral fractures, and 6 horses had a second fracture after treatment for the first fracture (at a corresponding site in the contralateral humerus in 5 horses and at a different site in the same humerus in 1 horse). In addition, 1 of these 6 horses had a third humeral stress fracture after treatment for the first and second fractures. Second fractures in 5 horses and second and third fractures in 1 horse were treated as separate events. Therefore, data were analyzed for 48 humeral stress fractures in 46 cases (including 1 horse that had previously had a tibial stress fracture). This represented 6% of the total number of Thoroughbred racehorses undergoing scintigraphy for a musculoskeletal problem during the period of the study.

Affected cases included 1 (2%) 1 year old, 13 (28%) 2 year olds, 21 (46%) 3 year olds, 9 (20%) 4 year olds, 1 (2%) 5 year old, and 1 (2%) 6 year old. Twenty-two were females, and 26 were males, of which 23 had been castrated. This sex distribution was not significantly different from sex distribution of an age-matched sample of the Australian racing population. Nineteen of 39 (49%) horses had not raced prior to diagnosis of a humeral stress fracture.

All cases were examined because of lameness. Severity of lameness at the time of initial examination was reported for 40 cases and was grade-1 in 5 cases, grade-2 in 12 cases, grade-3 in 15 cases, and grade-4 in 8 cases. Many of the cases had a history of more severe lameness (grade-4) soon after exercise that had improved within 24 hours. Cases with the most severe lameness typically had an obviously shortened cranial phase of the stride at the walk and trot. Results of manipulative tests of the upper portion of the forelimb were reported in the records of 10 cases. Six had signs of pain and exacerbation of the lameness after manipulation of the elbow and shoulder joints, including flexion, adduction, and abduction of the upper portion of the limb. Diagnostic anesthesia of sites remote from the stress fracture site was performed as part of the lameness examination in 26 cases, all of which had lameness less severe than grade 4 at the time of initial examination. Severity of the lameness did not improve in any of these cases after diagnostic anesthesia, and in many cases, the lameness worsened with trotting.

Scintigraphic and radiographic findings in horses with humeral stress fractures—Of the 48 humeral stress fractures identified, 19 (40%) involved the right forelimb, and 29 (60%) involved the left forelimb. For horses with a single humeral stress fracture, the proportion of fractures involving the left forelimb was not significantly different from the proportion involving the right forelimb. All fractures identified were confined to 4 distinct locations, with 18 (37%) involving the caudoproximal cortex of the humerus, 2 (4%) involving the cranioproximal cortex, 20 (42%) involving the caudodistal cortex, and 8 (17%) involving the craniodistal cortex (Fig 4). There was a significant (P < 0.001) difference in proportions of fractures involving the caudal and cranial humeral cortices, with the caudal cortex affected in 79% of fractures. Areas of IRU other than fracture sites were not identified in any of the horses.
In the 6 horses in which fractures occurred in the contralateral limb at a later date, 4 fractures involved the caudodistal cortex. One horse developed 3 fractures on separate occasions—the first was in the caudodistal cortex of the left humerus, the second was in the caudodistal cortex of the right humerus, and the third was at the original fracture site in the left humerus. The initial left humeral stress fracture had been considered completely healed on the basis of results of follow-up scintigraphy performed at the time of diagnosis of the right humeral fracture. The third fracture occurred 2 years and 26 race starts after the first fracture had been diagnosed.

Thirty-two of the 48 (67%) humeral stress fractures were radiographed, and radiographic evidence of a stress fracture was seen in 18 (56%). Radiographic findings consisted of a callus and fracture line in 5 (28%) sites, a callus alone in 12 (67%) sites, and a fracture line alone at 1 (5%) site. Radiographic evidence of a stress fracture was seen for 11 of the 16 caudoproximal fractures that were radiographed, both of the cranioproximal fractures that were radiographed, none of the 7 caudodistal fractures that were radiographed, and 5 of the 7 craniodistal fractures that were radiographed.

Median lameness grades in cases with cranioproximal, caudoproximal, and caudodistal fractures were 3, and median grade for cases with craniodistal fractures was 2. There was no significant (P = 0.152) association between fracture site and severity of lameness.

Treatment of horses with humeral stress fractures—For all horses with humeral stress fractures, treatment consisted of exercise restriction for 3 to 4 months. Exercise restriction was as described for horses with tibial stress fractures.

Outcome of horses with humeral stress fractures—Follow-up time ranged from 0.3 to 8.1 years (mean, 3.3 years). None of the horses with humeral stress fractures in this study developed complete fractures during the follow-up period. However, 1 horse suffered a complete fracture of the contralateral humerus while in race training after rehabilitation. Race records were available for 28 horses that raced in Australia. The other 2 horses that raced after treatment of a humeral stress fracture did so in New Zealand and the United States, and detailed race records were not obtained for these horses.

Twenty of the 39 (51%) horses raced prior to fracture (median number of starts, 5; range, 1 to 40), and 30 of 39 (77%) raced after fracture (median number of starts, 8.5; range, 1 to 33). Twenty-four horses had ≥3 starts after fracture. Median time to return to racing after fracture diagnosis could be calculated for 28 horses and was 7.5 months (range, 5 to 22 months). Of the 9 horses that did not race after treatment of a humeral stress fracture, 5 were voluntarily retired to stud or because of a lack of ability, 1 was retired for an unrelated reason (superficial flexor tendonitis), 1 was euthanized after suffering a complete fracture of the contralateral humerus after returning to training, and 2 were lost to follow-up.

Comparison of race records before and after fracture was possible for 16 horses, excluding the horses that developed a second or third humeral stress fracture. No significant (P = 0.632) difference was found between mean earnings per start before fracture and mean earnings per start after fracture. For these horses, median numbers of starts before and after fracture were 5 (range, 1 to 40) and 10.5 (range, 1 to 33), respectively. Mean earnings per start before and after fracture were $7,887 (range, $0 to $50,050) and $3,717 (range, $0 to $7,250), respectively. Eight of these 16 horses had increased earnings per start after treatment for humeral stress fracture, and 8 had decreased earnings per start.

Comparison of tibial and humeral stress fractures—Horses with humeral stress fractures had a significantly (P = 0.01) higher prevalence of fracture recurrence (6/39; 15%) than did horses with tibial stress fractures (1/61; 2%). For all but 1 horse with a humeral stress fracture, the recurrence involved the corresponding site in the contralateral limb. Similarly, bilateral fractures included corresponding sites in the 2 horses with bilateral humeral fractures and in 8 of the 10 horses with bilateral tibial stress fractures.

Median age of humeral stress fracture cases (3 years) was significantly (P < 0.001) greater than median age of cases with a tibial stress fracture (2 years). Horses with a humeral stress fracture had a significantly (P = 0.04) greater number of starts prior to fracture identification than did horses with a tibial stress fracture.

Discussion
In the present study, tibial stress fractures were more commonly identified than humeral stress fractures in Thoroughbred racehorses. Similar findings were reported in a review of the results of scintigraphic imaging.
ic examination of the musculoskeletal system in Thoroughbred racehorses and a study\(^5\) of the incidence of fractures in Thoroughbred racehorses. Furthermore, greater numbers of tibial stress fractures have been reported in the literature.\(^1,6,7\) Therefore, it appears that the tibia may be more susceptible to stress fracture than the humerus in racing Thoroughbred horses.

Tibial stress fractures were seen predominately (71%) in 2-year-old horses in the present study, whereas humeral stress fractures were seen more commonly (46%) in 3-year-old horses. These findings are in agreement with those of previous studies.\(^1,6,7\) In addition, tibial stress fractures were seen predominantly (70%) in unraced or lightly raced horses, as indicated by the median of 2 race starts prior to fracture diagnosis. In comparison, humeral stress fractures were seen predominantly in horses that were more extensively raced, as indicated by the median of 5 race starts prior to diagnosis. Significant differences in age and number of starts were found between horses with tibial stress fractures and horses with humeral stress fractures. Since both age and number of starts were confounding factors, it is difficult to determine which is the contributing factor. However, it is clear that Thoroughbred racehorses develop tibial stress fractures earlier in their careers, compared with humeral stress fractures, which appear more commonly in slightly older horses that have raced previously. No sex predilection was demonstrated in either the tibial or humeral stress fracture group in this study, which is consistent with results of previous studies.\(^1,2,7\)

Horses with stress fractures of the tibia were typically moderately lame, with a median lameness grade of 3. Thirty-five of 46 (76%) horses with a tibial stress fracture had a positive response to an upper limb flexion (spavin) test. In comparison, although palpation and percussion of the tibia have been suggested as being useful in the diagnosis of tibial stress fractures,\(^4,8\) in the present study, only 8 of 27 (30%) horses had a positive response. The low sensitivity of these tests is likely attributable to an inability to directly palpate or percuss the most common site of fracture in the caudolateral cortex because of overlying musculature. Horses with humeral stress fractures were also moderately lame, with a median lameness grade of 3. Elbow and shoulder joint manipulation (flexion, adduction, and abduction of the upper portion of the limb) exacerbated the lameness in 6 of 10 horses in which it was performed. These findings suggest that these manipulations may be useful diagnostic tests in the examination of horses suspected to have tibial or humeral stress fractures.

A previous study\(^7\) suggested a possible limb predilection among horses with humeral stress fractures; however, no evidence of a limb predilection was found for horses with tibial or humeral stress fracture in the present study. Simultaneous bilateral fractures were seen infrequently in horses with humeral stress fractures (2/39; 5%) but were more common in horses with tibial stress fractures (10/61; 16%), which is consistent with findings of previous studies.\(^4,6,7\)

All of the tibial stress fractures located in the proximal or middle third of the tibia involved the caudolateral tibial cortex. Similarly, other studies\(^6,7\) have reported the caudolateral cortex to be most commonly affected. Scintigraphic findings for horses with proximal and mid-diaphyseal tibial stress fractures consisted of focal intense IRU, whereas fractures involving the distal third of the tibia had a more diffuse pattern of intense IRU that was not consistently localized to any specific part of the cortex. Previous studies\(^6\) have identified these distal stress fractures in a variety of sites, including the cranial and caudomedial cortices of the tibia.

Typically a focal cortical area of intense IRU is associated with an acute stress fracture.\(^7\) However, in 8 of 62 (13%) horses with tibial stress fractures in the present study, a focal area of mild IRU was seen in the affected or contralateral tibia, in addition to the fracture site. This increased metabolic bone activity was interpreted as a form of exercise-induced (adaptive) bone remodeling or stress reaction.\(^4,6,7\)

The most common fracture location for horses with tibial stress fractures in the present study was the mid-diaphysis, which is in agreement with results of previous studies\(^6\) in Thoroughbreds and Standardbreds. On the other hand, others have reported the proximal portion of the diaphysis as being the most common site of tibial stress fracture in Thoroughbred racehorses.\(^1,4,7\)

Previously, it has been suggested the difference in frequency between these 2 sites may be related to breed and racing gait, with Standardbred racehorses more likely to have fractures of the caudal aspect of the mid-diaphysis.\(^7\) Results of our study and of a previous's suggest that breed and racing gait do not appear to influence the site of tibial stress fractures.

In vivo measurements of strain in the tibia have documented that, at a walk, tensile strain develops in the craniolateral aspect of the cortex in the middle and proximal portions of the tibia but that strain becomes more torsional distally.\(^8,9\) It is likely that similar strain characteristics are present at faster gaits and are associated with compressive strains in the caudal aspect of the cortex in the proximal and middle portions of the tibia. The repeatable distribution of fractures localized to the caudolateral cortex in the middle and proximal portions of the tibia is likely attributable to stress concentration in this area of the cortex. In comparison, during faster exercise, the distal portion of the tibia may experience torsional strains, allowing fractures in the distal portion of the tibia to develop without a specific cortical distribution.

Humeral stress fractures were identified in 4 sites in the present study, and fracture of the cranioproximal cortex has, to our knowledge, not been reported previously. The caudal cortex was most commonly affected, with distal fractures slightly more common than proximal fractures. Others report the caudoproximal cortex as the most frequently affected site.\(^1,2\) The different fracture distribution in the present study may be a consequence of differences in training regimens or track surfaces or a reflection of the small sample sizes in some previous studies. To the authors’ knowledge, no studies of in vivo humeral stress fractures have been performed in horses.

No association was found between the severity of lameness and the fracture site in horses with tibial or
Humeral stress fractures were less reliably identified radiographically (18/32; 56%). Fractures in the caudoproximal, cranioproximal, and craniodistal cortex were radiographically demonstrable, whereas none of the 7 fractures of the caudodistal cortex were seen radiographically. It is likely that the site of IRU in the caudodistal cortex corresponded to fractures in the proximal aspect of the epicondyles or the olecranon fossa. This would explain why radiographic demonstration of a stress fracture was difficult on the standard flexed ML projection. Other projections, particularly oblique projections, may aid in identification of the site of these caudodistal fractures.

In this study, horses with either tibial or humeral stress fractures were treated successfully with rest and restricted exercise. Time from diagnosis to first racing start was similar for horses with tibial and humeral stress fractures and comparable to time in a previous report. Results of this study support the use of this exercise restriction program, which facilitated uncomplicated healing of both tibial and humeral stress fractures and allowed a return to training after approximately 12 weeks without propagation to complete fracture.

Recurrence of tibial stress fractures was not common in the present study, and a similar low recurrence rate has been reported previously. This low recurrence rate indicates that an adequate fracture and tibial remodeling response sufficient to withstand repetitive high speed exercise occurred. Conversely, humeral stress fractures recurred significantly more commonly than tibial stress fractures. Recurrent humeral stress fractures were typically seen at the corresponding site in the contralateral limb. None of the horses with a recurrent fracture had bilateral fractures or evidence of increased bone activity at the subsequent fracture site at the time of examination for the initial fracture.

To our knowledge, this relatively high incidence of recurrent humeral stress fractures has not been reported previously, and it appears that horses with a humeral stress fracture have a greater risk of refracture, compared with horses with a tibial stress fracture. For this reason, horses rehabilitating from humeral stress fractures warrant careful attention when they resume training, particularly with respect to the effects of training on bone adaptation. In horses with a previous humeral stress fracture that develop a contralateral forelimb lameness, a humeral stress fracture should be strongly considered as a potential cause of the lameness.

In this study, the numbers of horses in each group for which performance before and after injury could be compared were limited. However, results of our analyses of mean earnings per start suggested that horses were able to return to previous athletic performance levels. Similar numbers of horses had increased and decreased mean earnings per start after treatment of a stress fracture. This fact, combined with the fact that most horses returned to racing, suggests that the prognosis for horses with these injuries is good.

References


