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On the nature of stress fractures*

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Since Briethaupt[2] first described "Fussgeschwulst" in 1885 in Prussian Army recruits with edematous and painful feet following long marches, stress fractures have been particularly associated with military training systems. Over the past 40 years, large series[19, 20, 25, 27, 28, 34] reflecting primarily military situations have reported this type fracture in practically every bone, most commonly involving the lower extremity. With the increase of leisure-time sporting activity, stress fractures no longer occupy principally the province of military medicine, but are a common problem among the civilian public, including the pediatric age group.

This paper reviews 17 patients with 21 stress fractures of the tibia, fibula, and femur who were treated over the past 18 months. The patients' ages range from 7 to 28 years and all were involved in competitive athletic activity. Sixteen tibial fractures (four bilateral), three fibular, and three femoral fractures were noted in this group (Fig. 1). The following case presentations illustrate the various loci of these fractures as well as the presenting symptoms and physical findings. A proposal regarding the possible pathophysiology of this condition is also discussed.

CASE 1

An intercollegiate diver presented with vague, aching pain in his leg of several weeks' duration following prolonged workouts. He had marked tenderness and swelling directly over the midtibia of his take-off leg. Roentgenograms confirmed the presence of a stress fracture within his anterior tibial cortex. His symptoms resolved completely following a 4-week period of nondiving.

CASE 2

An intercollegiate miler whose training included running approximately 2,000 miles yearly noted pain on the medial aspect of his thigh, the pain being increased with activity and relieved with rest. Roentgenograms demonstrated a stress fracture of the medial cortex of the midfemur, which became asymptomatic following 6 weeks of rest.

CASE 3

An intercollegiate basketball player noted left shin pain with vigorous basketball play. He had marked swelling and tenderness over the midportion of the tibia and roentgenograms demonstrated persistent periosteal elevation. An excisional biopsy at this site demonstrated thick, fibrous, vascular periosteum with subperiosteal new bone formation. Similar complaints were later noted in his opposite shin and serial roentgenograms of both tibiae demonstrated stress fractures in various stages of repair (Fig. 2). Despite rest periods and casting, he still remained symptomatic when attempting to resume high level basketball competition.

CASE 4

A 14-year-old middle distance runner, whose training included running 30 to 40 miles per week, noted progressive activity-related pain in his lower right leg. His leg was swollen and tender at the

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distal, posterior-medial tibia on examination, and initial roentgenograms demonstrated a periosteal response. Follow-up films 3 weeks later demonstrated maturation of the periosteal callus. A technetium-99m bone scan at the time of the initial visit demonstrated increased uptake compatible with that of a stress fracture. He quickly became asymptomatic on a program of rest.

	AGE (YRS)	SPORT	LOCATION
C E	10	TRACK	TIBIAE (2)
N M	20	TRACK	FEMUR
A S	14	TRACK	TIBIA
P A	15	TRACK	TIBIA
D D	18	TRACK	TIBIAE (2)
M H	19	TRACK	TIBIAE (2)
B S	18	TRACK	TIBIA
T. O	20	MARATHON	FEMUR
J. B.	12	BASKETBALL	TIBIA
M A	20	BASKETBALL	TIBIAE (2)
C G	10	BASKETBALL	TIBIA
R. M.	11	BASEBALL	TIBIA
H. D.	21	DIVER	TIBIA
W. H.	16	DANCER	TIBIA
P. S.	28	TENNIS	FIBULA
J. S.	8	HOCKEY	FIBULA
J. P.	7	?	FEMUR

17 PATIENTS; 21 FRACTURES:
16 TIBIA; 3 FEMUR; 2 FIBULA

Fig. 1.

CASE 5

An 8-year-old boy was involved in vigorous play at a hockey camp 2 weeks prior to being seen. He complained of activity-related pain in his distal leg after skating, and there was tenderness and swelling present at the distal fibula. Initial roentgenograms demonstrated a periosteal response at the clinically tender site. He was taken off skating for 2 weeks and became asymptomatic and follow-up roentgenograms 1 month later showed a healed stress fracture (Fig. 3).

DISCUSSION

Roentgenographic confirmation of stress fractures was done by Stechow[31] in 1897 to document metatarsal fractures of army recruits. Despite the use of roentgenograms, this diagnosis has still been fraught with difficulty and incorrect diagnoses

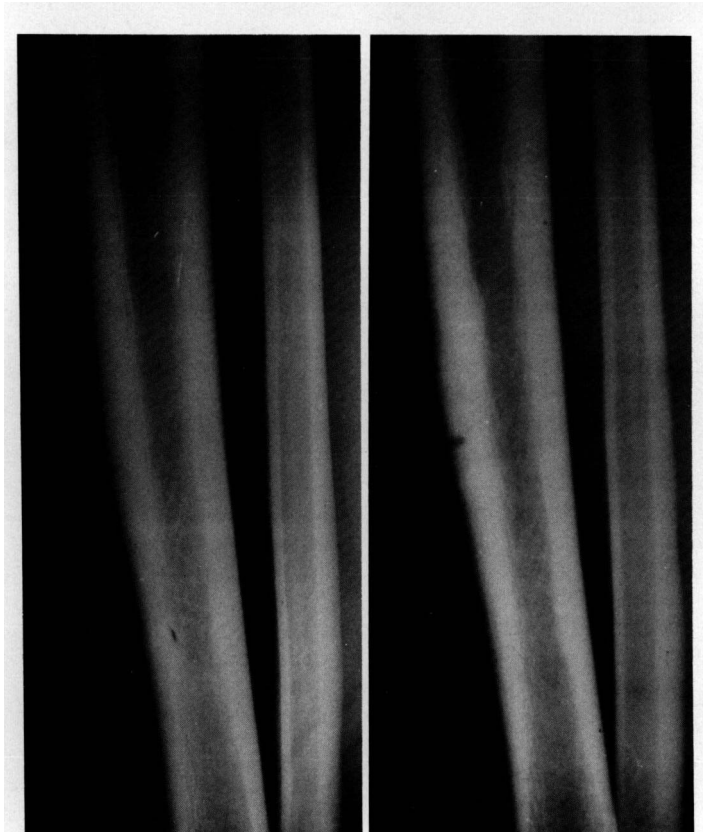

Fig. 2. *Roentgenograms of both tibiae of a basketball player demonstrate stress fractures in various stages of repair.*

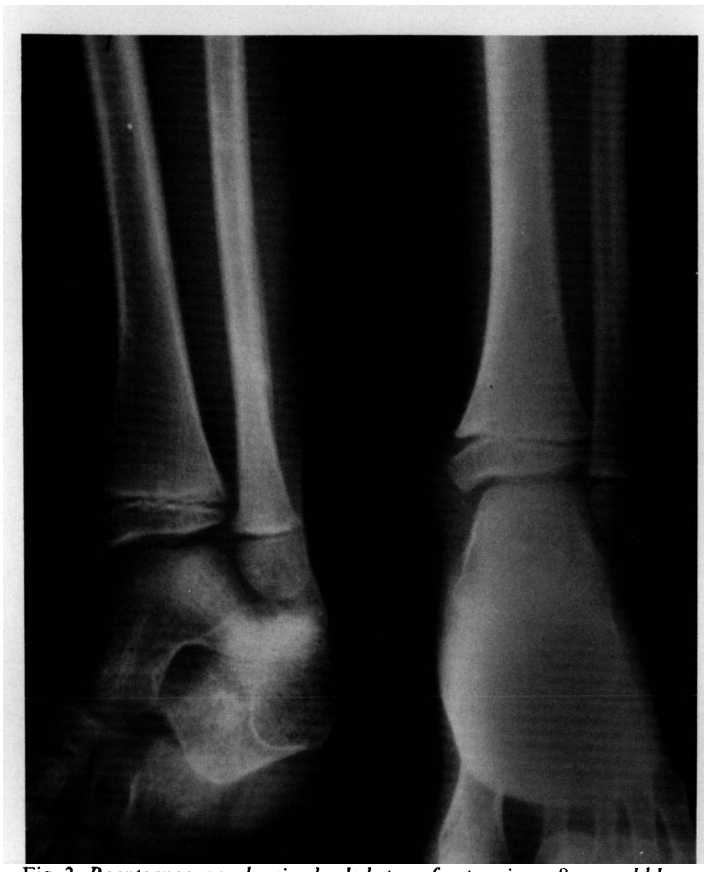

Fig. 3. *Roentgenogram showing healed stress fracture in an 8-year-old boy.*

have included Paget's disease, osteosarcoma, osteomyelitis (both acute and chronic), luetic or tuberculous periostitis, gout, rickets, or osteomalacia, osteoid osteoma, "shin splints," or "growing pains."[34] The terms most commonly given to this entity are "fatigue" or "stress" fracture. However, during the long and descriptive history of this condition, no fewer than 17 appellations have been used.[12, 27] Because of the limited presenting signs, both clinically and roentgenographically, the diagnosis can often be difficult and indeed may only be confirmed retrospectively.

A high index of suspicion must be maintained to detect stress fractures in anyone engaged in a repetitive type activity which causes a focal, mechanical type of pain, i.e., one aggravated with activity and relieved with rest.

Although not diagnostic, the most helpful physical signs are focal swelling and tenderness to palpation over the involved area. Roentgenographic changes will reflect both the duration of the disease as well as the magnitude of stress undergone. Such changes may not develop if the patient voluntarily eliminates the activity which creates the pain as most patients do, who are not in situations where continued maximal performance is demanded. Thus, there will not be a sequence of rarefaction, callus formation, or propagation of a fracture line. Serial roentgenograms in several views are needed to document the presence of progression of a stress fracture in patients whose initial roentgenographic examinations are often negative, but in whom symptoms persist. Tomograms or magnified views may be needed for further confirmation.

As recently described by Hanley et al.,[21] Garrick,[17] and Geslien et al.,[18] technetium-99m bone scans may provide documentation of stress fractures sooner in patients with a history and physical findings compatible with this problem, but roentgenographically silent stress fractures. The accumulation of the radionuclide at the site of increased metabolic activity secondary to mechanical disturbances in these stress fractures will allow earlier treatment which will then result in decreased morbidity.

Briethaupt[2] felt that the condition was caused by a traumatic inflammation of the tendon sheaths and differentiated this from a Morton's neuroma from which he suffered. Since his time the etiopathology of this lesion has remained cryptogenic despite numerous theories advanced as to its cause. In the largest biopsy series reported, Johnson et al.[24] felt that the stress fracture may be a manifestation of an accelerative rate of remodeling of circumferential lamellar bone. In contrast, Friedenberg[16] raised the question of a possible delayed union because of the paucity of callus present and the prolonged treatment time required in his cases. However, in two biopsied cases in our series, subperiosteal new bone formation was readily apparent.

Although stress is a prelude to any fracture, the type under discussion bears a particularly prolonged temporal relationship to its onset of symptoms. Analogies between stress fractures in bone with fatigue fractures within metals have been advanced by several authors.[1, 13, 22, 33] However, bone is a heterogeneous, anisotrophic material; a dynamic reparative tissue that requires stress for normal formation and remodeling. Studies by Evans[13] and others[4, 29, 36] on cyclically loaded bone sections do not take into account the effects of the surrounding soft tissues, primarily the muscle envelope that creates or resists such stresses. Nor do these studies account for the variability in rates of loading in vivo.

A previous concept of stress fractures was that fatigue failure within bone is preceded by fatigue within the surrounding muscles, which then allows excessive forces to be transmitted to the underlying bone.[8, 22, 23] We believe that the reverse of this is the mechanism involved in the development of stress fractures. The highly concentrated muscle forces, acting across a specific bone due to demands imposed from particular repetitive tasks, enhance the loading which occurs simply from direct weight bearing on the affected part. Indeed, weight bearing is not an essential requirement for such fractures as demonstrated by rib fractures secondary to chronic cough.[15] It is the rhythmic, repetitive muscle action that causes subthreshold mechanical insults which summate beyond the stress-bearing capacity of the bone. It is then when the mechanical integrity is lost and the stress fractures become manifest. Hence, the onset of the fracture will be a function of the amount and duration of the stress occurring in concert with the degree of reparative ability of that bone (Fig. 4).

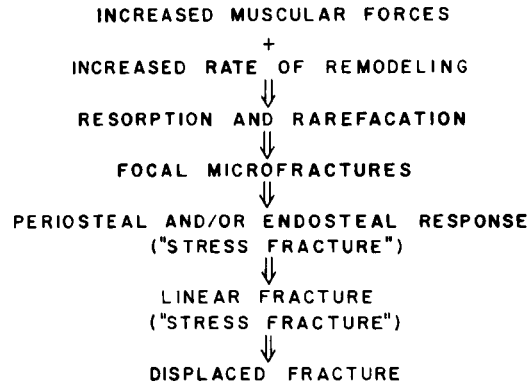

Fig. 4.

Repeating Mueller's work, Chamay and Tschantz[6] demonstrated "slip lines" within dog ulna cortices that had been intermittently subjected to dynamic compressive vs. static compressive loads. They postulated that obliteration of the calculi caused secondary periosteal hypertrophy at zones of such plastic deformative lesions. Radin et al.,[30] Todd et al.,[32] and others[5] have demonstrated that trabecular microfractures occur in given numbers within human femoral head specimens. Since these microfractures occur in otherwise normal human bone, stress fractures may be but an extension of physiologically present microfractures. Because of excessive demands by the patient, forces are increased on adjacent structures with a cascade progression of such microfractures to become clinically manifest as a stress fracture.

Interestingly, in only three animals have stress fractures been documented: thoroughbred racing horses,[7, 12] racing greyhounds,[9] and man.[10–12] These all have been specifically trained to produce maximum performance with certain types of repetitive physical exertion. Sufficient time is often not available for the normal reparative processes of bone to withstand the relentless forces demanded by the athlete. The tibial fracture suffered by the diver and basketball player were felt to be due to forces caused by the large posterior muscle groups causing increased tension across the anterior cortex of the tibia. Similar fractures have been reported in ballet dancers on their "dominant" leg.[3, 26] Previously reported spondylitic defects in football linemen[14] and gymnasts[23, 35] may be manifestations of stress fractures within the pars interarticularis.

Reports among military recruits[19, 20, 25, 28] often alluded to this condition occurring more frequently in the less physically fit individuals. How-

ever, Morris and Blickenstaff[27] commented that it was the usually highly motivated recruits in the military systems who tended to suffer the fatigue fractures. Indeed, most of the patients reported in this paper suffered their fractures during mid- to late season at times when they were fully conditioned and at maximal performance. Five fractures of the leg occurred in children less than 14 years old while involved in team play. Hence, suspicion of this diagnosis must also be entertained in pediatric age groups participating in active play.

Treatment will vary with the location and magnitude of the fracture. Abstinence from athletic activity for 3 to 4 weeks is the main therapeutic modality, with ambulatory casts used for the overly enthusiastic person to enforce limited activity. The decrease in activity will often abort the stress fracture cycle and thus prevent the production of a complete fracture. Once the fracture is established, however, the treatment often becomes difficult and the recovery period prolonged, especially in athletes who are expected to return to maximal functional activity.

SUMMARY

It is felt that stress fractures are caused by excessive, repetitive muscle forces acting across the affected bone. These fractures should be suspected in participants of athletic endeavors who present with a history of persistent, focal, activity-related pain regardless of their stage of physical conditioning. Associated physical findings are localized tenderness and swelling without evidence of a generalized systemic response. Bone scans and serial roentgenograms including specialized views may be required for documentation. Limitation of the athletic activity is the hallmark of the treatment program.

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EDITORIAL COMMENT

Dr. Victor H. Frankel, *Seattle, Washington:* The authors correctly point out that the onset of a fatigue fracture will be a function of the amount and duration of stress occurring in a bone, in concert with the degree of reparative ability of that bone. All fractures can be pictured as a continuum resulting from this interplay of force and repetition (Fig. A). A fracture may result from the application of a single load, if that load induces a stress in the bone that exceeds its normal yield limit. Although fatigue fractures can be found to lie along this same curve, hopefully the curve tails out into a horizontal line, indicating that if the stress is kept below a certain level these fractures will never occur; no matter how great the number of repetitions. Although this situation is true for many metals, it may not be true for bone, as the rapidity of the repetitions may preclude necessary remodeling.

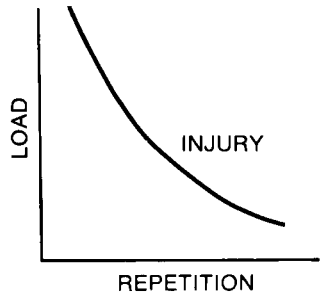

Fig. A. *Hypothetical fatigue curve.*

In my opinion, there are many etiologies for the production of fatigue fractures. One is simple overload brought about by muscle contraction, as emphasized by the authors. Another may be an altered stress distribution in the bone, brought about by continued activities in the presence of muscle fatigue. A third factor is a change in the ground reaction force, which alters the stress pattern in the bone. A fourth mechanism may be high repetition of stress application, even though the stresses are kept at a relatively low value. Chamay[1] has emphasized that stress fractures occur when bone is loaded repeatedly in the plastic region. This is perhaps the common mechanism for fatigue fractures; however, these fractures may also occur if the bone is loaded only in the elastic region but with a very large number of rapid repetitions.

Fatigue fractures may occur on either the tension or the compression side of the bone. It should be emphasized that a fatigue fracture on the tension side, resulting in a crack in the bone, is of a very serious nature, because the bone may rapidly go on to a complete fracture. Fatigue fractures that occur on the compression side of the bone seem to result from a much slower process, in which repair mechanisms are more easily mobilized, thus preventing a complete fracture.

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Authors' Reply: We agree that the production of stress fractures may be multifactorial, but wish to emphasize the role of muscles as both protectors of bone from excessive force and as initiators of forces across the bone. As noted in the paper, bone, an autoreparative tissue, is not analogous to metals and their type of failure. Failure within bone, presenting as a stress fracture, seems to be secondary to the diminished time available for the necessary remodeling processes due to "command performances" in the patients. As such, a fourth variable, time, is required in the hypothetical graph presented by Dr. Frankel.