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Radiographic and histologic analyses of stress fracture in rabbit tibias*

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ABSTRACT

Sequential changes in remodeling of the internal structure of the tibia caused by controlled, excessive jumping and running were studied in 20 rabbits. Vascular changes and circulatory disturbances within the cortical bone occurred before osteoclastic resorption. Degeneration and necrosis of osteocytes due to circulatory disturbances also occurred. Periosteal new bone formation, found at and after 12 days of the experiment, was a compensatory reaction to support the tibia weakened by accelerated osteoclastic resorption. Small cracks appeared at the cement line and developed through the neighboring cement line of the haversian systems. At 21 days, incomplete fracture of the tibial cortex was found in two rabbits. Complete fracture through one side of the cortex was seen in one animal at the 50th day of the experiment. In this study, however, most of the tibias did not have visible fracture lines after a period of stressful exercise. This result suggests that most tibias adapt to changes in stress requirements through proper internal remodeling so that a complete fracture does not occur.

In a stress fracture, there is no initial, overt break as a result of a single traumatic incident. Rather, there is a gradual alteration of the bone structure caused by repeated, non-violent, and usually unaccustomed stress that may or may not eventually result in a complete fracture. The most common site of stress fracture in athletes is the tibia, with the peak incidence occurring in middle-distance runners.[2,16,39] Stress fracture of the tibia was first described by Aleman[1] in 1929. He noted that 100 cases of so-called "periostitis tibia ab excerito" were reported each year in the Swedish army and were considered to be "insufficiency fractures of the tibia." Devas,[9] in 1958, was the first author to report stress fracture of the tibia in athletes. He believed that shin splints in athletes were essentially an atypical type of stress fracture of the tibia that were difficult to diagnose, since the fracture line might involve only one cortex of the bone and might not appear on radiographs in the early stages.

Similar stress fractures have also been found in race horses and dogs[10,11] but only a few studies of the pathologic features of tibial stress fracture have been reported. Recently, several animal experiments were conducted to study the relationship between changes in stress and bone remodeling.[5,22,42] In these studies, however, radiographic and histologic variations of bone remodeling during exercise were not followed up in detail throughout the training period. In addition, it would be more valuable to study bone remodeling in more closely simulated athletic activities.

To study pathologic changes in the internal structure of the tibia caused by controlled, excessive jumping or running, we performed an experiment with a rabbit model. An electric cage with high pulsive voltage was used to induce animal jumping and running activities under a controlled frequency and period. Radiographic and histologic methods of analysis allowed description of the developing pathologic signs of stress fracture formation throughout a 60 day period.

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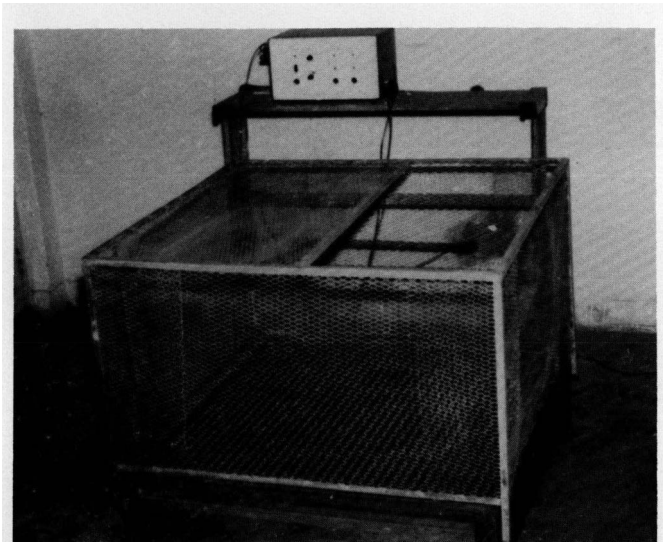

Figure 1. Electric cage with high voltage and low current.

MATERIALS AND METHODS

Twenty male rabbits of the same species were used in this study. Their weights ranged from 2.5 to 2.8 kg. Eighteen of the rabbits were in the experimental group, and the other two served as controls.

The electric cage with high voltage (15,000 volts) and low current (100 to 200 microamperes) used in this study was designed at the National Research Institute of Sports Science of China (Fig. 1). The intensity, duration, and interval of stimulation could be controlled. The experimental rabbits were trained to jump and run within the cage in response to each electrical stimulation. With each brief electrical stimulation, the rabbits jumped forward and upward, to a height of about 15 cm, using the hindlimbs, and then ran. Each stimulation lasted 0.2 to 0.4 seconds, followed by a 20 second interval of rest. This sequence of stimulation was performed for 2 hours per day, with a 10 minute break after the first hour of "training." The exercise program was carried out 6 days per week for a continuous period of 60 days. Thus, each experimental animal jumped approximately 180 times per hour, or 360 times per day, under the cage environment. During the remaining time, they were allowed to engage in normal activities, the same as those observed in the control animals.

This exercise regimen did not seem to cause any untoward effect on the well-being of the experimental animals, since they exhibited the same feeding and activity patterns as the controls. Most of the animals in the experimental group had weight gains comparable to those of the control animals during the period of experiment.

The experimental rabbits were sacrificed according to the following schedule: One rabbit on days 2, 4, 7, 10, and 12 of "training;" two rabbits on day 14; one rabbit on day 16; and two rabbits on days 21, 30, 40, 50, and 60. One of the two rabbits in the control group was sacrificed at the beginning of the experiment and the other at the end.

Both tibias of each animal were harvested, immersed in 10% formalin, and then stored for a period of 1 month in 5% edetic acid to decalcify them. Histologic slices were then taken from the sites of periosteal reaction, as shown on radiographs, and paraffin slices were prepared with hematoxylin and eosin, van Gieson, and periodic acid-Schiff stains. Nondecalcified slices from one tibia of a control group rabbit and one tibia from the 4 week training group were prepared by grinding. Lateral and anteroposterior radiographs of the tibias were made before histologic preparation.

RESULTS

Radiographic changes

Progressive periosteal reaction was found radiographically in 20 tibias of 18 rabbits (55%) as the training progressed, whereas the remaining tibias showed only soft tissue swelling or no radiographic change. The changes at different chronologic periods are described as follows:

- At the seventh day (after approximately 2,160 jumps), only the soft tissue around the tibia was swollen and enlarged. The tibial cortex was normal (Fig. 2).
- At the 14th day (after approximately 4,320 jumps), some periosteal reaction was noted. The outline of the tibial cortex was still normal radiographically (Fig. 3).
- At the 21st day (after approximately 6,480 jumps), periosteal reaction was more obvious, and increased periosteal new bone formation was present. The thickness of the cortex in the tibial shaft increased, but the edge of the cortex was still smooth (Fig. 4).
- After 30 days (8,640 jumps), the periosteal new bone formation was markedly increased, the cortex was thickened, and the edge of the cortex had become irregular (Fig. 5).
- The changes mentioned above were also found at 50 and 60 days. In addition, S-shaped angular deformities of the tibias were observed radiographically (Fig. 6).

Although cortical fracture and microfracture were found in this study, no obvious fracture lines through the cortex were noted on any of the radiographs of the tibias. Nine of the 11 rabbits with radiographic changes had bilateral changes, but the severity was not symmetrical. The lesions were located in the medial one-third of 16 tibias (80%), the distal one-third of three tibias (15%), and the upper one-third of the one tibia (5%). None of these findings were observed in the two control animals.

Pathologic changes

Osseous changes—On the second day of the experiment, the number of erythrocytes was increased in the vessels of the haversian canals (Fig. 7). By the fourth day, the vessels of the haversian canals were dilated and congested (Fig. 8). Nonstructured cylindrical eosinophilic hyaline thrombosis occurred in some vessels of the haversian canals which showed a positive reaction to the periodic acid-Schiff stain (Fig. 9).

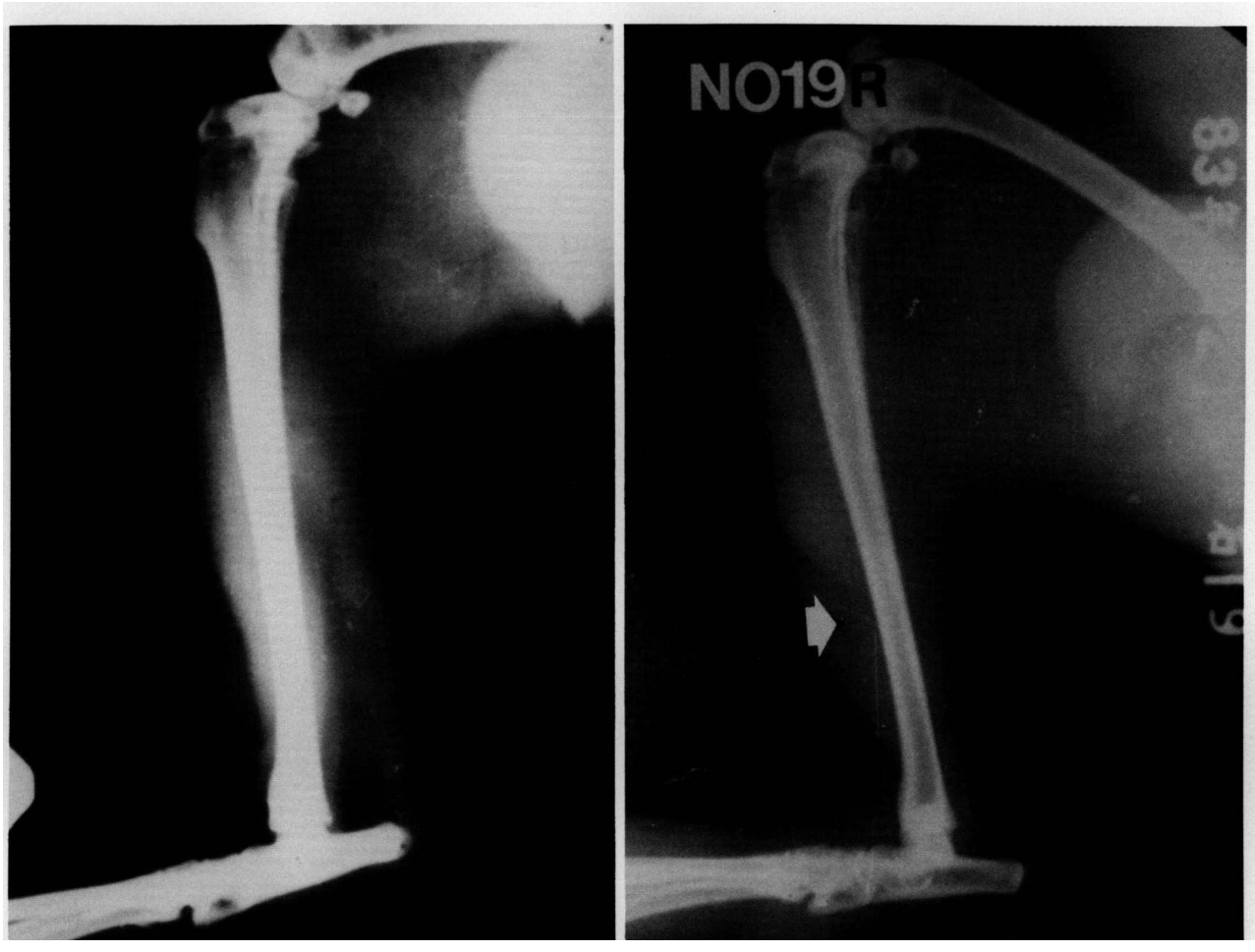

Figure 2. Left, normal roentgenogram of rabbit's tibia, lateral view. Right, swelling of soft tissue (arrow) surrounding tibia on seventh day of experiment.

By the seventh day of the experiment, generation of osteoclast and resorption cavities were observed in the cortex of the tibia. Osteoclastic resorption in the haversian canals resulted in enlargement of the canals. Osteoclastic resorption was also seen at interstitial lamellae, where it caused formation of cavities. Within the cavities, one or two large osteoclasts with multiple nuclei and some osteoblasts were observed (Fig. 10). Simultaneously, the osteocytes shrank or occasionally disappeared in the lacunae of partial haversian systems (Fig. 11). Obvious osteoclastic resorption and a large number of cavity formations were noted at the 14th day of the experiment.

On the 10th day, small cracks appeared at the cement line of the haversian system, especially on the anterior and medial regions of the tibia (Fig. 12). The edge of the crack was rough and darkly stained. No osteoclasts or osteoblasts appeared in the small cracks. Incomplete fracture of the tibial cortex was found in two rabbits at the 21st day of the exercise. The fracture lines were irregular and were formed by the convergence of several neighboring cracks in the haversian system. One was located at the outer circumferential lamellae, and the other was near the inner circumferential lamellae (Fig. 13). On the 50th day, a cortical fracture was seen that had some osteoblasts on the rough fracture edge. The fracture line was transverse and involved only one side of the cortex (Fig. 14).

Periosteal changes and new bone formation

On the fourth day of exercise, the collagen fibers of the periosteum had a loose appearance, but no evidence of cellular proliferation was found (Fig. 15). By the seventh day, however, fibroblasts and fibrocytes had increased in number, and the formation of collagen fibers was seen. With increasing experimental time, the periosteum progressively thickened, and capillaries became abundant (Fig. 16).

By the 12th day of the experiment, subperiosteal osteoblastic activity had increased, with a single layer of cells along the surface of bone. There was a progressive accumulation of osteoblasts surrounded by interstitial bone tissue which resulted in "woven bone" (Fig. 17). New bone formation was maximum after 21 days. In addition, a great number of chondrocytes appeared in the new bone (Fig. 18).

Figure 3. Flocculent periosteal reaction in second week.

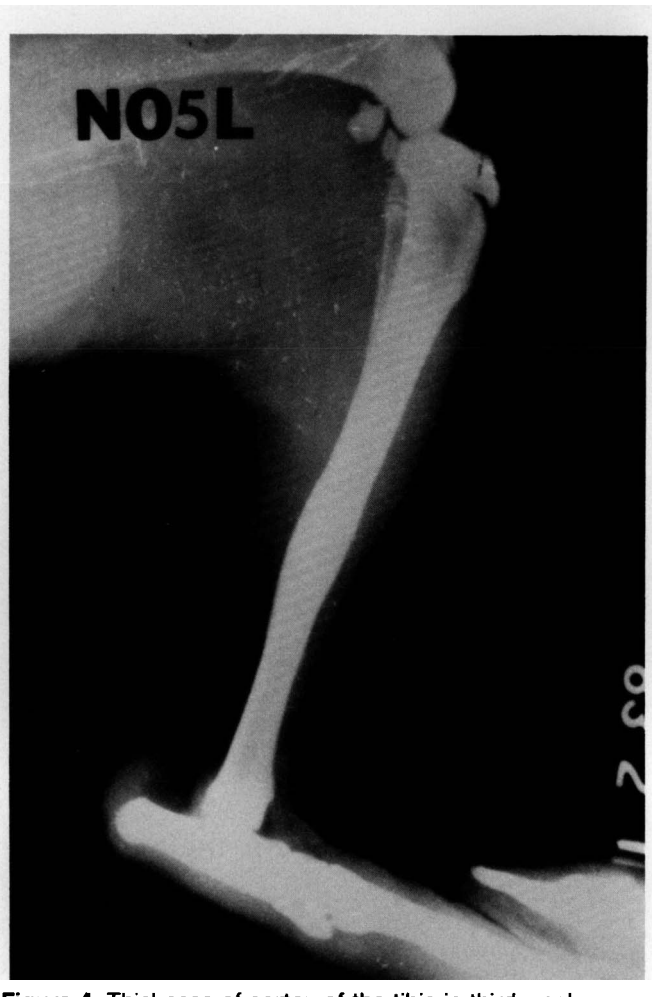

Figure 4. Thickness of cortex of the tibia in third week.

Bone remodeling occurred in both the newly formed bone and original bone of the tibial cortex. Osteoclasts accompanied the large amount of new bone. An increase of capillaries was accompanied by the osteoclastic process and was followed by some haversian canal-like structures which appeared gradually as the new bone began to change to mature bone (Fig. 19). In the original bone, enlargement of the haversian canals and formation of cavities were also due to activity of the osteoclasts. A large number of osteoblasts appeared along the enlarged haversian canals and cavities in the interstitial lamellae. The cavities were gradually filled in by new bone. In the last stage of this experiment, as a result of the fusion of periosteal new bone and original bone during the remodeling process, the tibial cortex became thickened (Fig. 20). All results of radiographic and pathologic changes are summarized in Table 1.

DISCUSSION

Cortical bone remodeling caused by overload stress

Wolff's law states that any change in the form or function of bone will cause definite changes in its internal architecture and secondary alterations in its external configuration. Bone is a dynamic, reparative material which requires a certain amount of stress for normal formation and remodeling. Decreased or increased stress will cause pathologic changes of the bone. It is well known that disuse or immobilization results in rapid osteoclastic resorption, which is followed by a decrease in osteoblastic activity.[25] Abnormal bone resorption within the tibia may also be found in some running or jumping athletes after a period of overtraining.[3,7,13,23,29,31,38] These examples indicate that bone is remarkably sensitive to stress.

In this study, osteoclastic resorption of the cortex appeared in animal tibias after the 7th day of experiment, and periosteal new bone formation was observed on the 12th day. Microfractures were seen by the 10th day, and one cortical fracture was noted on the 50th day of forced jumping and running. Obvious bone resorption and a large number of cavity formations appeared at 14 days. A great amount of periosteal new bone formation was observed after the 21st day of exercise. These results are very similar to those of Johnson et al.,[21] who obtained biopsies of tibial stress fractures from military recruits.

Acceleration of osteoclastic resorption caused by overload stress is the initial pathologic change that occurs in the

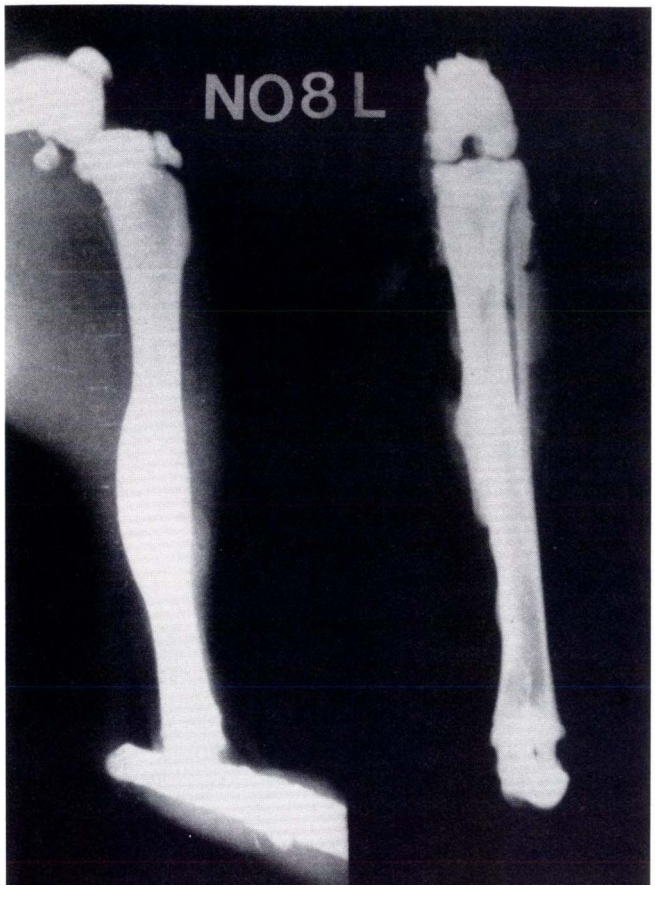

Figure 5. Obvious periosteal reaction on 30th day of experiment. Surface of cortex bone has become irregular.

Figure 6. S-shaped angular deformity of tibia on 50th day.

development of stress fracture of the tibia. However, the mechanism that causes this increase in bone resorption is still unknown. In this study, vascular changes and disturbances of circulation in the haversian systems, with engorgement, vasodilation, and hyaline thrombosis, were observed microscopically before osseous resorption took place. This disturbance may have resulted in ischemia and anoxia of bone that would lead to degeneration and necrosis of osteocytes. Ischemia and anoxia may have been responsible for the increased osteoclastic activity that created resorption cavities within the cortex. These resorption cavities were then filled with harder lamellar bone through deposition by osteoblasts. However, the process of bone formation appeared to lag behind osteoclastic resorption, temporarily disturbing the equilibrium between bone resorption and regeneration. Using intermittent tetracycline bone labeling, Johnson et al.[20] found that an average of 90 days was required for a newly formed resorption cavity to circumferentially fill with mature bone. Therefore, in a fatigue environment, the strength of the tibia would be reduced because of this disturbance.

Periosteal proliferation and new bone formation

It has been noted for a long time that excessive running and jumping can induce not only skin soreness[17–19,27,35] but also

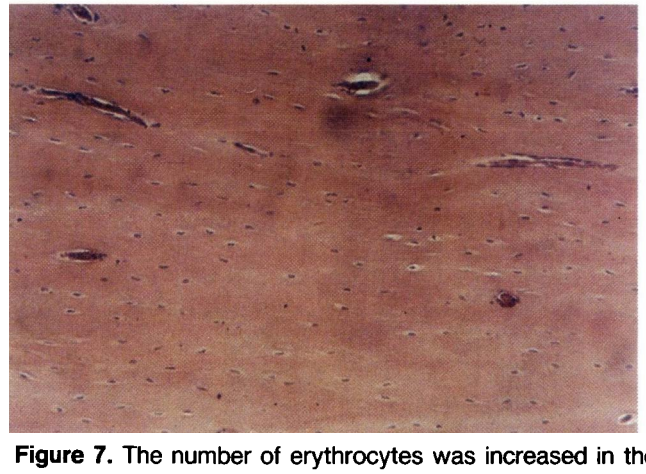

Figure 7. The number of erythrocytes was increased in the vessels of bone. (Longitudinal section: hematoxylin and eosin, ×100.)

periosteal reaction in the tibia. At first, Deutschlander believed that the periosteal reaction was caused by a hematogenous infection.[8] Hirayama[32] and Mironova et al.[24] stated that periostitis was produced by repeated pulling of the periosteum by the attached muscles. Some authors[15,32] found

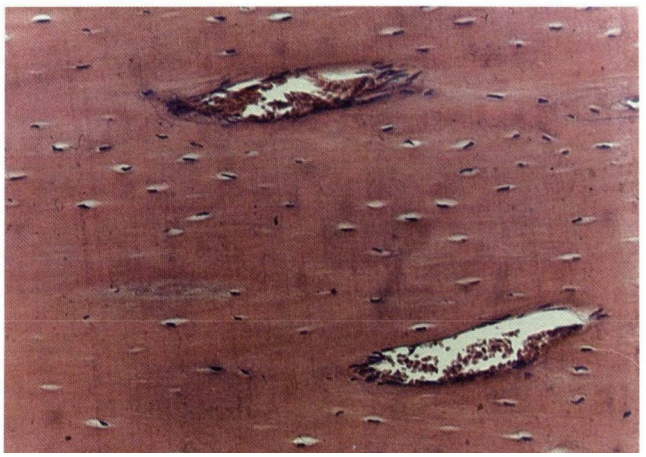

Figure 8. Dilated and congested vessels in haversian canals. (Longitudinal section: hematoxylin and eosin, ×200.)

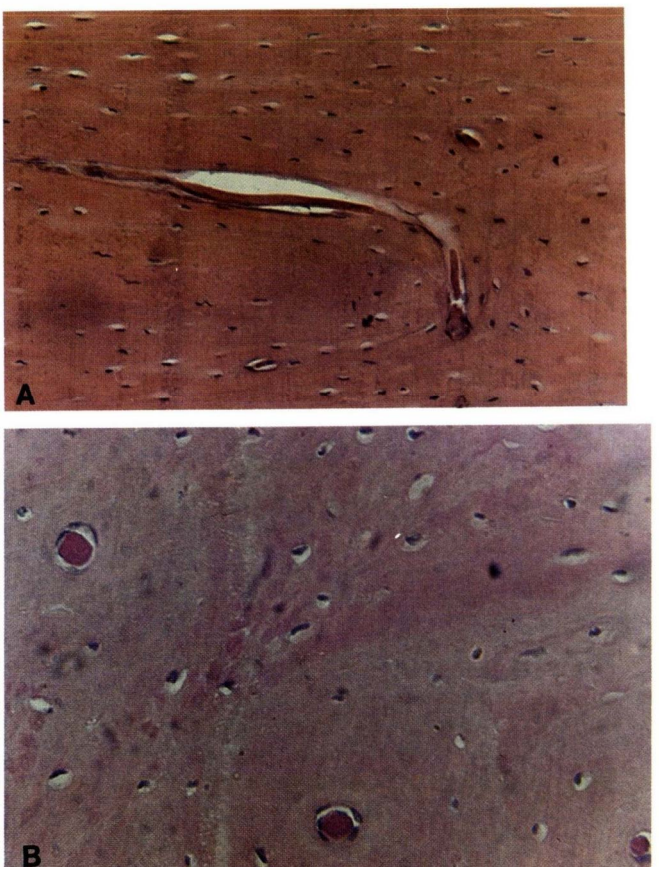

Figure 9. A, hyaline thrombosis in vessels of haversian canal. (Longitudinal section: hematoxylin and eosin, ×400.) B, hyaline thrombosis in vessels of haversian canals. (Cross-section: periodic acid-Schiff, ×400.)

that the periostitis was related to fatigue of local tissues, and it was termed "fatigue periostitis of the tibia." A few authors have pointed out that periosteal proliferation and

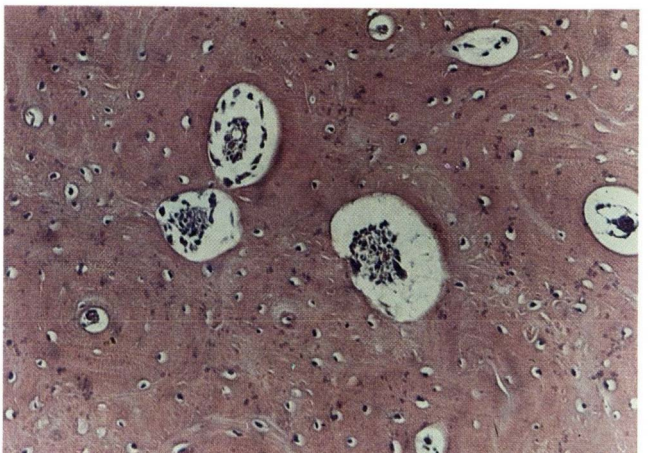

Figure 10. Osteoclastic resorption at haversian canals and interstitial lamellae. Osteoclasts and osteoblasts are in resorption cavities. (Cross-section: hematoxylin and eosin, ×100.)

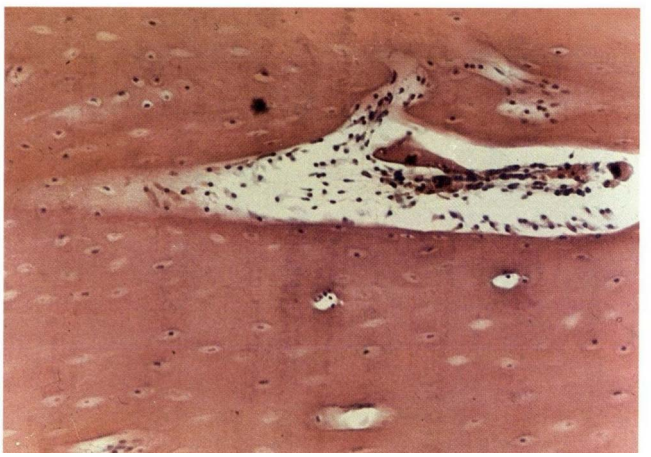

Figure 11. Osteocytes shrank or disappeared in lacunae. (Longitudinal section: hematoxylin and eosin, ×200.)

Figure 12. Small crack at cement line of haversian system. (Cross-section: hematoxylin and eosin, ×400.)

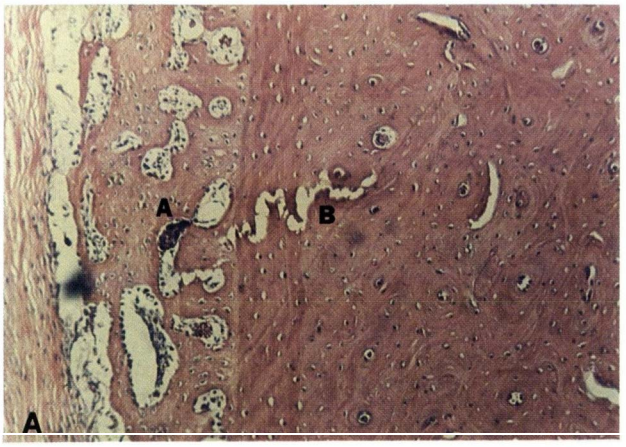

Figure 13. A, microfracture in outer circumferential lamellae. A, new bone; B, microfracture. (Cross-section: hematoxylin and eosin, ×40.) B, microfracture at inner circumferential lamellae. (Cross-section: hematoxylin and eosin, ×100.)

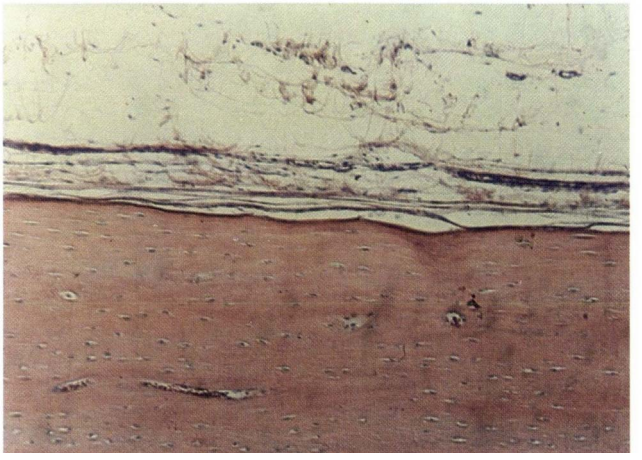

Figure 15. Swelling of periosteum. (Longitudinal section: hematoxylin and eosin, ×100.)

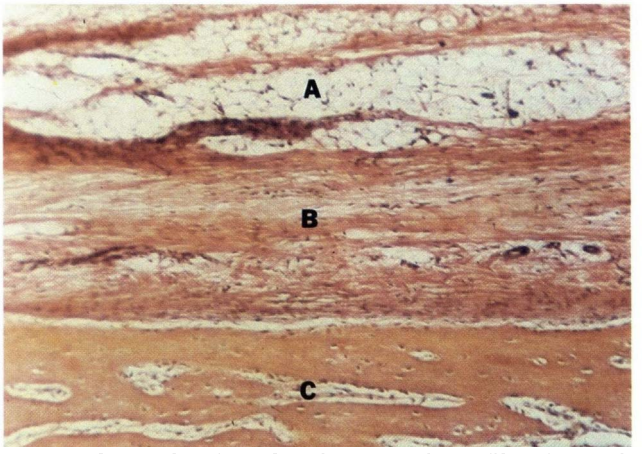

Figure 16. Proliferation of periosteum. A, proliferation of fat tissue within periosteum; B, proliferated periosteum; C, tibial cortex. (Longitudinal section: van Gieson, ×40.)

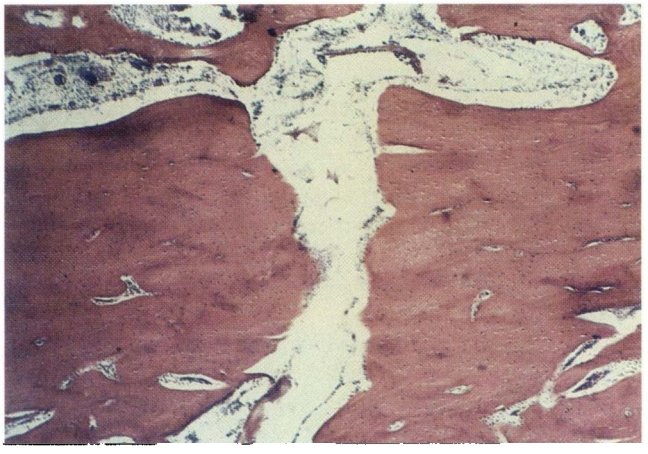

Figure 14. Fracture in one side of tibial cortex. (Longitudinal section: hematoxylin and eosin, ×40.)

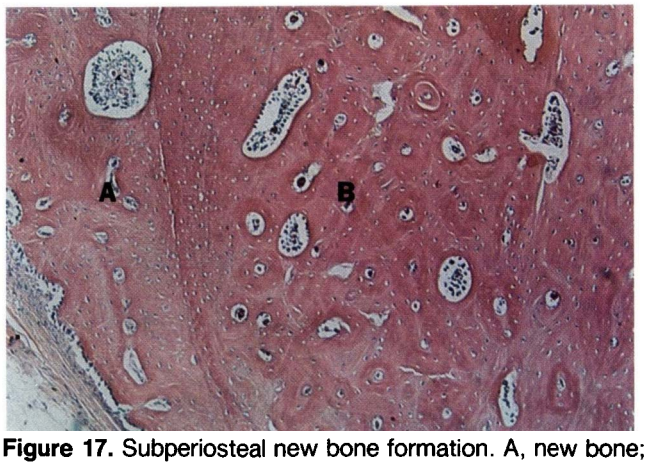

Figure 17. Subperiosteal new bone formation. A, new bone; B, cortex. (Cross-section: hematoxylin and eosin, ×100.)

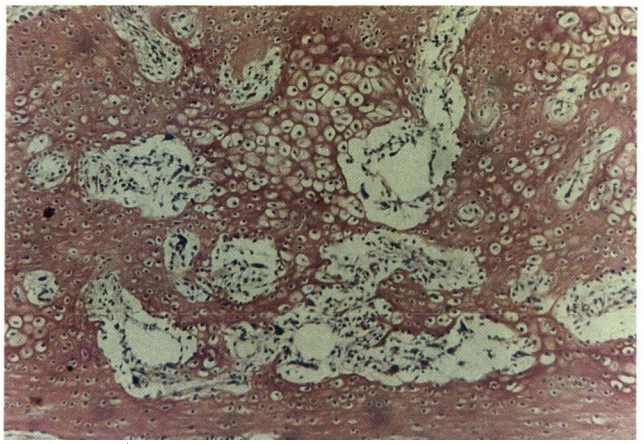

Figure 18. Chondrocytes in newly formed bone. (Longitudinal section: periodic acid-Schiff, ×100.)

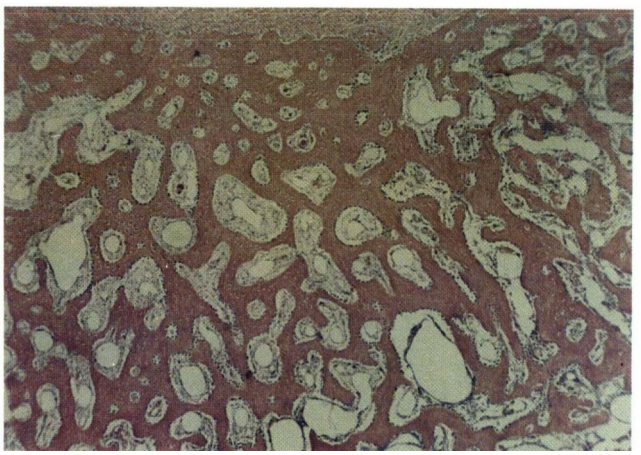

Figure 19. New bone remodeling. (Cross-section: hematoxylin and eosin, ×100.)

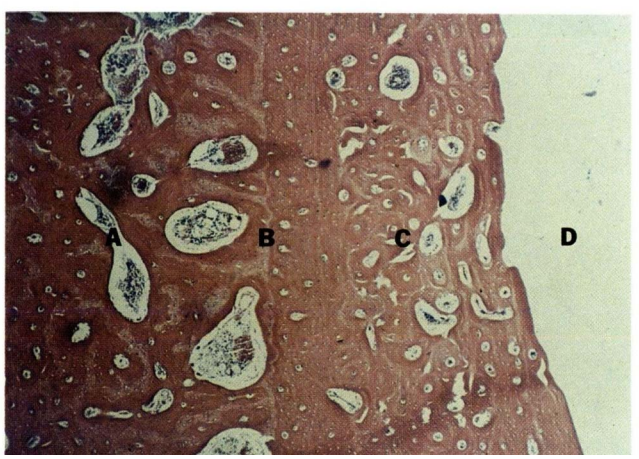

Figure 20. Tibial cortex remodeling. A, new bone; B, fusion region; C, original bone; D, medullary cavity. (Cross-section: hematoxylin and eosin, ×40.)

new bone formation may provide temporary support to the tibia weakened by bone resorption.[12,41]

Results of the present study indicate that periosteal proliferation and new bone formation appear only after bone resorption. Periosteal new bone formation generally appeared on the anteromedial side of the tibia, in the same area in which active bone resorption occurred. Therefore, it is believed that this new bone formation is a compensatory reaction of living bone to meet the stress requirement of the tibial cortex which has been weakened temporarily by bone resorption.

Clinically, therefore, when periosteal new bone formation is noted radiographically, abnormal resorption or fracture lines in this area should be considered. In this study, we found that progressive periosteal callus formation was the only finding on radiographs. Therefore, for the diagnosis of tibial stress fracture, a definite fracture line need not be present.

Stress fracture

Stress fracture of the tibia has received attention since it was first described by Aleman in 1929.[1] In 1956, Burrows[4] reported five cases of insufficiency fracture of the tibia in ballet dancers and termed it "fatigue fracture of the tibia." Devas[9] reported on 17 cases of stress fracture of the tibia in athletes in 1958. In his study, fracture lines in the tibia were found in 11 cases, and the other six showed only periosteal reaction. Clement,[6] in 1974, noted that in overload stress, fatigue of the muscular system takes place and results in a loss of the shock absorption effect of the muscles. Then the additional stress is applied directly to the bone tissue and causes periostitis. Ultimately, with further stress overload, stress fracture can result. With bone scanning, stress fracture of the tibia can be diagnosed as early as 3 weeks before radiographic changes appear.[14,28,30,34,36,37,40] In follow-up studies, patients with the diagnosis of shin splints later had radiographic periosteal reaction or fracture lines.[9,41] Morris[26] and Roub et al.[33] expressed the belief that stress fracture of the tibia is not a single event like acute traumatic fracture but, rather, a process of alteration of bone.

In this experiment, small cracks around the cement line of the haversian systems exemplify the commonly recognized microfracture. These small cracks can be formed under a localized fatigue stress condition during the running and jumping activities. We also found that the small cracks propagate through the neighboring cement line of the haversian systems when the activities continue. Finally, if fatigue stress persists and new bone formation is delayed, cortical fracture may ensue.

In this study, only one tibia showed a cortical fracture at 50 days. Most of the tibias demonstrated bone remodeling after a period of overtraining, but did not exhibit visible fracture lines. This finding may be due to the shortness of the experimental period and lack of severity of the exercise program. However, the results seem to suggest that most of the tibias may adapt gradually to the stress environment

TABLE 1
Summary of radiographic and pathologic changes

Day	Bone		Periosteum	
	Radiograph	Histology	Radiograph	Histology
2	Normal	The number of erythrocytes increased in the vessels of bone	Normal	Normal
4	Normal	Vessels congested and thrombosis in haversian canals	Normal	Swelling
7	Normal	Osteoclastic resorption	Normal	Slight periosteal proliferation
10	Normal	Small crack at cement line	Normal	Periosteal proliferation and increase in osteoblasts
12	Normal	Resorption cavities and small crack	Normal	Periosteal proliferation and increase in osteoblasts
14	Normal	Resorption cavities and small crack	Periosteal reaction	New bone formation
16	Normal	Bone remodeling	Periosteal reaction	New bone formation
21	Cortical thickening	Incomplete fracture (two tibias)	Periosteal callus	New bone formation
30	Cortical thickening	Bone remodeling	Periosteal callus	New bone remodeling
40	Edge of cortex irregular	Bone remodeling	Periosteal callus	New bone remodeling
50	An S-shaped angular deformity	Cortical fracture (one tibia)	Periosteal callus	New bone remodeling
60	Cortical thickening	Bone remodeling	Periosteal callus	New bone remodeling

through an internal remodeling process so that complete fracture does not occur.

CONCLUSIONS

- Stress fracture of the tibia caused by excessive stress is not periostitis or a single event (like acute traumatic fracture) but is a sequential pathologic process of impairment and repair of bone, including the periosteum.
- Accelerated resorption of bone is the initial stage of stress fracture of the tibia. Excluding the direct effect of stress, osteoclastic resorption may be a result of circulatory disturbances within the haversian systems.
- Periosteal proliferation and new bone formation are compensatory responses of living bone to elevated stress magnitude and frequency. They can provide temporary splinting that supports the tibia weakened by bone resorption.
- Fracture may appear if excessive stress continues in a tibia weakened by osteoclastic resorption. However, such fractures do not occur in most tibias because the bone adapts to changes in stress requirement through proper bone remodeling that is regulated by a series of compensatory mechanisms.

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