

Rapid #: -15586283

CROSS REF ID: **1012110**

LENDER: **HUA :: Main Library**

BORROWER: **COD :: Main Library**

TYPE: Article CC:CCL

JOURNAL TITLE: Radiology

USER JOURNAL TITLE: Radiology

ARTICLE TITLE: RPC of the Month from the AFIP

ARTICLE AUTHOR: Sweet, Donald E

VOLUME: 99

ISSUE: 3

MONTH: 06

YEAR: 1971

PAGES: 687-693

ISSN: 0033-8419

OCLC #:

Processed by RapidX: 1/1/2020 9:49:52 PM



This material may be protected by copyright law (Title 17 U.S. Code)

19**Rapid #: -15586283****RapidX Upload**

Status	Rapid Code	Branch Name	Start Date
New	COD	Main Library	01/01/2020 05:28 AM
Pending	HUA	Main Library	01/01/2020 05:28 AM
Batch Printed	HUA	Main Library	01/01/2020 05:34 AM

CALL #: **MSX 616.075705 R129**

(10030838)

LOCATION: **HUA :: Main Library :: medxs**

Article CC:CCL

REQUEST TYPE:

JOURNAL TITLE:

USER JOURNAL TITLE:

HUA CATALOG TITLE:

ARTICLE TITLE:

ARTICLE AUTHOR:

VOLUME:

ISSUE:

MONTH:

YEAR:

PAGES:

ISSN:

OCLC #:

CROSS REFERENCE ID:

VERIFIED:

BORROWER:**COD :: Main Library**

 This material may be protected by copyright law (Title 17 U.S. Code)
1/2/2020 8:57:36 AM

香圖
港書
大館
學



THE UNIVERSITY OF HONG KONG LIBRARIES
Lending Services Division
Pokfulam Road, Hong Kong

Circulation enquiries: (852)39175945 / 39172202

E-mail: libcir@hku.hk

Interlibrary Loan enquiries: (852)3917-2215 / 3917-5895

E-mail: interlib@hku.hk

Fax: (852)25174615

Associate Librarian, Engagement and Lending Services: 39177001

University of Hong Kong Libraries

© The copy is for purposes of private
study or scholarly research only.

You should delete the file as soon as a
single paper copy has been printed out
satisfactorily.

RPC¹ of the Month from the AFIP²

Maj. DONALD E. SWEET, USAR MC, and Lt. Col. ROBERT M. ALLMAN, USAF MC

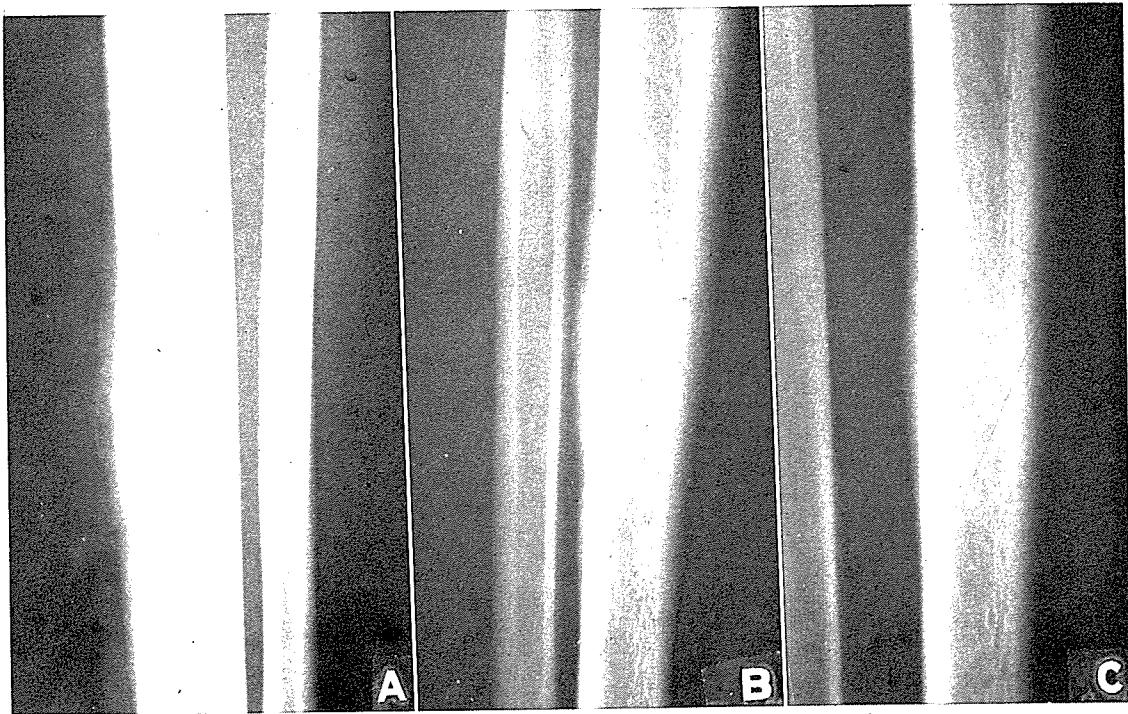


Fig. 1 (AFIP Negatives 70-2225-2 and 70-2225-1).

THE ABOVE roentgenograms are those of a 20-year-old Caucasian man who was asymptomatic until five weeks prior to examination, when he noted aching over the anterior surface of the right tibia. There are several diagnostic possibilities for the lesion shown in Figure 1, including osteosarcoma, stress fracture, osteoid osteoma, and Garré's osteomyelitis. How would you analyze this patient's solitary bone lesion, and what is your list of differential possibilities?

¹ RPC = Radiological-Pathological Correlation.

² From the Registry of Radiologic Pathology (R. M. A., Associate Radiologist), American Registry of Pathology, Armed Forces Institute of Pathology (D. E. S., Staff Pathologist, Orthopedic Pathology Branch), Washington, D.C. Accepted for publication in February 1971.

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official nor as reflecting the views of the Departments of the Army, the Air Force, or of Defense. sjh

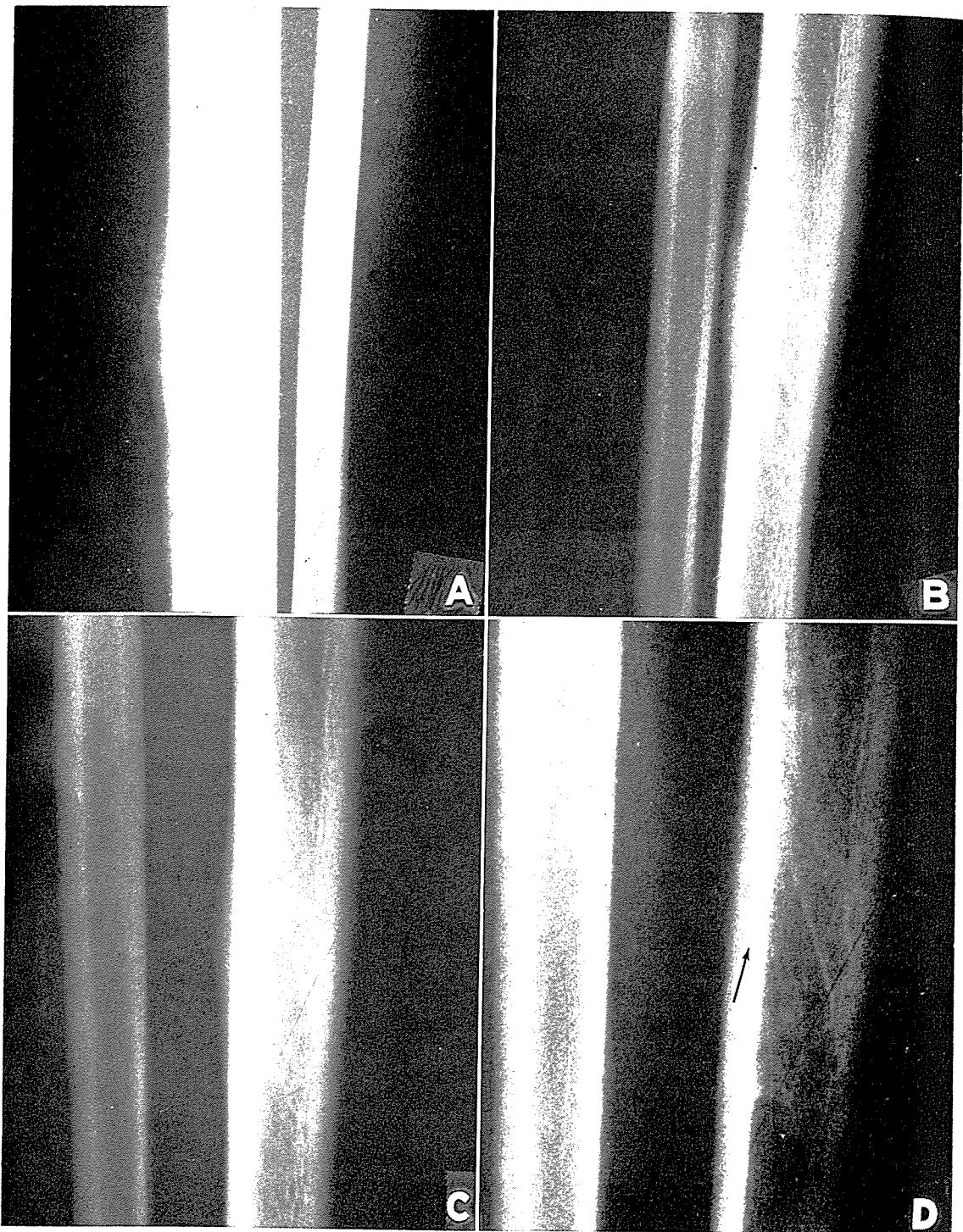


Fig. 2 (AFIP Negatives 70-2225-2 and 70-2225-1). Periosteal new bone formation seen in Figure 2, A-C extends around the anteromedial aspect of the tibia. Endosteal new bone reinforcement is demonstrated best in Figure 2, C. The new bone overlies a poorly marginated, dimly discernible radiolucency which is confined to the cortex. The lucent zone is visualized best in Figure 2, D, which is a photographic enhancement of Figure 2, C.

RADIOLOGICAL FINDINGS

Periosteal new bone formation is present at about the junction of the middle and distal thirds of the tibia and extends around the anteromedial aspect of the diaphysis (Figs. 2, A, 2, B, and 2, C), overlying a poorly marginated focal area of radiolucency which is confined to the cortex. The vague radiolucency can best be appreciated by comparison with the normal density of the cortex above and below the level of the lesion. Figure 2, D is photographically enhanced to illustrate this feature. The endosteal surface beneath the lesion also shows new bone reinforcement.

DISCUSSION

The periosteal bone formation is solid and narrow and appears to represent a reinforcing osteoblastic response to the dimly discerned underlying osteolysis. The subtle radiolucency (an osteoclastic response) is small and local and involves only the cortex. The absence of a soft tissue mass or evidence of intramedullary disease further attests to the focal intracortical containment of the lesion. These features favor the diagnosis of a benign lesion; since neoplasms are not intracortical, osteosarcoma can be excluded from the differential list. Thus the only diagnostic possibilities would be inflammatory or repair processes such as osteoid osteoma, Garré's osteomyelitis, and stress fracture.

Garré's osteomyelitis produces a densely sclerotic and more widespread response without an accompanying area of radiolucency and can thus be eliminated as a possibility. Osteoid osteoma is classically seen as a larger area of dense sclerosis often surrounding a radiolucent nidus less than 1 cm in diameter, which may in turn contain some calcification. The radiolucency in this case is ill-defined and elongated, while that of osteoid osteoma is fairly well circumscribed and tends to be more rounded. Moreover, the absence of extensive dense sclerosis makes intracortical osteoid osteoma an unlikely candidate. The remaining possibility is a so-called stress fracture, the end result of a bone reaction that may

or may not go on to become a fracture. Because of the reactive nature of the lesion, its radiographic appearance varies with the time of its discovery. Components of the lesion are (a) periosteal new bone formation, (b) a vague intracortical radiolucency, and (c) endosteal new bone formation. In the tibia, the preferred location is the junction of the middle third of the diaphysis with either the proximal or distal third.

CLINICAL HISTORY

The patient noted the onset of dull aching over the anterior medial border of the right tibia during the third and fourth week of basic military training. The pain was only partly relieved by aspirin and was aggravated by activity. There was no history of trauma, and local swelling noted during basic training had subsided at the time of biopsy.

There was slight tenderness on palpation of a firm, raised area overlying the lesion. Laboratory data were noncontributory. Concern about possible malignancy resulted in surgical resection of the lesion.

PATHOLOGICAL FINDINGS

Multiple cross-sections of the tibia showed striking periosteal reactive bone formation and multiple holes in the original cortex (Fig. 3). These holes, as well as the confluent lytic defect seen in Figure 4, represent areas of cortical resorption by osteoclastic activity. This correlates with the radiolucency seen radiographically, which was barely discernible because of the periosteal reaction. Figures 4 and 5 show the radial streamers of reactive periosteal bone which reinforced the weakened cortex and caused the periosteal thickening on x-ray films. Figure 5 also shows the circumferential lamellar patterns of the undisturbed part of the cortex. Figure 6 illustrates a resorption cavity with active osteoclasts. Figure 7 shows multiple small spicules of reactive endosteal bone.

Pathological Diagnosis: Stress fracture of the tibia.

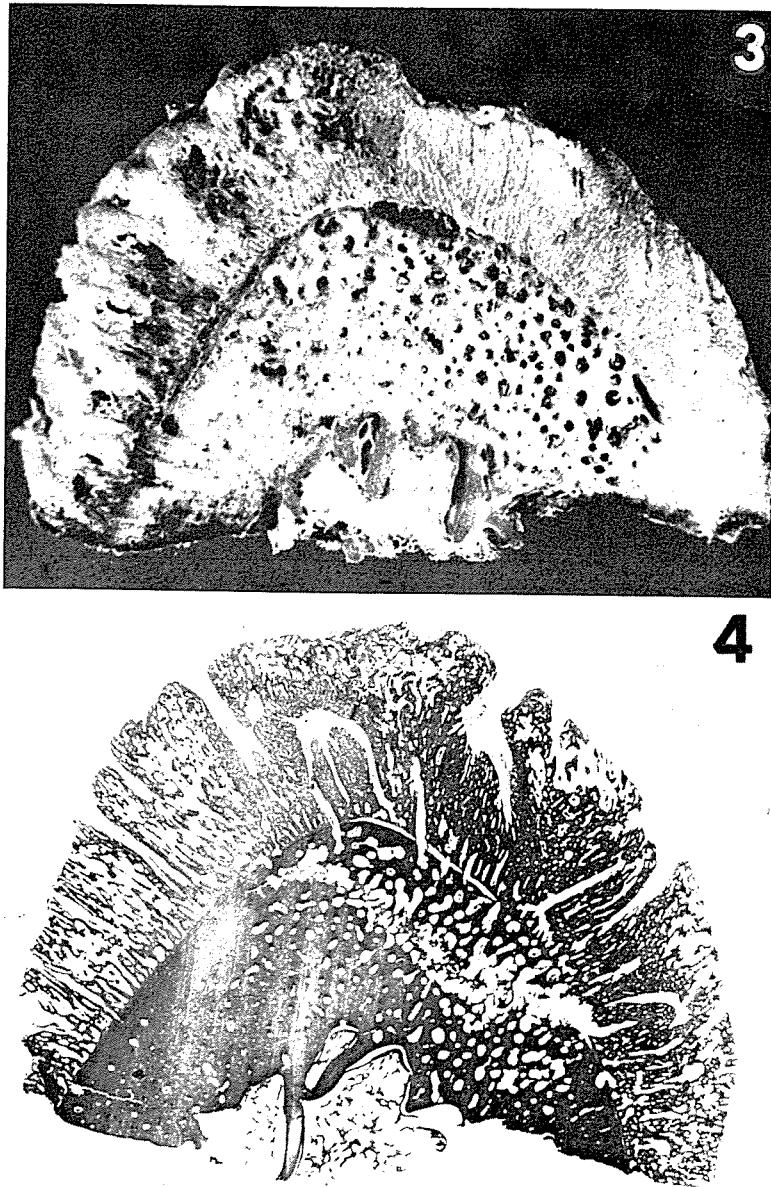


Fig. 3 (AFIP Negative 70-1851-2). The cortex is riddled with multiple holes. A thick periosteal reactive bone formation overlies the area.

Fig. 4 (AFIP Negative 70-1851-1). Multiple small holes merge into a confluent lytic defect in the cortex. Periosteal new bone has been laid down in radial streamers to reinforce the weakened cortex.

DISCUSSION AND CORRELATION

Stress or fatigue fractures are often not true fractures, since there is no loss of structural continuity. They begin at sites of accelerated normal remodeling of cortical bone. During skeletal growth, diaphyseal bone is initially laid down as circumferential lamellae by the periosteum. As the

individual develops, resorption cavities appear, due to the removal of bone by the osteoclasts. These cavities are subsequently refilled with new lamellar bone by the osteoblasts. These replaced units (osteones) are identifiable histologically as haversian systems and are characteristic of adult remodeled bone.

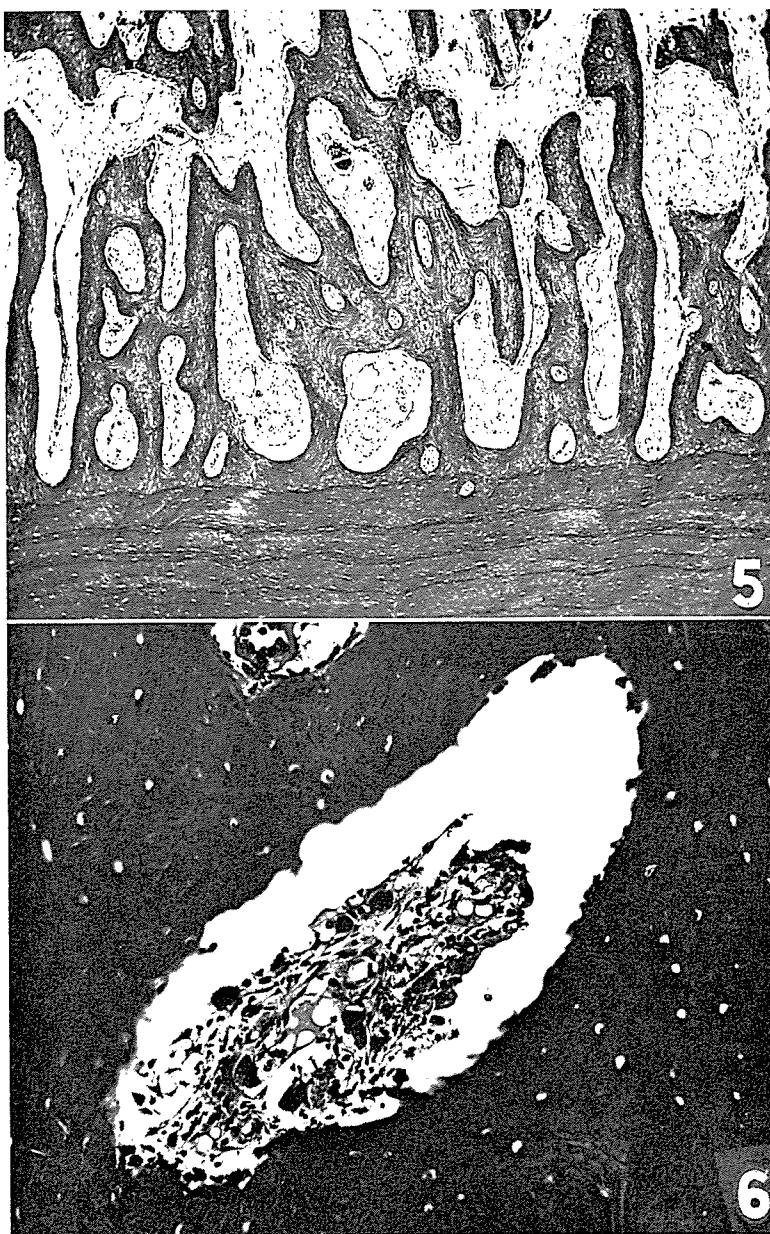


Fig. 5 (AFIP Negative 71-958-1). The radial streamers of periosteal new bone form a striking contrast to the circumferential lamellar patterns of the undisturbed part of the cortex.

Fig. 6 (AFIP Negative 71-958-2). A high-power view of one of the holes in the cortex shows that they represent fields of intense osteoclastic activity.

Osteonal remodeling varies from bone to bone and from one part of a bone to another, depending upon the activity of the patient, since muscle action accelerates the rate of osteonization. Excess use may lead to excessive resorption of bone and focal areas of slight radiolucency, as observed in stress fractures. In the normal sequence of re-

modeling, resorption of bone is followed by replacement. However, replacement is a slow process, whereas resorption proceeds rapidly, producing a temporarily weakened cortex. Sufficient weakening leads to periosteal reinforcement until the refilling process has caught up and solidified the cortex. Tibial stress fractures usually occur at a

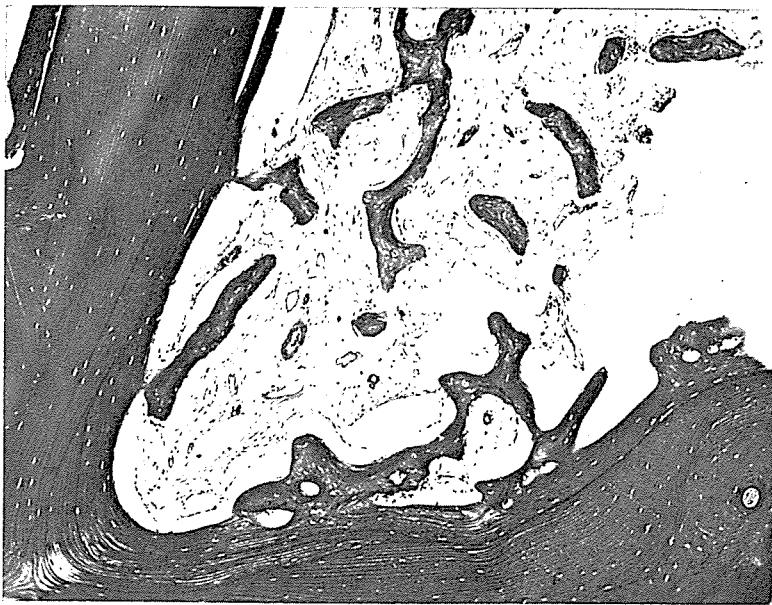


Fig. 7 (AFIP Negative 71-958-3). Multiple small spicules of reactive bone reinforce the endosteal surface of the cortex.

specific site where such osteonal remodeling is laggard and at a time when there is a sudden need for rapid remodeling due to overuse. The remodeled osteonal bone is mechanically superior to the pre-existing circumferential bone.

Initial symptoms begin with sharply localized pain and tenderness brought on by activity. Striking osteoclastic resorption of cortical bone is the initial stage in the osteonal remodeling sequence. In a few days, the cortex may be riddled with resorption cavities (Figs. 3 and 4). This phase continues for two to three weeks. Periosteal reinforcement (Figs. 4 and 5) appears during the second week and reaches a peak at about six weeks. Refilling of the resorption cavities begins at about three or four weeks and may take months to be completed. If a fracture develops, it usually occurs during the third week at the peak of bone loss and before there is sufficient reinforcement. Many of these tibiae do not fracture at all, as in the case reported here. If the mechanical stress is reduced, the rate of resorption slows down so that repair can keep pace. With moderate use, such repair is effective. Hyperostosis, *i.e.*, an excessive periosteal reaction

associated with radiologically marked endosteal reaction, results from repeated periods of overuse without completion of the refilling process between such periods. Once the cortex is fully osteonized, stress fractures will not develop.

Stress is necessary for normal osteone formation. Excessive stress accelerates the normal process; a true fracture occurs only when the removal of cortex is accelerated beyond the capacity of the periosteal reaction to offer adequate reinforcement.

ACKNOWLEDGMENTS: The authors are indebted to Dr. Lent C. Johnson, Chief, Orthopedic Pathology Branch, Armed Forces Institute of Pathology, for the help that made this paper possible.

Registry of Radiologic Pathology
Armed Forces Institute of Pathology
Washington, D.C. 20305

RECOMMENDED READING

1. Burrows HJ: Fatigue infraction of the middle of the tibia in ballet dancers. *J Bone Joint Surg* 38-B: 83-94, Feb 1956
2. Devas MB: Stress fractures of the tibia in athletes or shin soreness. *J Bone Joint Surg* 40-B: 227-239, May 1958
3. Devas MB: Shin splints, or stress fractures of the metacarpal bone in horses, and shin soreness, or stress fractures of the tibia, in man. *J Bone Joint Surg* 49-B: 310-313, May 1967
4. Devas MB, Sweetnam R: Stress fractures of

the fibula; a review of fifty cases in athletes. *J Bone Joint Surg* 38-B:818-829, Nov 1956

5. Elton RC, Abbott HG: An unusual case of multiple stress fractures. *Milit Med* 130:1207-1210, Dec 1965

6. Evans DL: Fatigue fractures of the ulna. *J Bone Joint Surg* 37-B:618-621, Nov 1955

7. Gilbert RS, Johnson HA: Stress fractures in military recruits—a review of 12 years' experience. *Milit Med* 131:716-721, Aug 1966

8. Johnson LC: The kinetics of skeletal remodeling. [In] Structural organization of the skeleton. Birth Defects Original Article Series. Washington, D.C., National Foundation, 1966, pp 120-132

9. Johnson LC: Histogenesis of stress fractures (abst.). *J Bone Joint Surg* 45-A:1542, Nov 1963

10. Johnson LC: Morphologic analysis in path-

ology. Chapter 29. [In] *Bone Biodynamics*, ed by HM Frost. Boston, Little, Brown, 1964, pp 607-609

11. Kroening PM, Shelton ML: Stress fractures. *Amer J Roentgen* 89:1281-1286, Jun 1963

12. Leveton AL: March (fatigue) fractures of the long bones of the lower extremity and pelvis. *Amer J Surg* 71:222-232, Feb 1946

13. Murray DS: Fatigue fractures of the lower tibia and fibula in the same leg. Report of a case. *J Bone Joint Surg* 39-B:302-305, May 1957

14. Pentecost RL, Murray RA, Brindley HH: Fatigue, insufficiency, and pathologic fractures. *JAMA* 187:1001-1004, 28 Mar 1964

15. Wilson ES, Katz FN: Stress fractures. An analysis of 250 consecutive cases. *Radiology* 92: 481-486, 480, Mar 1969

Stress Fracture

RPC of the Month from the AFIP

Maj. DONALD E. SWEET, MC USAR, and Lt. Col. ROBERT M. ALLMAN, USAF MC

ABSTRACT—Stress fractures represent mechanical disturbances and develop over a period of days or weeks, in contrast to traumatic fractures which develop abruptly at the moment of injury. They are generally not fractures in the usual sense of loss of structural continuity but represent a focal acceleration of the normal remodeling process in cortical bone. If bone loss is rapid and excessive, a true fracture may become superimposed.

INDEX TERMS:—Fractures • Stress, fractures

Radiology 99:687-693, June 1971

