Management of orthopedic complications of metabolic bone disease

BERNARD N. STULBERG, MD AND J. TRACY WATSON, MD

■ Orthopedic manifestations of metabolic bone disease are usually related to structural compromise of the skeleton. Fractures due to osteoporosis are the most often encountered problem. In this overview, the authors focus on the commonly encountered fractures related to osteoporosis and discuss concerns and complications related to their management. In addition, they point out the subtle ways in which less commonly encountered metabolic bone diseases, such as osteomalacia, hyperparathyroidism, and Paget's disease, might come to the attention of the orthopedist. Appropriate criteria for medical and surgical management are suggested.

☐ INDEX TERMS: BONE DISEASES, METABOLIC; FRACTURES; OSTEOPOROSIS ☐ CLEVE CLIN J MED 1989; 56:696–703

WHILE the orthopedist is most likely to be involved with traumatic disturbances of bone, he or she must also be fully aware of the major metabolic abnormalities that influence a patient's functional capacity and the bone's structural stability. In this article, we focus on injuries that can be disabling or even life-threatening, as well as costly to the patient, the patient's family, and to society. We discuss the functional and anatomic compromises that occur with fractures, how orthopedic management has evolved to meet the needs of patients with fractures, and the difficulties encountered in fracture management. We also consider areas less commonly perceived as being influenced by metabolic bone disease. We begin with a discussion of the consequences of osteoporosis, or diminished bone volume, and subsequently touch on osteomalacia, hyperparathyroidism, Paget's disease, and avascular necrosis (AVN).

From the Department of Orthopaedic Surgery, The Cleveland Clinic Foundation. Submitted May 1988; accepted Nov 1988.

Address reprint requests to B.N.S., Department of Orthopaedic Surgery, The Cleveland Clinic Foundation, One Clinic Center, 9500 Euclid Avenue, Cleveland, Ohio 44195.

OSTEOPOROSIS

Osteoporosis, or a decrease in bone volume, is a common disorder that decreases the strength of bone. Calcium deficiency is the primary cause of osteoporosis in the elderly, and nutritional studies have demonstrated that most Americans do not ingest enough calcium for their age. Regional loads put certain functional areas at particular risk of fracture. A Colles' fracture associated with moderate trauma indicates an increased risk for subsequent hip fractures. Among women experiencing Colles' fracture at age 70 or older, hip fracture risk is increased by 50%, and the risk of a hip fracture is increased more than sixfold among men with Colles' fracture.[1] Although different types of bone loss may lead to higher incidences of specific types of fractures[2,3] (for example, trabecular bone loss more commonly results in spinal compression fractures, and cortical bone loss is more likely to produce proximal femoral fractures), we focus on the problems associated with the most common frac-

A,B

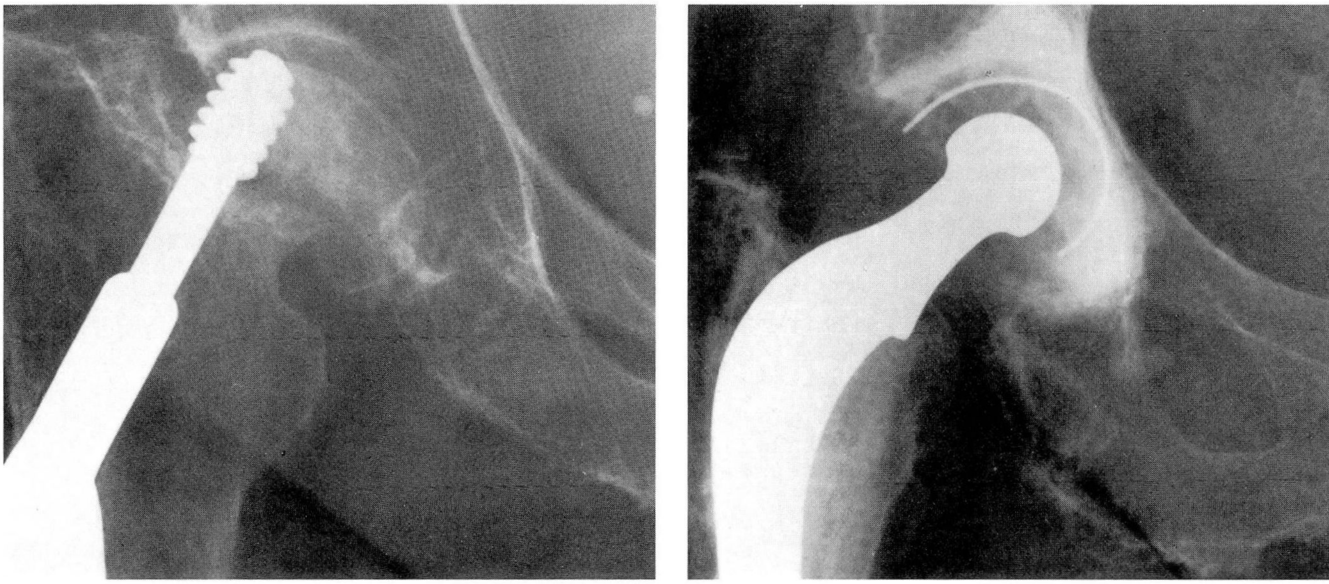


FIGURE 1A. Failure of internal fixation of displaced femoral neck fracture. This radiograph demonstrates nonunion of the fracture with subsequent slippage of femoral head and protrusion of the hip screw. FIGURE 1B. Conversion to cemented total hip replacement has allowed pain-free function for this 72-year-old woman.

tures. These are fractures of the wrist, spine, pelvis, and proximal femur.

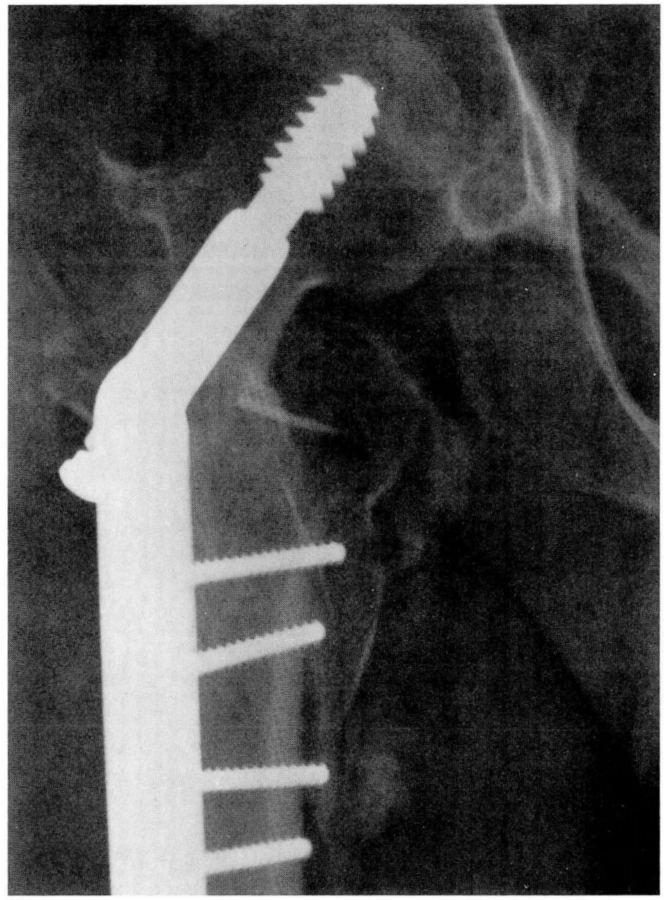
Wrist fractures

Fractures at the wrist are most commonly encountered when a patient falls on an outstretched hand. The wrist is dorsiflexed, and the forces are transmitted to the distal part of the radius. The most common fracture encountered is the Colles' fracture. This fracture may extend into the joint, be associated with an injury to the distal ulna, and can result in shortening, angulation, and dorsal displacement of the wrist. The most common difficulty in treating this fracture is trying to maintain the length of the wrist joint while maintaining mobility at the wrist. Treatment is usually by manipulation and casting. If healing potential is normal, then casting for six weeks should be sufficient. However, some fractures are quite comminuted, making it difficult to hold the fracture in the correct position. External fixation with pins in the bones on each side of the fracture may be required. The surgeon's difficulty is in judging when immobilization can be discontinued so that motion may be started. An orthopedist may accept a cosmetically less satisfactory result to obtain a functionally adequate one. Complications seen with this fracture include malunion, median nerve compression, reflex sympathetic dystrophy, stiffness, shoulder-hand syndrome, weakness, and tendinous adhesions, as well as surgical complications related to pin fixation (if used).[4]

It is important to realize that a triad of injuries may coexist in a patient with Colles' fracture. Humeral head tuberosity avulsion fractures as well as surgical neck fractures of the humerus may be overlooked in someone falling on an outstretched hand. A minimally displaced femoral neck fracture may also be present and should not be missed.

Spine

Compression fractures of the spine most commonly occur in the area of the lower thoracic or the upper lumbar spine at the thoracolumbar junction. These fractures are usually wedge-type fractures, are caused primarily by flexion movements of the spine, and are stable fractures.[5] The risk of associated neurologic injury is small. The most common vertebral bodies involved are those between T-10 and L-2. Typically, the patient has had a a minimal fall, landing directly on the back or buttocks. Pain is usually in the midline of the back. Patients may be unable to move, preferring to lie flat on the back. Examination reveals tenderness, occasionally marked, in the midline of the spine over the spinous process of the involved vertebrae. Neurologic examination shows no evidence of spinal cord or nerve root impingement. Bed rest is the mainstay of treatment. Narcotic analgesics



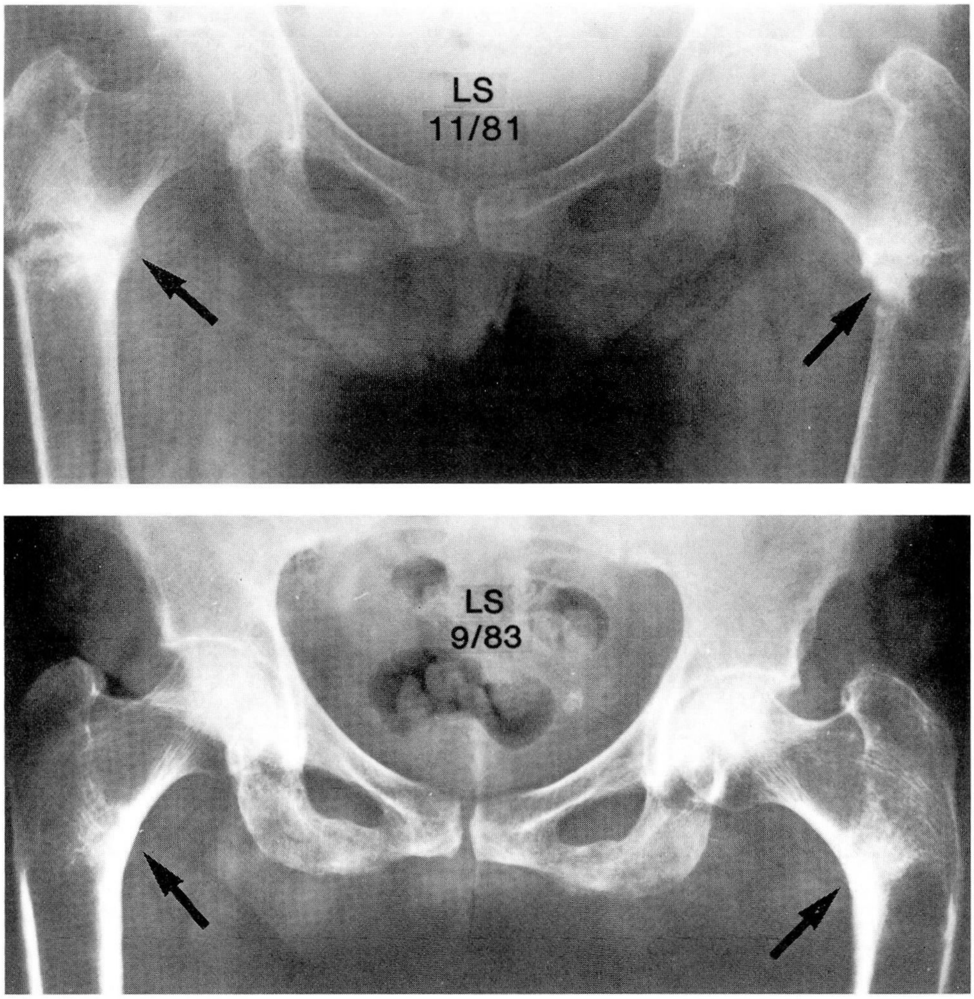
A,B
C

FIGURE 2A. A substantially comminuted intertrochanteric fracture with a subtrochanteric extension. FIGURE 2B. Internal fixation allowed for early stabilization and mobility. FIGURE 2C. The fixation used was insufficient for this man's activity level and healing potential. Fixation and stabilization were lost.

should be kept to a minimum, and careful nursing care to avoid bed sores is required. During the period of immobilization, deep vein thrombosis, ileus, and urinary retention must be prevented. When possible, usually five to seven days following injury, patients should begin physical therapy for progression to ambulation. A walking aid, such as a walker, is required. Occasionally a patient will require a back brace or corset. This should not be used for more than a few weeks. Back exercises should be started as soon as tolerated. Most fractures should heal within six weeks. Therapy is aimed at preventing further fracturing and includes stretching and strengthening exercises for the abdominal and lumbar muscles.

Pelvis

Fractures around the hip generally involve fractures of the proximal femur and can result in serious disability. They should be differentiated from the commonly encountered fracture of the pelvis, which also immobilizes the patient. Pelvic fractures in the osteoporotic patient usually occur in the anterior aspect of the pelvis, unlike the pelvic fractures associated with high-energy impact injuries, which can result in substantial vascular or neurologic injury. In the osteoporotic patient, however, these pelvic fractures are usually confined to the ischium or pubis and are often unilateral, but may involve a "straddle fracture," a bilateral fracture of superior and inferior pubic rami.[6] This area does not bear much weight. The fractures are stable. No vascular or neurological sequelae are likely. Treatment is bed rest until symptoms subside sufficiently to allow ambulation. Early displacement of the fracture can occur, and protected ambulation with partial or nonweight-bearing methods should be emphasized for the first three to four weeks. Even in the severely osteoporotic patient, recovery within four to six weeks is to be expected. Only occasionally will these fractures fail to unite.[7]



A
B

FIGURE 3A. This patient was referred for treatment of bilateral subtrochanteric fractures. Transverse and broad fracture lines are characteristic Looser's zones of osteomalacia. **FIGURE 3B.** Correction of mild renal abnormalities and calcium and vitamin D supplementation allowed healing and ambulatory status for this formerly sedentary woman.

Proximal femur fractures

Fractures of the proximal femur are of two types: either fractures of the femoral neck or fractures of the intertrochanteric region of the proximal femur. It is necessary to differentiate between these fractures because of the nature of the blood supply to the proximal femur and because that blood supply may be compromised by the fracture. Treatment will differ for these two fractures because of the differing potential for disrupting the blood supply with subsequent compromise of healing or loss of blood flow to the femoral head.

Treatment options in fractures of the femoral neck vary with the degree of displacement of the fracture and the obliquity of the fracture line. The greater the displacement, the greater the disruption of the blood supply. Garden's classification is the most commonly used classification of these fractures. Garden type I and type II fractures have minimal displacement. These fractures can be fixed in position with screws, pins, and plates with a high degree of certainty that the fracture will heal and that AVN (segmental collapse) will not occur. In Garden type III and IV fractures, displacement is substantial, loss of blood supply is more likely, and the chance of nonunion or AVN is greater (*Figure 1*). The orthopedist may be faced with difficult decisions with these fractures. In a younger patient, all efforts should be made to preserve the femoral head. Thus, reduction and

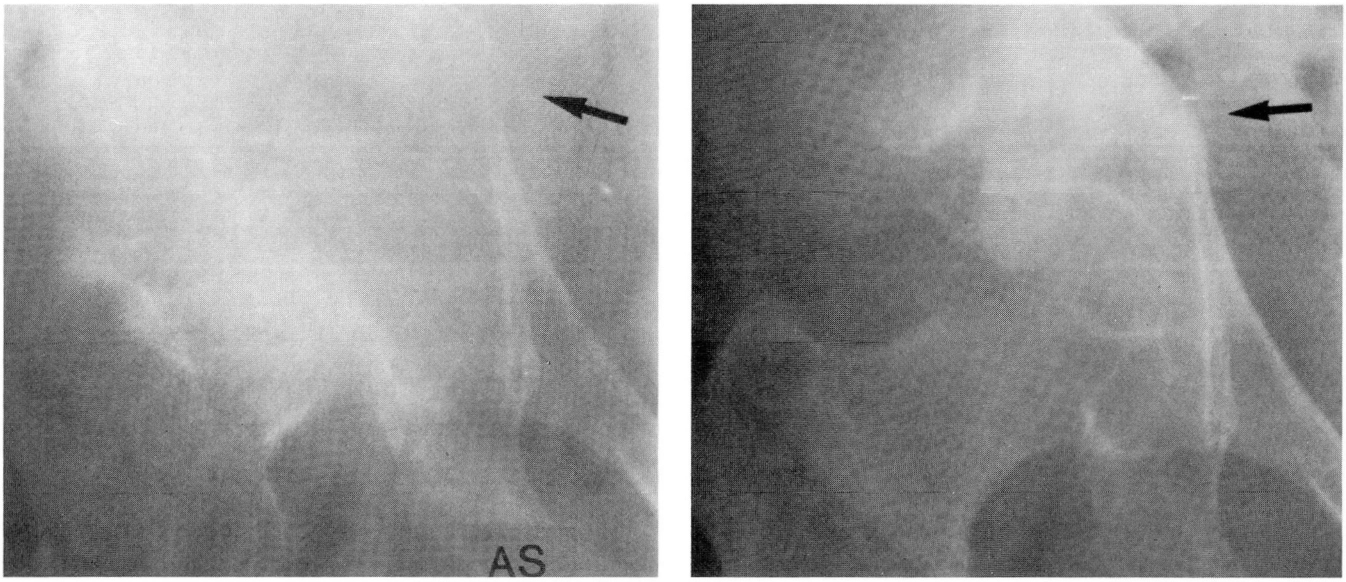


FIGURE 4A. This patient experienced acute pain in her right hip one week before scheduled parathyroidectomy. Fracture through a brown tumor in the right ilium above the acetabulum was treated by bed rest. FIGURE 4B. Eight months following parathyroidectomy the bony skeleton had recalcified and the fracture had healed. The patient was fully ambulatory without walking aids.

fixation of these fractures, supplemented by bone grafting, is appropriate. However, recent analysis from a multicenter study of femoral neck fractures in younger patients revealed AVN in 29% and nonunion in 18%, regardless of the method of treatment.[8] For elderly patients, replacement of the femoral head with artificial devices may be more appropriate. The disadvantage of the increased magnitude of the surgery required is offset by the early mobility that is possible. Surgical complications of internally fixed fractures include inadequate reduction, inadequate fixation, loss of fixation or reduction, late segmental collapse, and nonunion.[9,10] Secondary prosthetic replacement after AVN or nonunion has shown excellent results with healthy patients.

Intertrochanteric fractures occur in an area below the critical blood supply to the femoral head and in an area of bone that can heal rapidly. These fractures are usually treated by internal fixation, using rods, screws, or plates according to the preference of the surgeon. These fractures can heal without surgery, but prolonged bed rest would be necessary and deformity of the bone would be likely.

When these fractures are comminuted, stabilizing them with orthopedic devices can be complex. Surgical complications, such as delayed wound healing, loss of reduction, loss of fixation, infection, delayed union, and malunion, indicate the difficulties facing elderly patients with compromised bone (*Figure 2*).

Studies have clearly identified the role of calcium in fracture healing. The usual source of calcium for fracture healing is the diet. If a person's diet is chronically deficient in calcium, mineralization must be achieved by cannibalizing the remaining skeleton, thus increasing the risk of fracture. It is, therefore, suggested that patients recovering from large bone fractures take supplemental calcium to meet at least the normal calcium requirement for their age group. Postmenopausal women and similarly aged men require 1500 mg calcium per day.[2]

Although the orthopedic surgeon's involvement in treating metabolic diseases of bone is mostly in managing the problems of osteoporosis, the surgeon's input may be needed for other problems as well.

OSTEOMALACIA

Osteomalacia is the inability to calcify the osteoid tissue that rims the bone. This condition can exist with normal bone volume or with substantial osteoporosis. It is important for the clinician to be aware of this entity. When fractures will not heal, osteomalacia may be present. It may also be present in patients undergoing renal

A,B

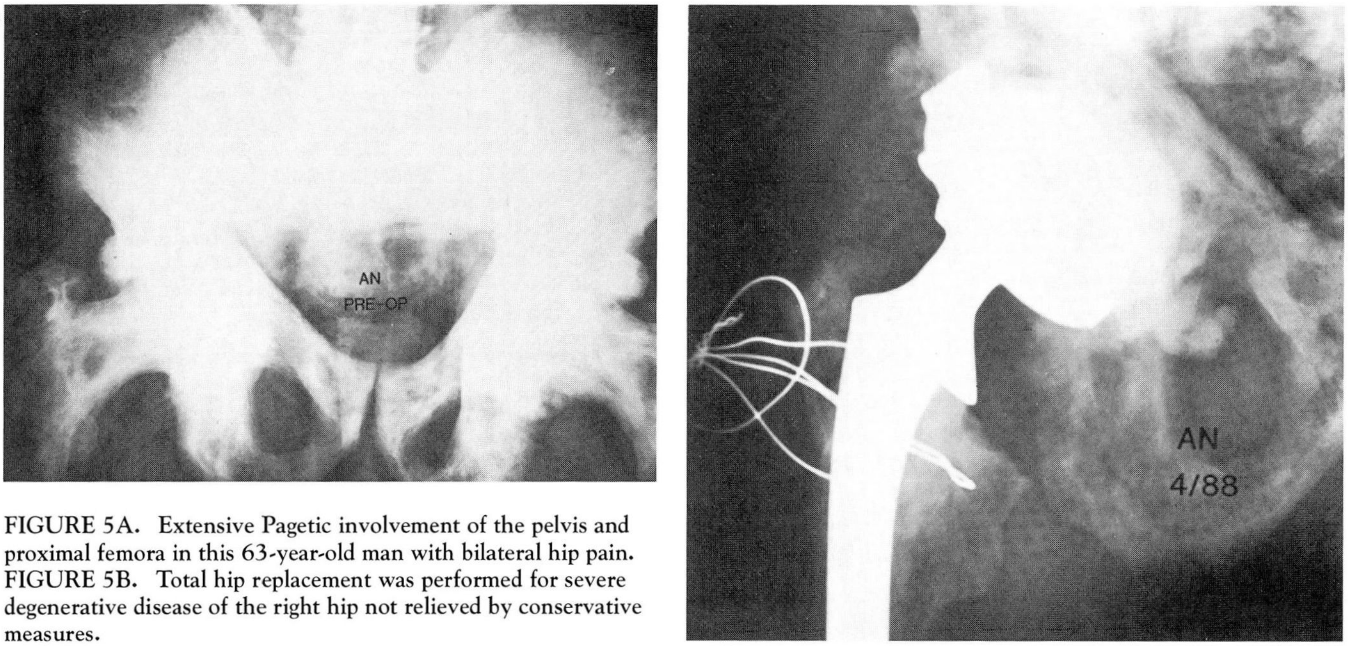


FIGURE 5A. Extensive Pagetic involvement of the pelvis and proximal femora in this 63-year-old man with bilateral hip pain. FIGURE 5B. Total hip replacement was performed for severe degenerative disease of the right hip not relieved by conservative measures.

dialysis who have repeated fracturing or persistent "bone pain." It may occur subtly in patients with mild chronic renal failure and produce gradual loss of ambulatory capacity, or it may be present in patients with systemic illness who develop AVN (*Figure 3*). The surgeon's role is to be alert to these possibilities, to encourage appropriate medical correction of the problem, and to assist in the diagnosis of the condition through iliac crest biopsy, if necessary. If surgery is required for mechanical compromise, it should be performed after the metabolic abnormality is corrected.[11]

Hyperparathyroid bone disease

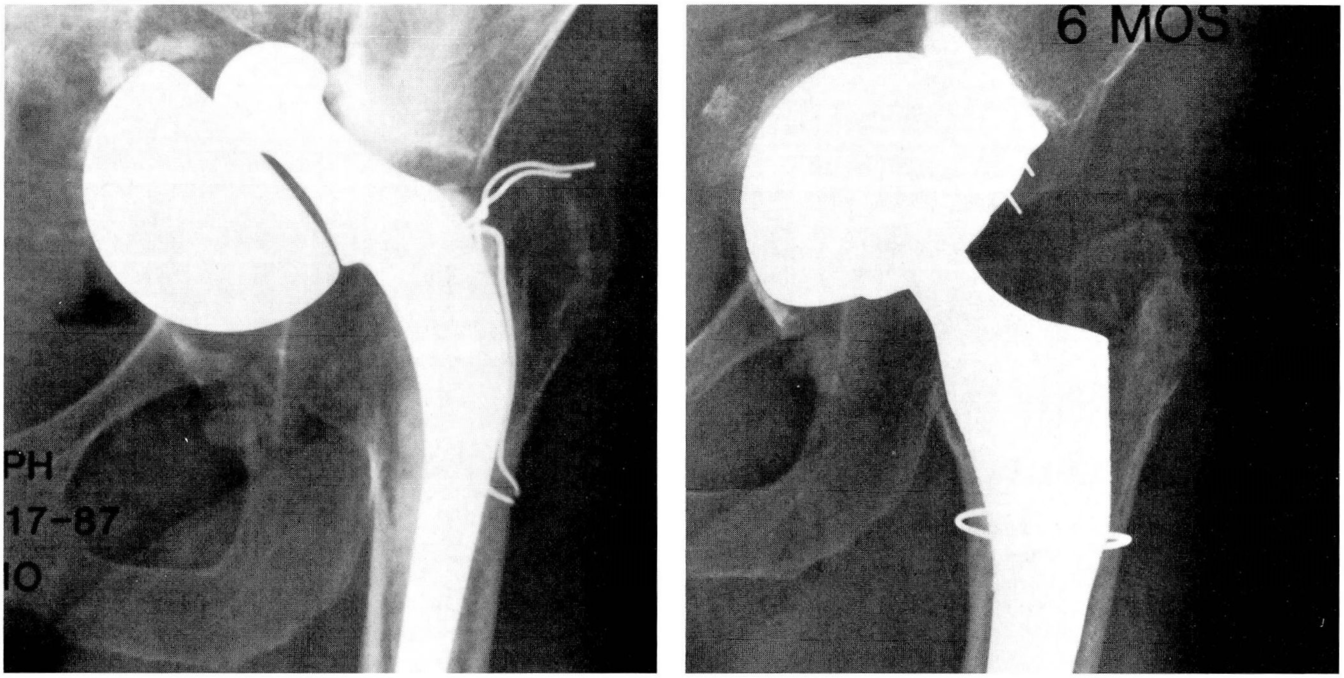
The orthopedic surgeon's role in the treatment of hyperparathyroid bone disease is usually secondary. Difficulties do arise if the condition is unrecognized or if the patient experiences mechanical compromise before the abnormality is corrected.[12] This occurs when a patient presents with bone pain in a weight-bearing area, particularly near a joint. Parathyroid hormone increases calcium resorption directly from bone by activating osteoclastic and osteocytic osteolysis as well as increasing the numbers of osteoclasts overall. This increase in osteoclasts can localize to an area in bone and replace the normal bone elements, producing a "brown tumor." Brown tumors can substantially compromise the mechanical strength of bone, and microfractures can occur. Orthopedic management focuses on diminishing forces across these areas. Surgery is rarely necessary and should not be performed until metabolic abnormalities are fully corrected (*Figure 4*).[13,14]

Paget's disease

A third metabolic bone disease that can involve the orthopedist is Paget's disease, which involves substantial increase in bone turnover (substantially increased bone resorption) and the inability of bone to remodel appropriately.[15] The change in stiffness of the bone leads to changes in the direction of loads across the joint surface, which can result in arthritis. Joint pain can be caused by either the Paget's disease or the arthritis, and appropriate therapeutic decisions can be difficult. Medical management of Paget's disease should precede any surgical intervention. If surgical intervention becomes necessary, extensive blood loss can occur and must be planned for. Deformity of the bone may also require special techniques or devices. Flexible intramedullary rods are preferred over plates and screws for long-bone fractures in patients with metabolic bone disease. Inability of bone to hold the fixation device leads to frequent complications of screw pull-out, plate fracture, and subsequent nonunion. Patients should be aware of these problems, even though results of surgical intervention, particularly at the hip and knee, can be as successful as those in patients without Paget's disease (*Figure 5*).[16,17]

Other areas

In several other areas of orthopedic surgery the in-



A,B

FIGURE 6A. 32-year-old woman with steroid-dependent rheumatoid arthritis and a failed revision of an acetabular component. Original hip replacement was performed in 1974. FIGURE 6B. Six months following revision to a cementless total hip replacement with acetabular bone grafting. Iliac crest biopsy revealed osteomalacia. Patient's graft healed after appropriate medical therapy was instituted.

fluence of metabolic bone disease is poorly understood, but these areas are now being studied using newer diagnostic techniques of bone density measurement and bone turnover measurement.[18–20] We have been using the techniques of bone densitometry and iliac crest biopsy to evaluate the results of AVN treatment, cementless total joint replacement, and revision of failed total hip replacements. These investigations have shown substantial subclinical bone turnover abnormalities in patients with AVN and have demonstrated an unexpectedly high incidence of osteoporosis in patients undergoing cementless total hip arthroplasty.[13] Patients undergoing revision arthroplasty have occasionally shown dramatic and previously unsuspected bone abnormalities that are correctable (*Figure* 6).

In summary, orthopedic surgeons can focus on the functional and anatomic compromises that patients suffer as a result of metabolic bone disease. Armed with an understanding of the interplay of metabolic and functional demands, the orthopedic surgeon can offer important perspectives on preserving and maintaining physiologic performance.

REFERENCES

1. Owen RA, Melton LJ, Ilstrup DM, Johnson HA, Riggs BL. Colles' fracture and subsequent hip fracture risk. Clin Orthop 1982; **171**:37–43.
2. Lane JM, Vigorita VJ. Osteoporosis in metabolic bone disease. Orthop Clin North Am 1984; **15**:711–728.
3. Lane JM. Metabolic bone disease. [In] Sculco TP, ed. Orthopaedic Care of the Geriatric Patient. St. Louis, C.V. Mosby Co., 1985, pp 235–268.
4. Dobyns JH, Linscheid RL. Fractures and dislocations in the hand. [In] Rockwood C, Green D, eds. Fractures. Philadelphia, J.B. Lippincott, 1975, pp 345–385.
5. Kaufer H. Fractures and dislocations of the spine. [In] Rockwood C, Green D, eds. Fractures. Philadelphia, J.B. Lippincott, 1975, pp 817–897.
6. Tile M. Disruption of the pelvic ring. [In] Tile M, ed. Fractures of the Pelvis and Acetabulum. Baltimore, Williams and Wilkins, 1984, pp 22–35.
7. Kane WJ. Fractures of the Pelvis. [In] Rockwood C, Green D, eds. Fractures. Philadelphia, J.B. Lippincott, 1975, pp 905–975.
8. Kyle RF, Swiantkowski M, Segal D, et al. Young femoral neck fracture. Presented at 51st Annual Meeting of AAOS, Atlanta, 1984.
9. Winter WG, Clawson DK. Complications of treatment of fractures and dislocations of the hip. [In] Epps CH, ed. Complications in Orthopaedic Surgery. Philadelphia, J.B. Lippincott, 1978, pp 403–412.
10. Clawson DK, Melcher PJ. Fractures and Dislocations of the Hip. [In] Rockwood C, Green D, eds. Philadelphia, J.B. Lippincott, 1975, pp

1012–1056.
11. Doppelt SH. Vitamin D, rickets and osteomalacia. Orthop Clin North Am 1984; **15:**671–686.
12. Chalmers J, Irvine GB. Fractures of the femoral neck in elderly patients with hyperparathyroidism. Clin Orthop 1988; **229:**125–130.
13. Stulberg BN, Bauer TW. Bone remodelling capacity and uncemented total hip arthroplasty. Transaction of the Orthopaedic Research Society 1988; **13:**551.
14. Stulberg BN, Licata AA, Bauer TW, Belhobek GH. Hyperparathyroidism, hyperthyroidism, and Cushing's disease. Orthop Clin North Am 1984; **15:**697–710.
15. Merkow RL, Lane JM. Current concepts of Paget's disease of bone. Orthop Clin North Am 1984; **15:**747–764.
16. Merkow RL, Pellicci PM, Hely DP, et al. Total hip replacement for Paget's disease of the hip. J Bone Joint Surg 1984; **66a:**752–758.
17. Stauffer RN, Sim FH. Total hip arthroplasty in Paget's disease of the hip. J Bone Joint Surg 1976; 58A:476–478.
18. Vigorita VJ. The tissue pathologic features of metabolic bone disease. Orthop Clin North Am 1984; **18:**613–629.
19. Schneider R. Radiologic methods of evaluating generalized osteopenia. Orthop Clin North Am 1984; **15:**631–651.
20. Resnick D, Niwayama G. Diagnosis of bone and joint disorders with emphasis on articular abnormalities. [In] Resnick D, Niwayama G., eds. Osteoporosis. Philadelphia, W.B. Saunders 1981, pp 1638–1681.