RADIOLOGY OF OSTEOPOROSIS EVALUATION AND INTERPRETATION

Alan S. Banks, D.P.M.
Brad Castellano, D.P.M.

Introduction

Loss of bone density is a common incidental radiographic finding in elderly, post-traumatic, and postoperative patients. However, a more recent understanding of the significance of osteoporosis has led to intense research to determine if simple radiographs can be helpful in evaluating osteoporosis. The primary care physician can play a large role in identifying patients at risk of developing the complications of fragile bones. Additionally, surgeons should be aware of the problems associated with osteoporosis following surgical or post-traumatic immobilization of an extremity. Understanding the etiology, radiographic appearance, and differentiation of the various conditions causing a loss of bone density will aid the clinician in choosing a rational treatment for his patient.

Determination of Bone Density

Many non-invasive methods of evaluating bone mineral density have been developed. Some of these methods have proven to be quite objective and to provide data which can be used to accurately predict the amount and rate of bone loss. Unfortunately the majority of these methods are quite involved and expensive. Accurate, practical, and readily available techniques of determining bone density are needed to identify those individuals suffering from or at risk of developing osteopenic conditions.

The methods presently being used to determine bone density may be divided into three categories. These categories are:

1. Quantitative methods
2. Semi-quantitative methods
3. Qualitative methods

Quantitative Methods

Quantitative methods are those which assign a relatively objective numerical value to the bone density. These techniques include radiogrammetry, radiographic photodensitometry, computed tomography, single and dual photon absorptiometry, and compton scattering (1-4).

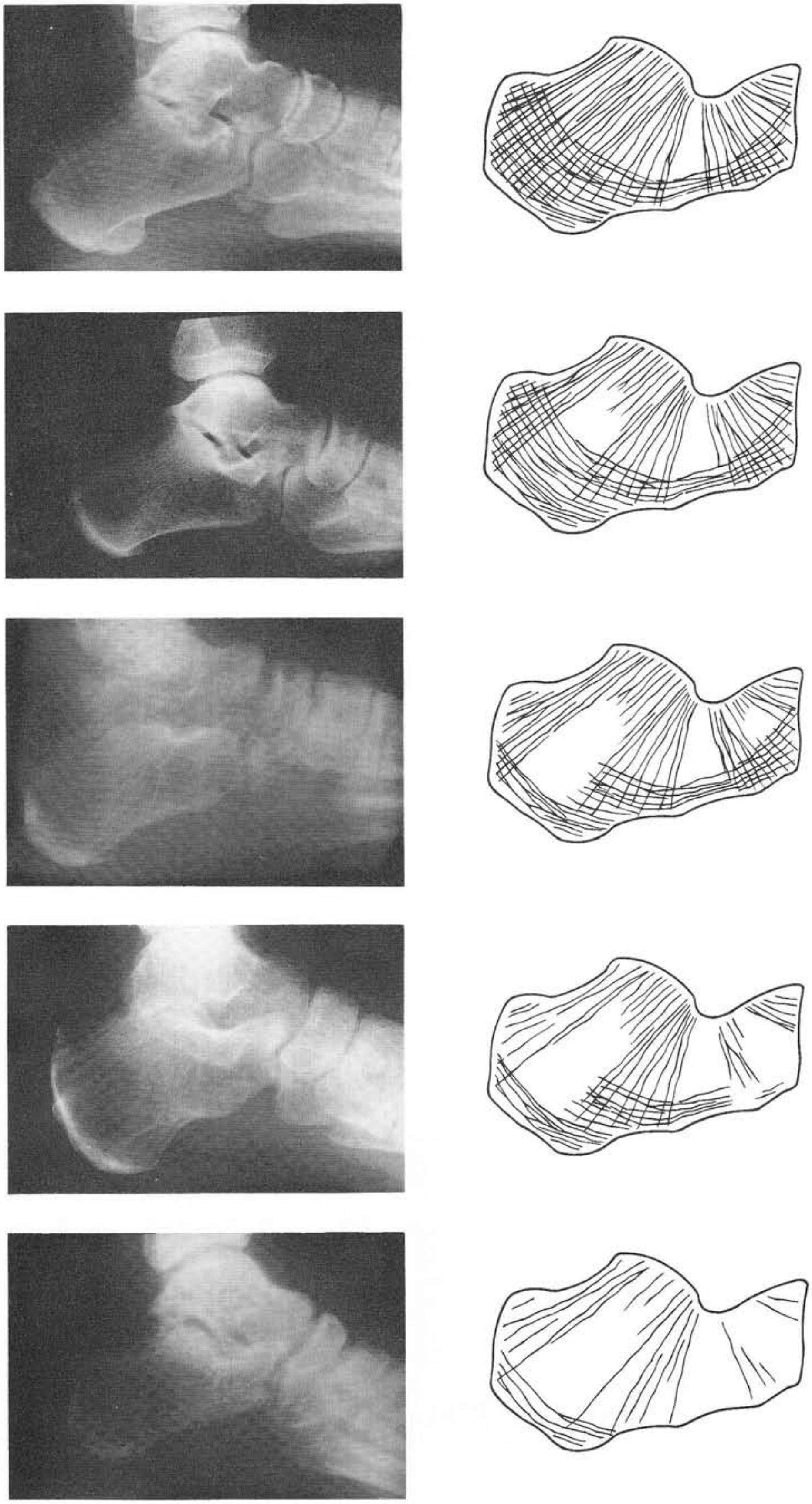
The methods for the most part are impractical for routine examination and presently are available only in limited numbers of research centers. It is important for the physician to be aware of these techniques should there be need for further evaluation of a patient.

Semi-quantitative Methods

Semi-quantitative methods of grading and diagnosing osteopenia are readily available to the physician via simple radiography. Singh and associates (5, 6) described a method of grading osteoporosis utilizing the radiographic patterns of trabecular bone in the head and neck of the femur. They found that the major trabeculae in the proximal femur dissolved in a fairly predictable and sequential manner as the osteoporosis increased in severity. The trabeculae in the femur and other weight-bearing bones are aligned to resist both tensile and compression forces. Tensile trabeculae were found to be the first lost in early senile osteoporosis followed by compression trabeculae in more latent disease.

A similar method was described by Jhamaria and associates (7) utilizing the trabecular pattern of the calcaneus as an index of osteoporosis. The calcaneus was felt to be a more accessible weight-bearing bone for radiographic study. The ease of positioning diminished soft tissue and increased distance from the gonads were felt to be advantages to this procedure as compared to Singh's method (Fig. 1).

The degree of osteoporosis identified by Singh's index was found to correlate with existing spinal disease and with iliac crest biopsies. Jhamaria's index demonstrated significant agreement with Singh's index and was well correlated with the age of the patient. Other authors have supported these findings (8, 9). Still others have disputed the significance of these types of determinations noting



A. Grade V. Normal abundant trabecular bone is present throughout with a normal distribution

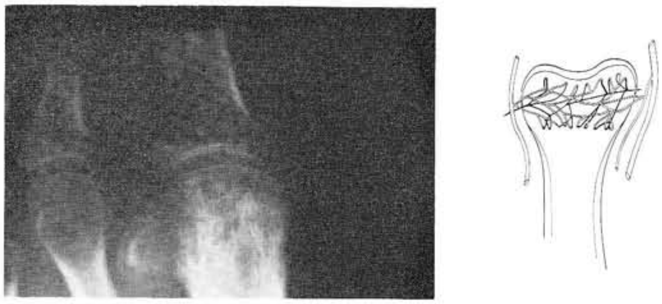
B. Grade IV. A wedge shaped defect is noted between two bands of posterior compression trabeculae. This is a normal variant.

C. Grade III. Represents borderline osteoporosis. Recession of the posterior tensile trabeculae which cross only the anterior band of the posterior compression trabeculae.

D. Grade II. Definite osteoporosis is seen. Anterior tensile trabeculae have disappeared and the posterior tensile trabeculae have recessed further.

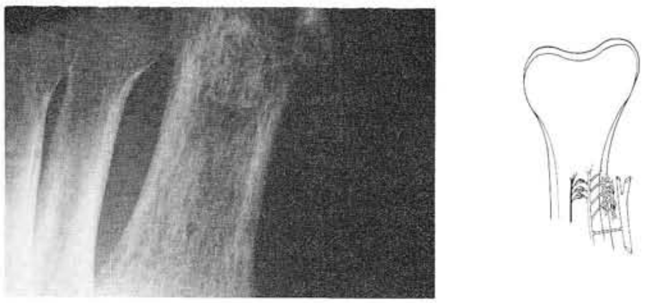
E. Grade I. Severe osteoporosis. Complete absence of tensile trabeculae is present. Compression trabeculae are reduced in number.

Fig. 1. Trabecular patterns described and graded by Jhamaria and associates were felt to represent increasing degrees of osteoporosis.



A. Epiphyseal vessels supply the subchondral bone plate and adjacent trabecular bone. Activation of this vascular network results in juxta-articular resorption.

B. Metaphyseal vessels perfuse the cancellous area of the neck of the long bone. Band like resorption across the metaphysis is seen with hyperemia induced osteopenia.



C. Periosteal and nutrient vessels supply the outer and inner cortical surfaces respectively. Intracortical, subperiosteal, and endosteal resorption are affected by these vessels.

Fig. 2. Circulatory fields of the typical long bone are shown. Isolated or combined patterns of hyperemia will cause predictable osteopenic morphology in the affected bone. Adapted from Allman and Brower, 1981.

Fig. 3. Anterior wedging of the second lumbar vertebrae is typical of the compression fractures suffered by patients with senile osteoporosis.

Fig. 4. Severe diffuse osteopenia is seen in this bedridden patient ana is typical of disuse osteoporosis.

Fig. 5. This 24 year old patient suffered a calcaneal fracture ten weeks prior to this examination. The serious spotty osteolysis with prominent juxta-articular involvement is very typical of reflex sympathetic dystrophy.

a poor correlation with quantitative methods and significant inter and intra-observer variation in grading values (10-12).

Qualitative Methods

Qualitative methods of determining bone density utilize simple radiographs, high detail radiography, and radionuclide bone scanning. These methods can be used to form a fairly accurate differential diagnosis. Sequential examination to assess treatment and the progression of the osteopenic condition can also be carried out.

Radionuclide bone scans utilizing technetium-99m are readily available in most nuclear medicine departments. Technetium-99m has a relatively short half-life of six hours. Ten to twenty millicuries are injected intravenously into the patient and images are subsequently produced in two to four hours. This dosage results in approximately 0.2 rads to the entire body. This is actually less than the dose received for a series of lumbosacral spine radiographs. By three hours after the injection 30% to 40% of the radionuclide is found in bone tissue.

Uptake of Technetium-99m is dependent upon the metabolic activity of the bone as well as bone blood flow. Areas of rapid bone turnover with high blood flow rate will exhibit increased uptake. Low flow rate and diminished metabolic activity will show a decrease in uptake.

A generalized decrease of technetium uptake is seen in patients with postmenopausal osteoporosis. However, this is a normal presentation in the older patient and therefore cannot be used to diagnose osteoporosis. Generalized osteopenia due to diseases causing high bone turnover rates such as renal osteodystrophy or osteomalacia, can be differentiated from senile osteoporosis by a bone scan showing prominent skeletal features (13).

Disuse and post-traumatic osteoporosis may on occasion mimic malignancy (14, 15). Such cases might easily be distinguished by the use of radionuclide scanning. Rapid destruction by lytic lesions will of course reveal an area of increased radionuclide uptake. The gradual process of disuse will show a less intense picture of bone metabolism.

Scintigraphic studies have also been used to evaluate patients with reflex sympathetic dystrophy. Kozin and associates (16) found that bone scans were helpful in distinguishing true reflex sympathetic dystrophy from other disorders with similar radiographic appearances. Increased periarticular uptake was found to be fairly specific for these patients. Further more, a high correlation between patients with good response to therapy and a positive bone scan was noticed.

Microradioscopy was first described by Meema (17) and utilizes fine detail radiography with optical magnification to describe resorptive patterns in bone. Patterns of abnormal bone resorption and porosity were found to be useful in differentiating thyrotoxicosis from senile osteoporosis. Later this method was used to analyze various other metabolic bone diseases (18, 19).

The technique involves imaging of the metacarpal on industrial grade film. This film has a small photographic grain size and therefore, produces a high resolution radiograph. This radiograph is then viewed through a 6 to 8 x magnifier. The result is a bone image comparable to low grade microscopy.

Patterns of bone resorption in general reflect the intensity of the metabolic process. Acute, high bone turnover tends to resemble chronic forms of osteopenia as it progresses to end stage disease. Five microradiographic patterns of bone resorption have been described.

1. *Trabecular resorption* is evidenced by metaphyseal and vertebral osteopenia. Patchy distribution of this form of resorption is seen in disuse types of disease. Tensile trabeculae are first to be lost followed by compression trabeculae.
2. *Subperiosteal bone resorption* is a common but less dramatic form of rarefraction and is seen in all forms of osteopenia.
3. *Endosteal osteoclastic activity* causes inner cortical surface resorption. This results in scalloping of the cortical surface. This pattern is primarily seen in more acute disease processes. Smooth thinning of the cortices is seen with chronic periosteal and endosteal resorption.
4. *Subchondral and juxta-articular bone resorption* have been described in many conditions which typically present with a high degree of associated arthropathy. Reflex sympathetic dystrophy and hyperparathyroidism are noteworthy examples. These aggressive forms of osteopenia may be distinguished from more common causes of this type of osseous erosion, i.e. rheumatoid arthritis, by absence of joint space narrowing.
5. *Intracortical absorption of bone* suggests a high bone turnover process (21). This form of rarefraction has been studied extensively by many authors (14, 17-19). Microradiographic evidence of striations or tunnels within the cortices is a hallmark of this form of osteopenia.

Striations are present to a clinically insignificant degree in normal individuals. They are formed by the continuing process of haversian canal remodeling and are also known as "cutting" and "closing" cones (21). Generally this pattern of resorption is seen only in more aggressive, rapid forms of osteopenia. It is not commonly associated with senile osteoporosis or true disuse osteoporosis (19, 22). Some authors have described intracortical resorption in disuse osteoporosis however, their examples were actually post-traumatic patients who should be differentiated from true disuse (14). Though not specific for reflex sympathetic dystrophy intracortical striations are seen in it most severe expression in this condition (20).

Patterns of Osteopenia

Bone is a dynamic structure which is influenced by many mechanical, hormonal, and nutritional factors. However, hormones and nutrients which do not reach their target organ cannot have an effect on that organ. Avascular necrosis demonstrates quite nicely the effects of decreased and increased circulation. Infarcted bone undergoes little structural change while the adjacent bone is rapidly dissolved by the hyperemic state resulting from the lysis of cells in the infarcted bone.

Disuse osteoporosis might seem to be a completely mechanically induced effect which results in bone resorption. However, this has been proven incorrect. In a classic study Burkhart and Jowsey (23) demonstrated the importance of circulating parathyroid hormone in mediating bone resorption. They found that osteoporosis did not develop in parathyroidectomized animals even though immobilization and hyperemia had been induced. This would seem to prove that circulating parathyroid hormone is required to cause the resorption seen in disuse atrophy. Secondly, hyperemia seems to increase the sensitivity of the immobilized part to this hormone.

Allman and Brower have demonstrated the significance of circulatory influences on the skeletal system (22). They described the effects of hyperemia as related to nutritional circulatory fields. A field was defined by the area of skeletal tissue supplied by a particular vascular network. Nutrient and periosteal arteries make up the network which affects diaphyseal bone. Metaphyseal vessels perfuse the primarily cancellous bone of that respective region. Finally, epiphyseal arteries enter the ends of the long bones supplying the subchondral region.

It has been theorized that hyperemia occurring selectively within one of these circulatory fields results in a distinct pattern of osteopenia which may be recognized on radiographs (22). Although hyperemia confined to one circulatory field can occur, it is more common to have either diffuse or simple combinations. Figure 2 shows the vascular networks in effect in these circulatory fields with examples of osteoporotic patterns affected by each.

Radiographic Evaluation

The earliest radiographic findings in senile or postmenopausal osteoporosis are seen in the vertebral column. Later appendicular skeletal findings of a less severe nature are noted. As much as 30% of the bone mass must be removed to cause the radiographic appearance of osteopenia. Therefore, an osteoporotic calcaneus will often mean the spine is severely involved.

Localized involvement is the most common presentation in the vertebrae. Normal bone density will often be seen adjacent to a compressed vertebral body. A relative increase in the cortical density of the vertebral end plates with loss of transverse (tensile) and later compression trabeculae will be seen.

Stress fractures due to cortical thinning may occur in senile osteoporosis. However, evidence of multiple stress fractures should alert the physician to the possibility of coexisting or primary osteomalacia (24). Characteristic areas of fracture in senile osteoporosis are the vertebral bodies, femoral necks, wrists, and on occasion the calcanei. Fractures of the spine usually appear as anterior wedging of the vertebral body. The majority of these fractures occur at the apex of the thoracic convexity and at the thoracolumbar junction (Fig. 3).

True disuse osteoporosis results following loss of mechanical stress provided by weightbearing or muscle contracture. This should be distinguished from post-traumatic osteoporosis which results following the combination of immobilization and hyperemia induced by trauma (13). Disuse osteoporosis is a low bone turnover process which results in irreversible loss of bone (Fig. 4). It appears similar to senile osteoporosis but is differentiated by its localized involvement. Bone reinforcement lines may be present in both forms of chronic adult osteopenia. These appear as transverse lines of radiodensity in the metaphyseal region of long bones. They may represent accentuation of previously hidden growth lines or can be developed de novo (25).

Post-traumatic osteoporosis and reflex sympathetic dystrophy are debatably varying degrees of one disease entity. The radiographic appearance of both processes is for the most part identical requiring clinical information to distinguish the two.

Patchy rarefraction is the most prominent feature in both post-traumatic osteoporosis and reflex sympathetic dystrophy. The severity of demineralization is more pronounced in the latter. Juxta-articular osteopenia is evident in both of these aggressive disorders (Fig. 5).

Summary

Radiological methods of evaluating generalized or local osteopenia have been presented. Semiquantitative and qualitative methods of determining relative bone density have been shown to be valuable, easily accessible ancillary methods of evaluating the osteopenic patient. Hormonal, nutritional, mechanical, and circulatory effects on the patterns of osteoporosis seen on radiographs have been presented.

References

1. Schneider R: Radiologic methods of evaluating generalized osteopenia. *Orthop Clin* 15:631-651, 1984.
2. Evans RA, McDonnell GD, Schieb M: Metacarpal cotical area as an index of bone mass. *Br J Radiol* 51:428-431, 1978.
3. Genant HK, Ettinger B, et al: Osteoporosis assessment by quantitative computed tomography. *Orthop Clin North Am* 16:557-568, 1985.
4. Shukla SS, Leichter I, Karellas A, Craven JD, Greenfield MA: Trabecular bone mineral density measurement in vivo: Use of the ratio of coherent to compton scattered photons in the calcaneus. *Radiology* 158:695-697, 1986.
5. Singh M, Nagrath AR, Maini PS: Changes in trabecular pattern of the upper end of the femur as an index of osteoporosis. *J Bone Joint Surg* 52A:457-467, 1970.
6. Singh M, Riggs BL, Beabout JW, Jonsey J: Femoral trabecular-pattern index for evaluation of spinal osteoporosis. *Ann Int Med* 77:63-67, 1972.
7. Jhamaria NL, LAL KB, Udawat M, Baneri P, Kabra SG: The trabecular pattern of the calcaneum as an index of osteoporosis. *J Bone Joint Surg* 65B:195-198, 1983.
8. Baratelli M, Cabitza P, Parrin L: The calcaneal index in the determination of osteoporosis. *Ital J Orthop Traumatol* 10:549-554, 1984.
9. Dequeker J, Gautama K, Roh YS: Femoral trabecular patterns in asymptomatic spinal osteoporosis and femoral neck fracture. *Clin Radiol* 25:243, 1974.
10. Cockshott WP, Occleshaw C, Webber C, Walter SD, O'Brien K: Can a calcaneal morphologic index determine the degree of osteoporosis? *Skel Radiol* 12:119-122, 1984.
11. Epstein DM, Dalinka MK, Kaplan FS, Aronchick JM, Marinelli DL, Kundel HL: Observer variation in the detection of osteopenia. *Skel Radiol* 15:347-349, 1986.
12. Lane J, Vigorita VJ: Osteoporosis. *Orthop Clin* 15:711-728, 1984.
13. Frost HM: *The Bone Dynamics in Osteoporosis and Osteomalacia*. Springfield IL, Charles C. Thomas, 1964.
14. Joyce JM, Keats TE: Disuse osteoporosis: Mimic of neoplastic disease. *Skeletal Radiol* 15:129-132, 1986.
15. Keats TE, Harrison RB: A pattern of post traumatic demineralization of bone simulating permeative neoplastic replacement: A potential source of misinterpretation. *Skeletal Radiol* 3:113-114, 1978.
16. Kozin F, Soin JS, et al: Bone scintigraphy in the reflex sympathetic dystrophy syndrome. *Radiology* 138:437-443, 1981.
17. Meema HE, Schatz DL: Simple radiologic demonstrtion of cortical bone loss in thyrotoxicosis. *Radiology* 97:9-15, 1970.
18. Meema HE: Radiographically detectable intracortical porosity. *Acta Radiol Diagnosis* 27:165-172, 1986.
19. Meema HE, Meema S: Improved roentgenologic diagnosis of osteomalacia by microradioscopy of hand bones. *AJR* 125:925-935, 1975.
20. Genant HK, Kozin F, et al: The reflex sympathetic dystrophy syndrome. *Radiology* 117:21-32, 1975.
21. Jaworski ZF, Meunier P, Frost HM: Observations on two types of resorption cavities in human lamellar cortical bone. *Clin Orthop* 83:279, 1972.
22. Allman RM, Brower AC: Circulatory patterns of deossification. *Radiol Clin North Am* 19:553-569, 1981.
23. Burkhart JM, Jowsey J: Pathological changes in bones and joints induced by injury. *Br Med J* 2:657-663, 1936.
24. Fogelman I, McKillop JH, Bessent RG, et al. The role of bone scanning in osteomatalia. *J Nucl Med* 19:245-248, 1978.
25. Kursunoglu S, Pale D, Resnick D, et al: Bone reinforcement lines in chronic adult osteopenia: A hypothesis. *Radiology* 158:409-415, 1986.