Differential Diagnosis in Conventional Radiology

By Francis A. Burgener and Martti Kormano

1506 Illustrations

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Preface

Conventional radiographs remain the backbone of our specialty despite the advent of new, fascinating imaging techniques such as ultrasonography, computed tomography and most recently nuclear magnetic resonance. In contrast to many of these newer methods, conventional radiology is practiced not only by radiologists but also by a large number of clinicians and surgeons. With each film one is confronted with radiologic findings that warrant interpretation in order to arrive at a main diagnostic impression and a reasonable differential diagnosis. To assist the film reader in attaining this goal our book is based upon radiographic findings, unlike most other textbooks in radiology that are disease-oriented. Since many diseases present radiographically in a variety of manifestations, some overlap in the text is unavoidable. To minimize repetition, the differential diagnosis of a radiographic finding is presented in tabular form whenever feasible. The tables list not only the various diseases that may present radiologically in a specific way, but also describe in a succinct form the characteristically associated radiographic findings and pertinent clinical data. Radiographic illustrations and drawings are included to visually demonstrate the radiographic features under discussion.

This book is meant for physicians with some experience in radiology who wish to strengthen their diagnostic acumen. Being a comprehensive outline of radiographic findings, we expect it to be particularly useful to radiology residents preparing for their speciality examinations. Any physician involved in interpreting radiologic film images should find this book helpful, in direct proportion to his curiosity.

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Acknowledgments

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The illustrations are in a class by themselves. Daniel B. Wopperer, M.D., an accomplished artist and radiologist at the University of Rochester Medical Center, has succeeded far beyond our expectations in capturing the sense of radiographic images.

We wish to acknowledge our great indebtedness and offer our thanks to the many radiologists whose cooperation helped make available this illustrative collection of films. Special thanks are given to Drs. Oscar H. Gutierrez, Richard W. Katzberg, Robert F. Spataro, and John C. Wandtke of the University of Rochester Medical Center, to Drs. Peter B. Dean, Tapio Helelä, Ilpo Lauteala, Kauko Satokari and Pekka Virtama of the University Central Hospital of Turku, and to Dr. László Tabár of the Central Hospital of Falun, Sweden. Further assistance was given by Drs. Georgine B. DeMarino, Peter Ferin, John D. Gaare, Diana M. Hutchings, James K. Katz, Thomas L. Miller and Daniel B. Wopperer, all residents at the University of Rochester Medical Center, and to Drs. Eero Mäkinen, Pekka Mäkelä, and Hellevi Rikalainen, all radiologists at the University Central Hospital of Turku.

Finally, we wish to express thanks to Belinda E. DeLibero, Nina Eerola and Pirjo Helanko for the numerous hours they spent in typing and retyping the manuscript and constructing the many tables.

Francis A. Burgener, M.D.
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Contents

Section 1 **Bone**

Chapter 1 **Osteopenia**

Osteopenia is defined as a decrease in bone density caused by reduced bone formation and/or increased bone resorption. Reduction in bone formation may result from either inadequate matrix formation (e.g., disuse osteoporosis) or inadequate matrix calcification (e.g., osteomalacia). Primary hyperparathyroidism is an example of too much resorption of both bone matrix and mineral. A combination of the aforementioned causes results in the undermineralization present in the majority of osteopenic disorders.

Approximately 30% of the bone mineral must be lost before a difference in the bone density can be detected by conventional radiography. It should also be borne in mind that the normal bone density changes with age, increasing from infancy to age 35 to 40 and then progressively decreasing at the rate of 8% per decade in women and 3% in men.

The radiographic findings of osteopenia are loss of bone density and cortical thinning. Osteopenia may either be generalized or localized and its differential diagnosis is discussed separately in Tables **1** and **2**.

In *osteoporosis* a combination of loss of bone density and cortical thinning may result in an apparent increase in density of the cortex and vertebral endplates, that appear as thin sharp lines. With further progression of the disease the vertebral bodies become biconcave and anteriorly wedged (Fig. **1A**). Bone resorption occurs preferentially in the transverse trabeculae, while the trabeculae along stress lines are accentuated. With the exception of osteogenesis imperfecta, bones do not bend in osteoporosis. A predisposition of fractures however, exists especially in the vertebral bodies, ribs, hips and wrists. Fracture healing is delayed and the callus formation poor. Abundant callus formation in osteopenic bones occurs however with Cushing's syndrome and osteogenesis imperfecta.

In *osteomalacia* a nonspecific loss of bone density is often the only radiographic sign. Blurring of both cortical margins and trabeculae results in a "ground glass" appearance of the involved bone and is more characteristic. This is often most obvious in the vertebral bodies (Fig. **1B**). In the skull a mottled appearance similar to hyperparathyroidism is characteristic. Bones have a tendency to bend and bowing deformities are commonly found in the thorax, vertebral column, pelvis and extremities. Pseudofractures (Looser's zones or Milkman's syndrome) occur frequently and represent infractions with incomplete healing. They are found in the scapula, ribs, clavicle, ischial and pubic rami, femur (especially neck), and other long bones.

Bony lesions are found in less than half of the patients with *hyperparathyroidism* (Fig. **1C**). Subperiosteal resorption along the radial margin of the phalanges is virtually pathognomonic. These erosions occur most often in the proximal and middle phalanges of the index and middle finger (Fig. **2**). Absorption of the terminal tufts and cortical striations ("tunneling of the cortex") are commonly associated. Endosteal resorption occurs in long bones. Resorption may also be evident in the acromial ends of the clavicles, the sacroiliac joints, the symphysis, in the calcaneus at the insertion of the plantar fascia and in the ribs (usually in the upper border of the upper ribs). The bone is softened resulting in secondary deformities such as basilar impression and

Figure **1** (**A**) **Osteoporosis.** Deossified, biconcave, or partially collapsed vertebral bodies with thin but dense-appearing endplates and prominent vertical trabeculae. (**B**) **Osteomalacia.** Uniform deossification with loss of trabecular detail ("ground-glass appearance") and compression fractures. (**C**) **Hyperparathyroidism.** "Rugger jersey spine" more commonly found in secondary hyperparathyroidism (renal osteodystrophy) whereas primary hyperparathyroidism often looks like Fig. 1 B.

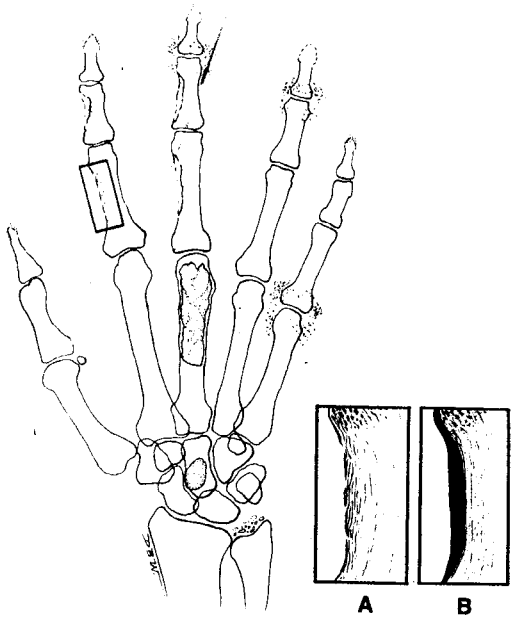

Figure **2** **Hyperparathyroidism** of the hand. Subperiosteal resorption and cortical striations usually best seen on the radial margins of proximal and middle phalanges of second and third finger. A magnified view of these findings is demonstrated in insert A, whereas insert B shows a normal cortex for comparison. Additional findings include resorption of the tufts, periarticular soft tissue calcifications, brown tumors (third metacarpal and capitatum), and joint cartilage calcification (often in articular disk between ulna and carpal bones).

kyphoscoliosis. Cyst-like lesions and so-called brown tumors occur in tubular and flat bones. While brown tumors heal after removal of the parathyroid adenoma and may eventually even become sclerotic (see Fig. **6** on page 15), cysts remain unchanged after treatment. Granular deossification of the skull results in a "salt and pepper" appearance. Resorption of the lamina dura around the teeth is commonly present. Soft tissue calcifications (especially arterial and para-articular), joint cartilage calcifications (especially menisci and the articular disc in the wrist), and nephroureterolithiasis, are common features of hyperparathyroidism.

An increased bone density is often associated with secondary hyperparathyroidism. In these cases thickening of the superior and inferior endplates of the vertebral bodies can result in a "rugger jersey spine" (Fig. **1C**).

Skeletal changes of different forms of hyperparathyroidism are identical, although brown tumors are more common in primary hyperparathyroidism, whereas osteosclerosis and extensive soft tissue calcifications favor secondary hyperparathyroidism.

Table 1 Differential Diagnosis of Generalized Osteopenia

Etiology	Comments
Osteoporosis	
Senile or postmenopausal	Most common form of osteoporosis. Females more often and more severely affected than males. Serum calcium, phosphate, and alkaline phosphatase within normal range.
Disuse atrophy	Prolonged immobilization of any cause (e.g., neuromuscular disorders, cast).
Protein deficiency (e.g., malnutrition, nephrosis) (Fig. 3)	Pure dietary protein deficiency is rare. More often it is associated with malabsorption (see under osteomalacia). Abnormal protein metabolism is the underlying cause of osteoporosis in *scurvy* (vitamin C deficiency) and different *endocrinologic disorders*.
Juvenile (idiopathic)	Between ages 8 to 14 years, characterized by abrupt onset of bone pain.
Osteogenesis imperfecta (Fig. 4)	Osteogenesis imperfecta congenita (fractures present at birth) and tarda (fractures absent at birth). Deformities resulting from recurrent fractures in later life and bone bending characteristic. Both disorders inherited.
Homocystinuria	Inherited disorder that presents radiographically as combination of osteoporosis, Marfan-like changes (e.g., arachnodactyly), and metaphyseal and epiphyseal widening.
Bone marrow hyperplasia or infiltrations (e.g., anemias, multiple myeloma, carcinomatosis) (Figs. 5 and 6).	Cause cortical thinning and trabecular resorption by pressure atrophy. Osteoporosis may be the only radiographic manifestation in multiple myeloma and carcinomatosis, but patchy osteolytic areas are often present. Generalized cystic appearance of particularly the flat bones is characteristic for *thalassemia*.
Collagen disease (especially rheumatoid arthritis)	Other more characteristic radiographic findings are often associated with the disease suggesting the correct diagnosis (see Chapter 6, page 65).

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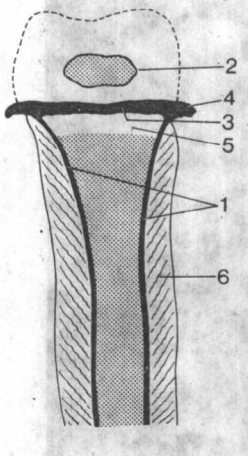

Figure **3** . **Scurvy.** Characteristic findings include: (1) Osteopenia with markedly thinned cortex, (2) thin, dense, ring-like calcification around epiphysis (Wimberger's line), (3) dense, linear calcifications in distal metaphysis ("white line of Fränkel"), (4) small bone spur immediately adjoining the "white line of Fränkel" (Pelken's spur), (5) radiolucent band proximal to the "white line of Fränkel" (Trümmerfeld zone), and (6) subperiosteal hemorrhage (calcifies only after institution of therapy). Epiphyseal separation or fragmentation in the region of metaphysis is commonly associated.

Table 1 Differential Diagnosis of Generalized Osteopenia (Cont.)

Etiology	Comments
Endocrine (e.g., hypogonadism, Cushing's syndrome, Addison's disease, diabetes mellitus, hyperthyroidism). See also under hyperparathyroidism in this table.	Hypogonadism: osteoporosis associated with delayed epiphyseal fusion (e.g., *Turner's syndrome, eunuchoidism*). Cushing's syndrome: chronic excess of glucocorticoids. Addison's disease: insufficiency of the adrenal cortex. Hyperthyroidism: often associated with cortical striations best seen in metacarpal bones.
Drug induced (e.g., steroids, heparin)	Steroids: large dosages over several months. Heparin: 15,000 to 30,000 units for six months or longer.
***Osteomalacia* (Fig. 7)**	
Deficient absorption of calcium and/or phosphorus: **1. vitamin D deficiency (dietary or lack of sunshine exposure),** **2. malabsorption (gastrointestinal disease, hepatobiliary disease, pancreatic disease),** **3. dietary calcium deficiency (extremely rare).**	Laboratory findings in osteomalacia: serum calcium slightly low to normal; serum phosphorus low; alkaline phosphatase elevated. Features in *rickets* (osteomalacia in children) include: indistinct, frayed, and concave metaphyses ("cupping") with perpendicular trabeculae extending into the epiphyseal area. Epiphyses appear blurred (DD: Scurvy: sharply outlined epiphyses). (Fig. **8**)
Defects in renal tubular or intestinal calcium phosphate transport system: **1. vitamin D-resistant rickets and pseudo-vitamin D deficiency rickets,** **2. renal tubular acidosis,** **3. de Toni-Debré-Fanconi syndrome.**	*Vitamin D-resistant rickets* (x-linked dominant) and pseudo-vitamin D deficiency rickets (autosomal recessive) present clinically similar (short stature, multiple fracturies, varus and valgus knee deformities and muscular weakness), but only the latter disorder is commonly associated with convulsion. *Renal tubular acidosis:* metabolic acidosis attributed to renal loss of alkali. Pathogenesis of osteomalacia in this condition unclear.

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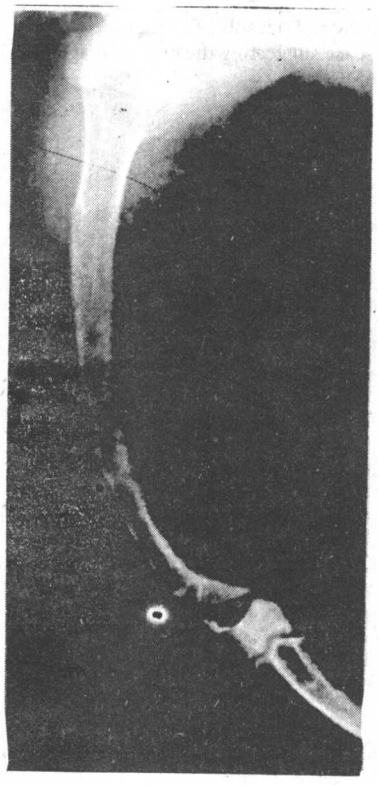


Figure **4** **Osteogenesis imperfecta.** Osteoporosis and bowing deformity characteristic.

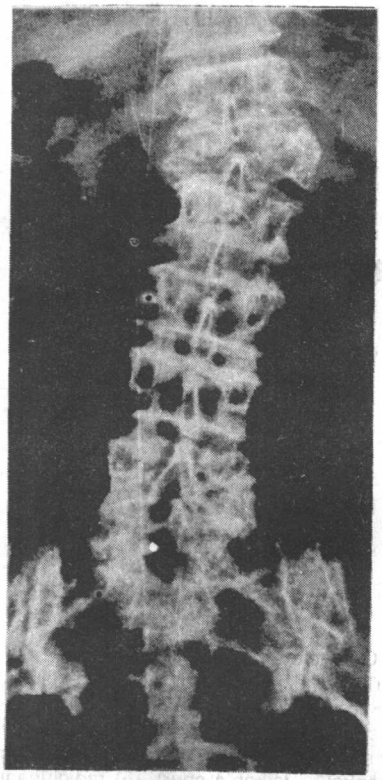
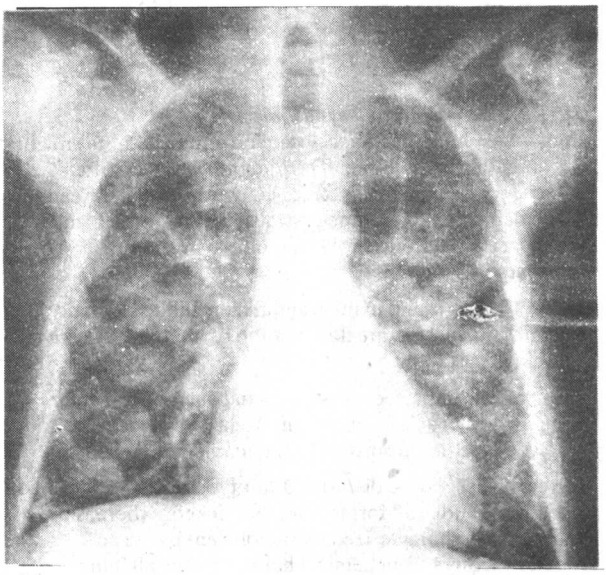


Figure **5** **Multiple myeloma** presenting as generalized osteopenia in the spine. Extensive destruction of L1 and destroyed left pedicle of L5 suggest in this case, however, the malignant process.



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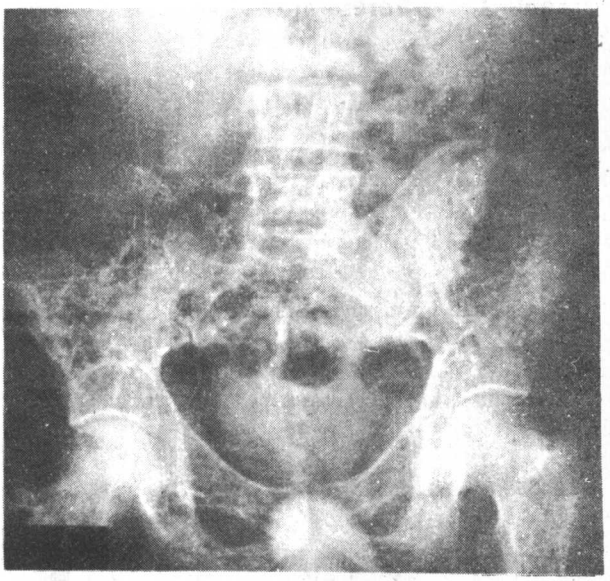

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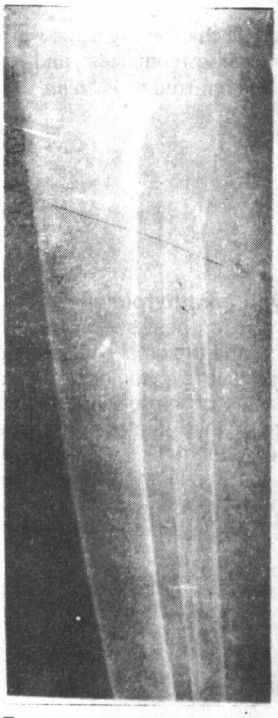
Figure **6** **Thalassemia major.** Chest (**A**) and pelvis (**B**). Generalized, cystic appearing osteopenia caused by red bone marrow hyperplasia with main involvement of the central or flat bones characteristic. Note the bulbous widening of the anterior ends of the ribs.



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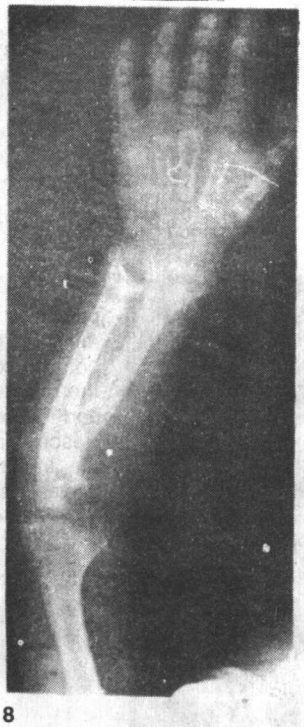

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Figure **7** **Osteomalacia.** Marked demineralization with blurring of the inner cortical margins and loss of trabeculations characteristic. Several pseudofractures are seen, presenting as sclerotic transverse lines in the tibia.

Figure **8** **Rickets.** Characteristic changes include: (1) osteopenia, (2) poorly calcified and defined epiphyses, (3) widening of the epiphyseal cartilage plate, (4) widening, cupping, and fraying of the metaphyses, (5) periosteal reactions, and (6) bowing deformities. Greenstick fractures are also commonly associated but not present in this case.

Table 1 Differential Diagnosis of Generalized Osteopenia (Cont.)

Etiology	Comments
Chronic anticonvulsant drug therapy.	Anticonvulsants and many tranquilizers induce hepatic enzymes that accelerate degradation of biologically active vitamin D metabolites.
Fibrogenesis imperfecta ossium and axial osteomalacia.	Fibrogenesis imperfecta ossium (axial and appendicular bone involved) and axial osteomalacia (only axial skeleton involved) are rare disorders found in middle-aged males.
Hypophosphatasia	Autosomal recessive disorder. Four types are distinguished: The three childhood forms resemble rickets, whereas the adult form is characterized by radiolucent bones and pseudofractures. Biochemical hallmark: low alkaline phosphatase.
***Hyperparathyroidism* (Fig. 9)**	
***Primary* hyperparathyroidism: parathyroid adenoma, primary chief cell or clear cell hyperplasia of all parathyroid glands, parathyroid carcinoma.**	Laboratory findings of primary hyperparathyroidism: Serum phosphate low, serum calcium high, alkaline phosphatase high in the presence of bone disease.
***Secondary* hyperparathyroidism: compensatory mechanism in any state of true hypocalcemia (e.g., hypovitaminosis D, malabsorption of calcium, chronic renal disease).**	In chronic renal disease, the skeletal changes are usually a combination of hyperparathyroidism, osteomalacia, and osteosclerosis. This complex is best referred to as *"renal osteodystrophy."*
***Tertiary* hyperparathyroidism: development of an autonomous parathyroid adenoma in chronically overstimulated hyperplastic parathyroid glands (e.g., post renal transplantation).**	

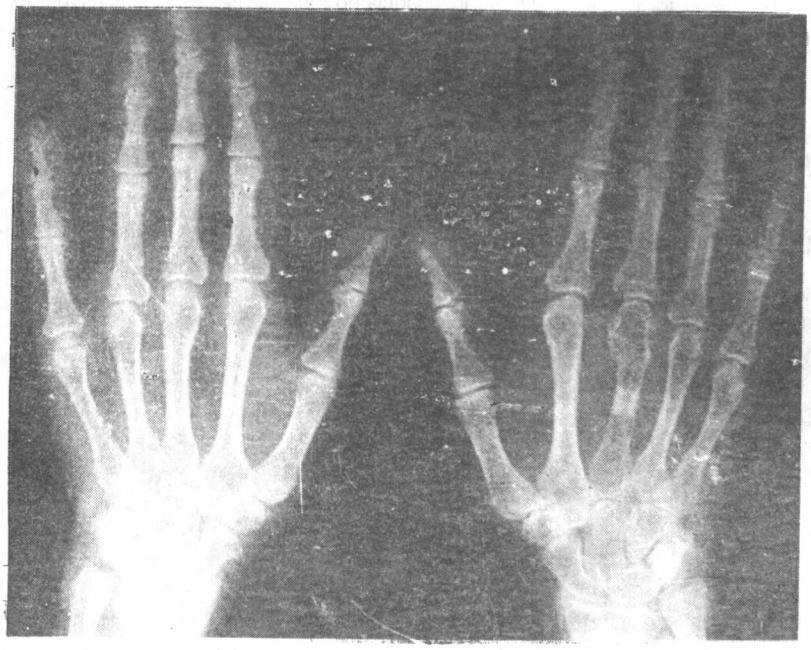

Figure **9 Hyperparathyroidism.** Subperiosteal resorption best seen along the radial margin of the proximal phalanges of both index fingers. Brown tumors involving the distal phalanx of the left index finger and entire right third metacarpal bone. Resorption of the tufts, especially in the thumbs. Cortex in metacarpals and phalanges shows fine striations. See also Fig. 2, p. 4.

Table 2 Differential Diagnosis of Localized Osteopenia

Etiology	Comments
Disuse atrophy (local immobilization): **1. fracture (more pronounced distal to the fracture site)** **2. cast** **3. neural paralysis** **4. muscular paralysis**	Besides identical radiographic features as in generalized osteopenia, the localized form can also have a patchy appearance due to spotty cortical thinning as in Sudeck's atrophy.
Sudeck's atrophy (Fig. 10)	Rapid development of spotty osteoporosis associated with painful soft tissue swelling following trivial trauma.
Burns and frostbites	Radiographic findings consist of osteoporosis, bone resorption, and bone necrosis.
Inflammatory: **1. rheumatoid arthritis** **2. osteomyelitis** **3. tuberculosis**	Localized osteoporosis is usually the first but nonspecific radiographic manifestation of any inflammatory disease.
Bone infarct and hemorrhage	In their early stages, both bone infarcts and hemorrhages produce localized demineralization. With healing, lesions become osteosclerotic.
Tumor (benign; malignant, primary or metastatic) (Fig. 11)	Osteolytic metastases and multiple myeloma must primarily be considered.
Paget's disease (lytic phase) (Fig. 12)	Skull: osteoporosis circumscripta. Long bones: usually a well-defined and V-shaped area of deossification.
Regional transitory osteoporosis	A painful self-limited osteoporosis most often found in the hip ("transitory demineralization of the femoral head") associated with disability of 2 to 4 months.
Shoulder-hand syndrome	Pain and stiffness in the shoulder combined with pain, swelling and vasomotor phenomena in the hand following an acute illness (e.g. myocardial infarction in which condition it is usually located on the left side). Radiographically, it resembles Sudeck's atrophy.

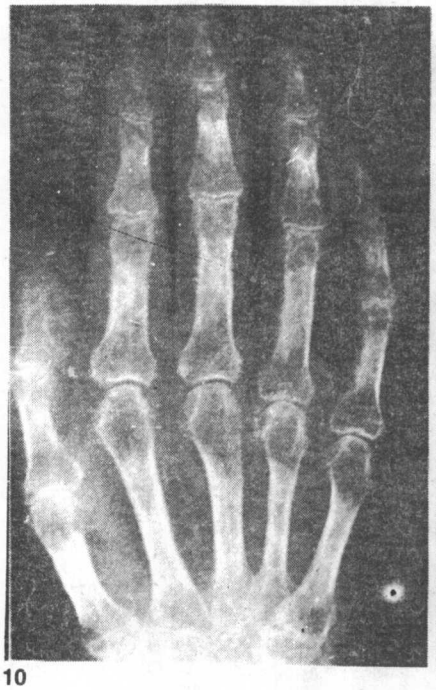

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Figure **10** **Sudeck's atrophy.** Patchy demineralization most severe near the joints characteristic.

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Figure **11** **Multiple myeloma.** Demineralization most pronounced near the joints similar to Sudeck's atrophy in Fig. 10. Note the small cortical destruction in the ulnar aspect of the distal radius.

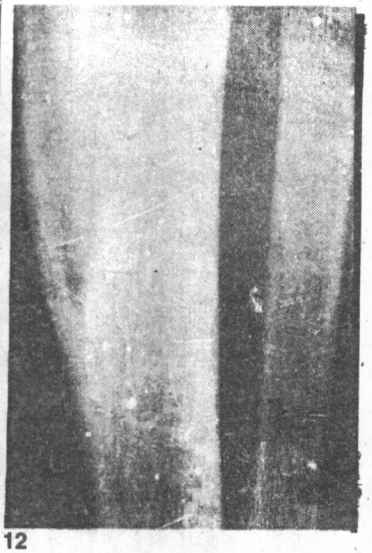

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Figure **12** **Paget's disease (lytic phase).** Well-defined V-shaped area of deossification associated with slight expansion of the tibia shaft characteristic for lytic phase in long bones.

Chapter 2 **Osteosclerosis**

Osteosclerosis is defined as an increase in bone density caused by an increased activity of osteoblasts or by osteogenic or chondrogenic tumor cells forming bone-like tissue. Calcification of tissue other than osteoid within bone or in its periphery may also increase the bone density radiographically.

The increase in bone density may be scattered or diffuse. This distinction appears useful in the differential diagnosis of osteoblastic reactions, since certain diseases may exclusively present as scattered (solitary or multiple) sclerosis. Accordingly, the differential diagnosis of these entities will be discussed separately in Tables **1** and **2**. Table **3** lists sites and commonly used eponyms of idiopathic aseptic necrosis.