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骨与关节影像学

第3版

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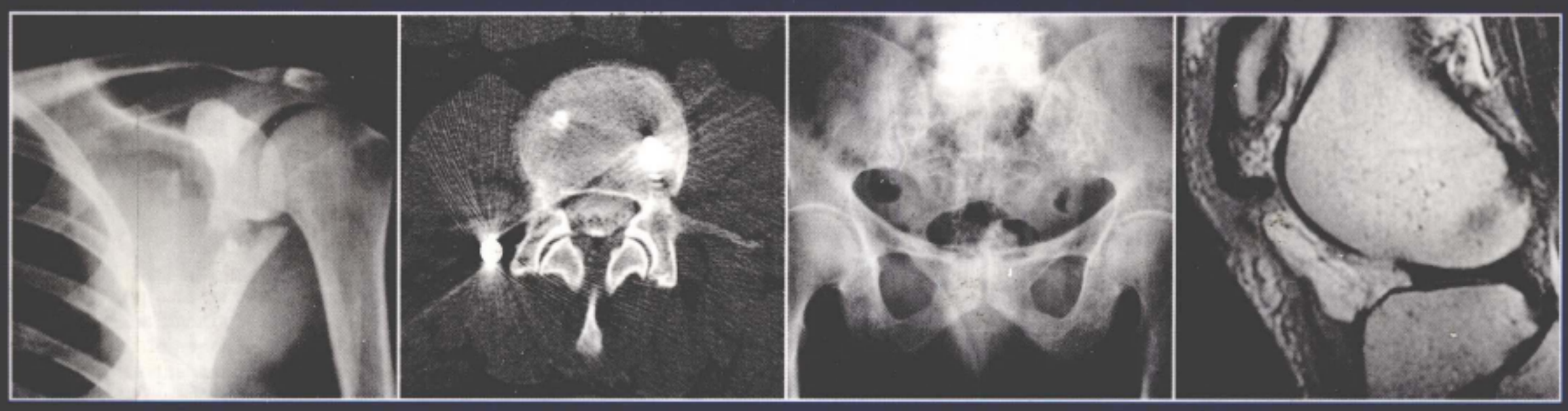

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Bone and Joint IMAGING

骨与关节影像学

第 3 版

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PREFACE

Nine years after the publication of the second edition of **Bone and Joint Imaging** and a few years after the publication of the fourth edition of the larger **Diagnosis of Bone and Joint Disorders**, the third edition of **Bone and Joint Imaging** is now ready for dissemination. In common with the first and second editions of this text, the purpose of this book is to present in a logical manner and easy-to-read format the information that we, the authors, believe is essential for those learning musculoskeletal imaging for the first time or for those reviewing the subject one more time. The subject of musculoskeletal imaging is ever changing and constantly growing in scope. Much of this growth relates not to the discovery of new processes or disorders but rather to the development and refinement of advanced imaging methods and techniques. Diagnostic methods now applied routinely to the analysis of musculoskeletal disorders include far more than conventional radiography: CT scanning, MR imaging, ultrasonography, radionuclide studies, and arthrography are among the additional methods that must be mastered by those interpreting images related to bone, joint, and soft tissue disorders. To summarize adequately the many imaging techniques and findings in a text any shorter than this, in our view, would not be appropriate or even possible.

The organization of the text follows that of the previous edition. Basic anatomy and physiology, diagnostic techniques, and postoperative imaging serve as introductory material; this material is then followed by sections dealing with imaging of most of the important diseases that affect the musculoskeletal system. Key images have been selected to illustrate the most important of the imaging findings, and a short but appropriate bibliography is included in each chapter. As before, we have included shortened versions of many chapters written by experts in the field that were part of the larger multivolume textbook. When compared with the second edition, however, there are significant changes in this third edition. Many subjects appear for the first time, countless new and improved illustrations are included, and references are updated. And to do this properly and on time, two editors rather than one have accomplished this task.

Both of us are confident that we have succeeded in condensing the essential material related to musculoskeletal imaging in a manageable textbook. But it is the readers who are the ultimate judge. We are hopeful that whether it is used for consultation on an intermittent basis or read in its entirety, the readers will enjoy the experience and be wiser for it.

DONALD RESNICK

MARK J. KRANSDORF

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To our residents and fellows
— for their motivation, enthusiasm, and, most important, inspiration

CONTENTS

SECTION XIV
Infectious Diseases 713

SECTION XV
Traumatic Diseases 789

SECTION XVI
Internal Derangement of Joints 905

SECTION XVII
Thermal, Iatrogenic, Nutritional, and Neurogenic Diseases 995

SECTION XVIII
Osteonecrosis and Osteochondrosis 1067

SECTION XIX
Tumors and Tumor-like Diseases 1109

SECTION XX
Congenital Diseases 1265

Basic Science

CHAPTER 1

Histogenesis, Anatomy, and Physiology of Bone

Donald Resnick, Stavros C. Manolagas, and Michael D. Fallon

SUMMARY OF KEY FEATURES

Bone is a unique tissue that is constantly undergoing change. It develops through the processes of endochondral and intramembranous ossification and is subsequently modified and refined by the processes of modeling and remodeling to create a structurally and metabolically competent, highly organized architectural marvel. Its cells, including osteoblasts, osteocytes, and osteoclasts, reside in organic matrix, primarily collagen, and inorganic material is deposited in a form that resembles hydroxyapatite. The process of mineralization is complex and incompletely understood.

Bone is essential in maintaining calcium homeostasis, or stabilization of the plasma level of calcium. Its cells are highly responsive to stimuli provided by a number of humoral agents, the most important of which are parathyroid hormone, thyrocalcitonin, and 1,25-dihydroxyvitamin D. Synthesis and resorption of bone, which normally continue in a delicate balance throughout life, are mediated by the action of such humoral agents through processes that include stimulation of osteoblasts to form bone and stimulation of osteoclasts to remove bone.

INTRODUCTION

Bone is a remarkable tissue. Although its appearance on radiographs might be misinterpreted as indicating inactivity, bone is constantly undergoing change. This occurs not only in the immature skeleton, in which growth and development are readily apparent, but also in the mature skeleton, through the constant and balanced processes of bone formation and resorption. It is when these processes are modified such that one dominates, that a pathologic state may be created. In some instances, the resulting imbalance between bone formation and resorption is easily detectable on the radiograph. In others, a more subtle imbalance exists that may be identified only at the histologic level.

The initial architecture of bone is characterized by an irregular network of collagen, termed woven-fibered bone, which is a temporary material that is either removed to form a marrow cavity or subsequently replaced by a sheetlike arrangement of osseous tissue, termed parallel-fibered, or lamellar, bone. As a connective tissue, bone is highly specialized and differs from other connective tissue by its rigidity and hardness, which relate primarily to the inorganic salts that are deposited in its matrix. These properties are fundamental to a tissue that must maintain the shape of the human body, protect its vital soft tissues, and allow locomotion by transmitting from one region of the body to another the forces generated by the contractions of various muscles. Bone also serves as a reservoir for ions, principally calcium, that are essential to normal fluid regulation; these ions are made available as a response to stimuli produced by a number of hormones, particularly parathyroid hormone, vitamin D, and calcitonin.

HISTOGENESIS

Developing Bone

Bone develops by the process of intramembranous bone formation (transformation of condensed mesenchymal tissue), endochondral bone formation (indirect conversion of an intermediate cartilage model), or both. At some locations, such as the bones of the cranial vault (frontal and parietal bones, as well as parts of the occipital and temporal bones), the mandible and maxilla, and the mid-portion of the clavicle, intramembranous (mesenchymal) ossification is detected; in other locations, such as the bones of the extremities, the vertebral column, the pelvis, and the base of the skull, both endochondral and intramembranous ossification can be identified. The actual processes of bone tissue formation are essentially the same in both intramembranous and endochondral ossification and include the following sequence: (1) osteoblasts differentiate from mesenchymal cells; (2) osteoblasts deposit matrix, which is subsequently mineralized; (3) bone is initially deposited as a network of immature (woven) trabeculae, the primary spongiosa; and (4) the primary spongiosa is replaced by secondary bone, removed to form bone marrow, or converted to primary cortical bone by the filling of spaces between trabeculae.

Intramembranous Ossification

Intramembranous ossification is initiated by the proliferation of mesenchymal cells about a network of capillaries. At this site, transformation of the mesenchymal cells is accompanied by the appearance of a meshwork of collagen fibers and amorphous ground substance. The primitive cells proliferate, enlarge, and become arranged in groups, transforming into osteoblasts, which are intimately involved in the formation of an eosinophilic matrix within the collagenous tissue. As the osteoid matrix undergoes calcification with the deposition of calcium phosphate, some of the osteoblasts on the surface of the osteoid and

woven-fibered bone become entrapped within the substance of the matrix in a space called a lacuna. These cells, now osteocytes, maintain the integrity of the surrounding matrix and are not directly involved in bone formation. Through the continued transformation of mesenchymal cells into osteoblasts, elaboration of an osteoid matrix, and entrapment of osteoblasts within the matrix, the primitive mesenchyme is converted into osseous tissue.

The ultimate characteristics of the tissue depend on its location within the bone: in cancellous areas of the bone, the meshwork of osseous tissue contains intervening vascular connective tissue, representing the embryonic precursor of the bone marrow; in compact areas of the bone, the osseous tissue becomes more condensed and forms cylindric masses containing a central vascular channel, the haversian system. On the external and internal surfaces of the compact bone, fibrovascular layers develop (periosteum and endosteum) and contain cells that remain osteogenic and give bone its ever-changing quality.

Endochondral Ossification

In endochondral (intracartilaginous) ossification, cartilaginous tissue derived from mesenchyme serves as a template and is replaced with bone (Fig. 1–1). The initial sites of bone formation are called centers of ossification. In tubular bones, the primary center of ossification is located in the central portion of the cartilaginous model, whereas later-appearing centers of ossification (secondary centers) are located at the ends of the models within epiphyses and apophyses. Vascular mesenchymal tissue or perichondrium, whose deeper layers contain cells with osteogenic potential, surrounds the cartilaginous model.

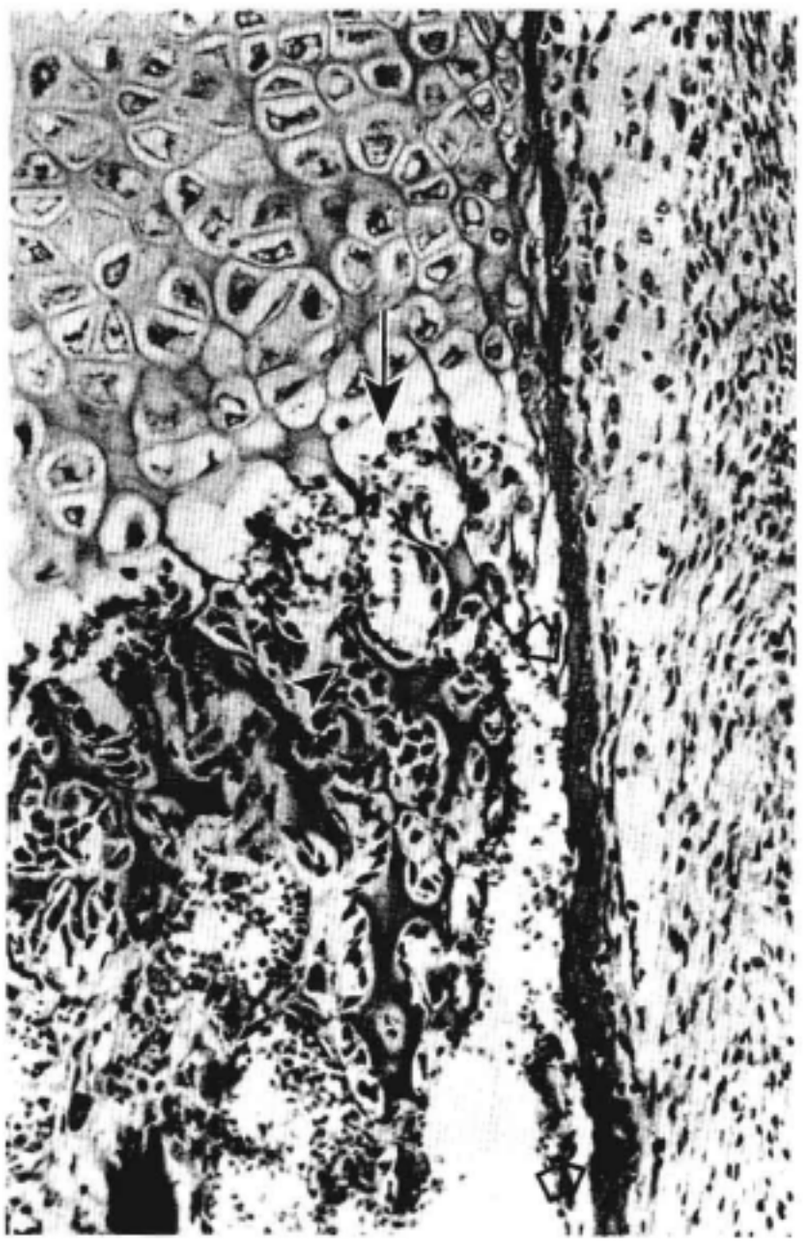

Figure 1–1. Endochondral and intramembranous ossification in a tubular bone: radius of a 4½-month-old fetus. The large and confluent cartilage cell lacunae are being penetrated by vascular channels *(solid arrow)*, thereby exposing intervening cores of calcified cartilage matrix. The osteoblasts are depositing osseous tissue on these cartilage matrix cores *(arrowhead)*. Observe the subperiosteal bone formation *(open arrows)*.

The initial changes in the primary center of ossification are hypertrophy of cartilage cells, glycogen accumulation, and reduction of intervening matrix. Subsequently, these cells degenerate, die, and become calcified. Simultaneously, the deeper or subperichondrial cells undergo transformation to osteoblasts, and through a process identical to intramembranous ossification, these osteoblasts produce a subperiosteal collar or cuff of bone that encloses the central portions of the cartilaginous tissue. Periosteal tissue is converted into vascular channels, and the aggressive vascular tissue disrupts the lacunae of the cartilage cells and creates spaces that fill with embryonic bone marrow. Osteoblasts appear and transform the sites of degenerating and dying cartilage cells into foci of ossification by laying down osteoid tissue in the cartilage matrix. Osteoblasts become trapped within the developing bone as osteocytes.

From the center of the tubular bone, ossification proceeds toward the ends of the bone. Similarly, the periosteal collar, which is actively participating in intramembranous ossification, spreads toward the ends of the bone, slightly ahead of the band of endochondral ossification. Through a process of subperiosteal deposition of bone, a cortex becomes evident, grows thicker, and is converted into a system with longitudinally arranged compact bone surrounding vascular channels (haversian system). The front of endochondral ossification that is advancing toward the end of the bone becomes better delineated, and it is this plate that ultimately becomes located between the epiphysis and diaphysis of a tubular bone and forms the growth plate (cartilaginous plate or physis). The plate contains clearly demarcated zones: a resting zone of flattened and immature cells on the epiphyseal aspect of the plate, as well as zones of cell growth and hypertrophy and of transformation, with provisional calcification and ossification on the metaphyseal or diaphyseal aspect of the plate.

The size and shape of the most recently formed portion of the metaphysis of a tubular bone depend on the effects of an encircling fibrochondro-osseous structure, designated the periphysis, that consists of the zone of Ranvier, the ring of La Croix, and the bone bark that they produce (Fig. 1–2). In this setting of progressive ossification of the diaphysis with longitudinal spread toward the ends of the bone, characteristic changes appear within the epiphysis. Epiphyseal invasion by vascular channels is followed by the initiation of endochondral bone formation, which creates secondary centers of ossification. The process is again characterized by cartilage cell hypertrophy and death, followed by calcification.

The epiphyseal ossification center at first develops rapidly, although later the process slows. The epiphyseal cartilage is thus converted to bone, although a layer on its articular aspect persists and is destined to become the