PALEOPATHOLOGICAL DIAGNOSIS AND INTERPRETATION

Bone Diseases in Ancient Human Populations

R. TED STEINBOCK

Harvard Medical School
Boston, Massachusetts

With a Foreword by

T. DALE STEWART, M.D.

Anthropologist Emeritus
Smithsonian Institution

This engrossing volume offers a systematic approach to diagnosing bone lesions in excavated skeletal material and interpreting the significance of such diseases in ancient human populations. An extensive review of the paleopathological literature is presented to familiarize the reader with current archaeological evidence for the antiquity of specific diseases. The medical literature is also reviewed to provide relevant clinical data vital for the diagnosis of diseases from dried bone specimens.

Physical anthropologists, pathologists, radiologists, orthopedists, and those interested in medical history will all find information applicable to their special fields in this unique study.

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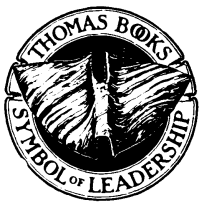

CHARLES C THOMAS · PUBLISHER
Springfield · Illinois · U.S.A.

Published and Distributed Throughout the World by
CHARLES C THOMAS • PUBLISHER
Bannerstone House
301-327 East Lawrence Avenue, Springfield, Illinois, U.S.A.

ISBN 0-398-03512-1
Library of Congress Catalog Card Number: 75-34701

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Library of Congress Cataloging in Publication Data

Steinbock, R. Ted.
Paleopathological diagnosis and interpretation.

Includes bibliographies and index.
1. Paleopathology. 2. Bones—Diseases—Diagnosis.
I. Title. [DNLM: 1. Bone diseases—History.
2. Paleopathology. QZ11.5 S819p]
R134.8.S73 616.07 75-34701
ISBN 0-398-03512-1

Printed in the United States of America
W-2

TO MY PARENTS

FOREWORD

I FIRST MET Ted Steinbock in 1972 while he was attending the Paleopathology Seminar at the National Museum of Natural History. Some of the circumstances of our association throw light on the way in which his book developed and on what makes his book such an important contribution to paleopathology.

Ted and I were drawn together by a shared interest in a small but excellently preserved skeletal collection from Indian Knoll, Kentucky. Clarence B. Moore, the famous gentleman-archaeologist from Philadelphia, had donated this collection to the Museum in 1915 at the conclusion of one of his annual steamboat expeditions exploring Indian sites along the Green River.

Ted and I knew, of course, that during the WPA days (1939-40) the Indian Knoll site had been further explored by field parties under the direction of Major Webb of the University of Kentucky, and the number of recovered skeletons extended to well over 1000. Moreover, the site had been determined to be some 5000 years old. Thus, the combined Indian Knoll collections constitute the largest assemblage of carefully excavated and well-preserved skeletons from any Indian site of such great age. Obviously, the correct identification of the diseases afflicting this, and any other, early population is a matter of great historical importance.

Ted was interested in the Indian Knoll population through his archaeological work in Kentucky and anthropological training at Harvard. My interest, on the other hand, was mainly in seeing that the research potential of the skeletons from this site was fully realized. In fact, only three years before Ted's visit to Washington I had called attention to the little known circumstance that someone at the Army Medical Museum (now the Armed Forces Medical Museum) had arranged with Moore to save pathological bones for that institution. This could mean, I

felt, that from the pathological standpoint the Indian Knoll collection in the National Museum is not a random sample.

Ted's concern about this matter was one of the first subjects we discussed. He wanted to know whether I had looked at the bones from Indian Knoll in the Armed Forces Medical Museum and, if not, whether I had any objection to his doing so. My answers to both questions being in the negative, in due course I received from him a detailed account of his findings. Later still he kindly reported on the condition of the larger part of the skeletal collection housed in Lexington, Kentucky, and on the examples of particular pathological conditions he had examined there. I could not help but be impressed by the systematic and thorough way in which he was pursuing the subject.

All this led in 1973 to an honors thesis which received a unanimous *summa* when submitted to the Department of Biology at Harvard. Ted then went on to Harvard Medical School and continued his interest in paleopathology. While still in medical school he has converted the thesis into book form. Although the book bears the same title and nearly the same table of contents as the thesis, the text and illustrations have been extensively revised and expanded. The earlier emphasis on the bone lesions present at Indian Knoll has given way to an extensive review of the paleopathological literature. Also, since the book is designed to provide a systematic approach to diagnosing bone lesions in excavated skeletal series and interpreting their significance in prehistoric human populations, the new emphasis is on documented clinical specimens.

A book of this sort has been needed, because few clinicians can claim competence in diagnosing diseases from gross bones alone, whereas the anthropologists who encounter such bones in great quantities rarely have sufficient knowledge of pathology to properly interpret osseous lesions. I congratulate Ted on the successful outcome of his endeavors and predict that as a result of his book the relatively new science of paleopathology will receive contributions of more lasting value than heretofore.

T. Dale Stewart, M.D.

INTRODUCTION

PALEPATHOLOGY AS DEFINED here is the study of diseases in ancient human populations as revealed by their skeletal remains. In certain instances, mummified tissues, ancient art forms, and literature can also be utilized. Paleopathology is important in providing information on the health status of ancient human populations. Furthermore, it provides new knowledge of the antiquity of specific diseases affecting bone and the end results of such diseases in human populations without medical technology.

The term paleopathology was popularized if not invented by Sir Marc Armand Ruffer at the turn of this century while working with the extensive collections of Egyptian mummies and skeletons. With few exceptions, the literature since then has consisted of short reports describing isolated specimens of bone pathology. The specimens are often regarded as interesting curios without attempting to interpret the significance of such osseous lesions in the populations represented by the individuals. More recently an epidemiological or population approach has been utilized to study the skeletal pathology of ancient human populations, and this provides much more meaningful information.

The purpose of this book is to provide a basic framework for those interested in diagnosing and interpreting bone lesions found in excavated skeletal series. The literature concerning the archaeological evidence of disease is reviewed here as a background for the presentation of the relevant clinical data on the major diseases of bone. This is primarily written for physical anthropologists who are the major contributors to the field of paleopathology. Pathologists and radiologists are more concerned with the microscopic and radiographic picture of bone disease than with the gross morphology of the dried bone specimen.

However, workers in both areas have made valuable contributions to paleopathology, and this book owes much to their clinical studies of bone disease.

This book is of necessity limited in scope and breadth. The gross morphology and radiographic appearance of the macerated bone specimen is emphasized here. Other techniques such as microscopic examination of sectioned bone are of questionable value in archaeological specimens, and further advances in this area are urgently needed. Mummified tissues offer an exciting opportunity for studying diseases which do not affect bone or bone diseases with soft tissue manifestations. Rehydration and staining techniques for dessicated tissues and fecal specimens are not described here as they could easily be the subject of an entire book. Only the major infectious, nutritional, metabolic, degenerative, and neoplastic diseases of bone are discussed here. These diseases account for the great majority of pathological lesions in excavated specimens.

Finally, although diagnosis is emphasized in this book, this does not imply that one can always specify the disease illustrated by the dried bone specimen. Such an attitude can only produce more confusion than enlightenment in the field of paleopathology. A more rational approach is to state the most likely diagnosis followed by a list of possible alternatives in order of decreasing likelihood. Again, I cannot overemphasize the importance of carefully considering the archaeological and epidemiological context of the pathological specimen in establishing the antiquity of disease and its prevalence in ancient human populations.

R. Ted Steinbock
Harvard Medical School

ACKNOWLEDGMENTS

MANY PEOPLE HAVE helped me either directly or indirectly in the preparation of this book during the past four years. Professor Jonathan Friedlaender at the Harvard Peabody Museum initiated my interest in paleopathology and physical anthropology. Professor Farish Jenkins at the Harvard Museum of Comparative Zoology deserves special thanks and appreciation for his readiness to help in all aspects of my education and for his constant encouragement during the hectic years of medical school. Dr. Alan Schiller, orthopedic pathologist at the Massachusetts General Hospital, kindly read the manuscript and offered many helpful suggestions.

My research in gross bone pathology includes skeletal material from several prominent institutions, and I owe much to the equally prominent people associated with these institutions. At the Smithsonian Institution I wish to thank Dr. Donald Ortner, Associate Curator of the Division of Physical Anthropology, for allowing me to attend the Smithsonian's seminar in paleopathology and inviting me to return and undertake my own research project. Special thanks and appreciation are extended to Dr. T. Dale Stewart, anthropologist emeritus at the Smithsonian. His pioneering studies in paleopathology and skeletal age changes have been sources of great inspiration and information for me. At the Armed Forces Institute of Pathology, I wish to thank Dr. Lent C. Johnson, chief of orthopedic pathology, for allowing me to examine the pathological specimens in the affiliated Army Medical Museum. Many interesting cases of bone pathology were examined at the Warren Anatomical Museum of Harvard Medical School. Mr. David Gunner, Assistant Curator of the collection, was extremely helpful in making the entire collection available for my research.

Certain chapters in this book greatly benefited from my con-

tact with workers in these areas. Dr. Don Fawcett, Professor of Anatomy at Harvard Medical School, has generously allowed me to use several of his illustrations in the first chapter on bone biology. Dr. Stanley Garn at the University of Michigan has provided excellent illustrations of transverse lines in living subjects. The difficult chapter on syphilis and other treponemal infections owes much to the advice of Dr. Cecil J. Hackett and Dr. E. Herndon Hudson. Dr. Hackett has also generously allowed me to use several illustrations from his two valuable studies on the bone lesions of yaws. Dr. Dan Morse kindly reviewed my chapter on tuberculosis and sent several photographs of paleopathological specimens for other chapters. Dr. Vilhelm Møller-Christensen of Denmark and Dr. Michel Lechat of Belgium provided helpful information and illustrations on archaeological and clinical examples of leprosy. Dr. Charles Short, Consultant to the Arthritis Unit at Massachusetts General Hospital, kindly reviewed the chapter on arthritis and provided several clinical radiographs. Dr. Jack Dreyfuss, Associate Professor of Radiology at Harvard Medical School, generously assisted in the final stages of manuscript preparation.

Several illustrations were graciously supplied by Mr. Don R. Brothwell, Dr. John E. Moseley, and Dr. Calvin Wells. Dr. E. B. D. Neuhauser, chief of radiology emeritus at Children's Hospital of Boston, and Mr. James Conway both provided excellent clinical roentgenograms of certain bone disorders. Many other people have helped with this book in various ways. They include Mrs. Sterling Ament, Mr. Lazlo Meszoly, Mr. Richard Parker, and Miss Priscilla White. To all these people I am grateful.

R. Ted Steinbock

CONTENTS

PALEOPATHOLOGICAL DIAGNOSIS AND INTERPRETATION

Chapter I

BONE AS A LIVING TISSUE

THIS CHAPTER PROVIDES a brief introduction to bone as a living tissue and to the main features of gross bone anatomy. A thorough knowledge of normal bone growth and remodeling and normal bone structure is necessary before one can appreciate the response of bone to disease and the consequent changes in its gross appearance. For further information on normal bone physiology and anatomy, the reader should consult the bibliography at the end of this chapter. General references in paleopathology are also provided.

GROSS BONE ANATOMY

Bone is a connective tissue specially modified to provide a rigid framework. It is composed of living cells imbedded in an extracellular matrix of collagenous fibrils made rigid by calcium salts. Bone provides for the internal support of the body, protection of vital organs, and attachment for muscles necessary in locomotion. Bone encloses the hematopoietic tissue or bone marrow which produces the blood elements. In addition, the skeleton acts as the main source of mobilizable calcium for the maintenance of calcium levels in the blood.

The bones of the skeleton fall into four morphologic groups: long, short, flat, and irregular. The long bones are the main components of the limbs, supporting the weight of the body and providing attachment for the muscles of locomotion. The humerus, radius, ulna, femur, tibia, and fibula are in this category.

The short bones consist of all the bones in the hands and feet. Some prefer to describe the metatarsals and metacarpals as short tubular bones because of their similarities to the long tubular bones of the limbs. The flat bones include the bones of the cranial vault, ribs, sternum, scapulae, and pelvic bones. These bones provide protection and wide areas for muscle attachment. They are also the major sites of red bone marrow or active hematopoietic tissue in the adult. The irregular bones consist of the vertebrae and many bones of the skull. The vertebrae are also a major source of red bone marrow.

A typical long bone illustrates many of the main features of gross anatomy (see Fig. 1). At the end of the bone is an *epiphysis* which bears articular cartilage in the living state. The epiphysis contains the secondary center of ossification and

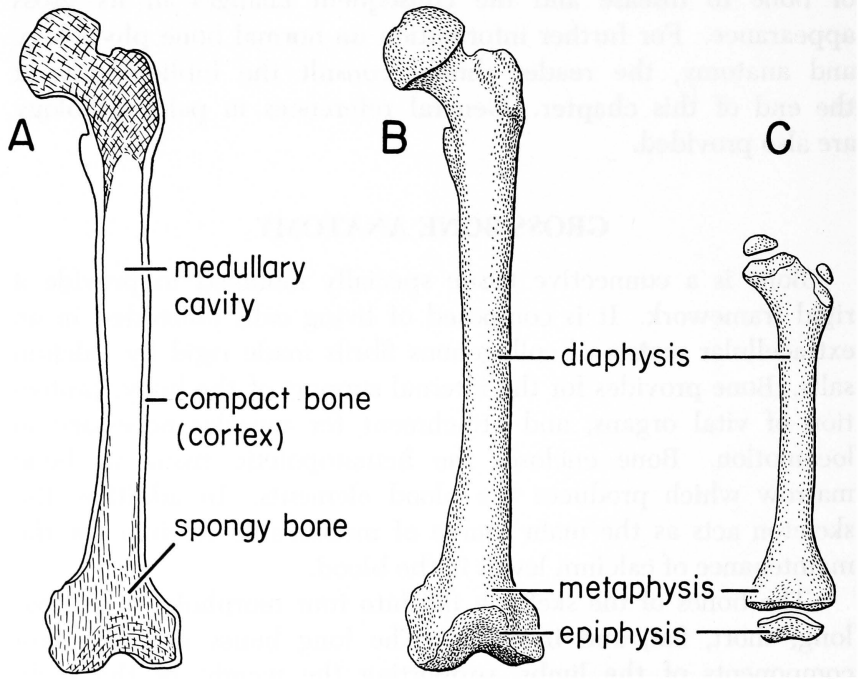

Figure 1. Gross anatomy of the femur. (A.) Longitudinal section revealing the medullary cavity and spongy or cancellous bone. (B.) External view of adult femur. (C.) External view of infant femur showing the epiphyses separate from the metaphyses and diaphysis.

epiphyseal disk and fuses to the *metaphysis* at some point during maturation. The metaphysis is immediately beneath the epiphyseal disk in the growing bone and extends into the medullary cavity as a loosely packed tissue which melds into the cortex of the *diaphysis* or shaft of the bone. The diaphysis is the central portion of a tubular bone and extends from the metaphysis of one end to the metaphysis of the opposite end.

Except where covered by cartilage for articulation with other bones, the entire bone is enveloped by a thin connective tissue membrane known as the *periosteum.* The periosteum does not persist in the dried bone specimen, but its osteogenic potential is very important in certain pathologic processes and in fracture repair. The periosteum adheres firmly to the underlying bone via bundles of collagen fibers and a rich network of capillaries and arterioles which permeate the bone wall or *cortex* through its system of vessel canals. During the growing years, the inner portion of the periosteal membrane contains numerous osteoblasts which lay down new bone. The number of periosteal osteoblasts is considerably decreased in adults but is still capable of producing new bone in response to inflammation or other stimulant.

Marrow Spaces and Endosteum

As mentioned earlier, the bones contain marrow for the synthesis of blood elements. The marrow is found mainly in the *medullary cavity* or canal of a long bone, but it is also located in all the cancellous spaces of the bone including their extensions along *Haversian canals* and other small vascular channels. The medullary cavity and cancellous spaces are theoretically lined by an osteogenic membrane called the *endosteum.* Although the endosteum is not a clearly defined and detachable membrane like the periosteum, the osteoblasts and potential osteoblasts of the marrow reticulum are important sources of new bone formation.

Bone Architecture

Bone can be divided into two main histological types: woven and lamellar bone. *Woven bone,* also known as fibrous or nonlamellar bone, is more primitive phylogenetically and consists

of collagen fibrils forming an irregular matrix. Woven bone forms the embryonic skeleton and is gradually replaced by lamellar bone by the age of one year. Woven bone may also be formed in many pathological states where there is a stimulus for rapid bone formation. For example, woven bone is the first bone produced in fracture repair and is subsequently replaced by the lamellar bone. Bone produced by the periosteum in response to infection is primarily woven until replaced by lamellar bone. Bone-forming tumors such as osteosarcoma may produce large amounts of the coarse-fibered woven bone.

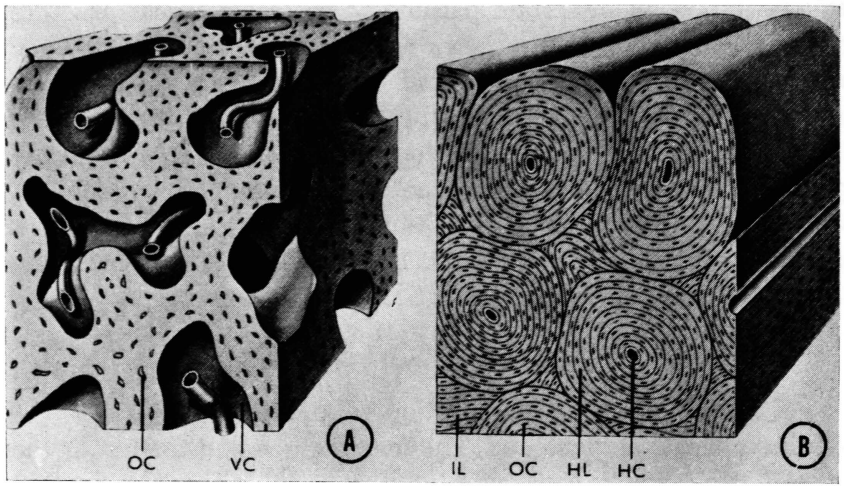

Figure 2. Diagram showing differences in structure of woven (A.) and lamellar bone (B.) HC, Haversian canal; HL, Haversian lamella; IL, interstitial lamella; OC, osteocyte; VC, vessel canal. (Courtesy of Dr. N. M. Hancox, 1972.)

In *lamellar bone,* the collagen fibrils are oriented parallel to each other within the plane of each lamella. The direction of the fibers changes in successive lamella to form a "laminated" sheet of tremendous strength. Virtually the entire skeleton is composed of lamellar bone which forms two main types of gross bone architecture: compact and cancellous or spongy bone.

Compact bone is dense lamellar bone making up the full thickness of the bone wall or *cortex*. Compact bone tissue is too thick to obtain its nutrition only from surface vessels and therefore relies on special vascular channels known as *Haversian canals*.

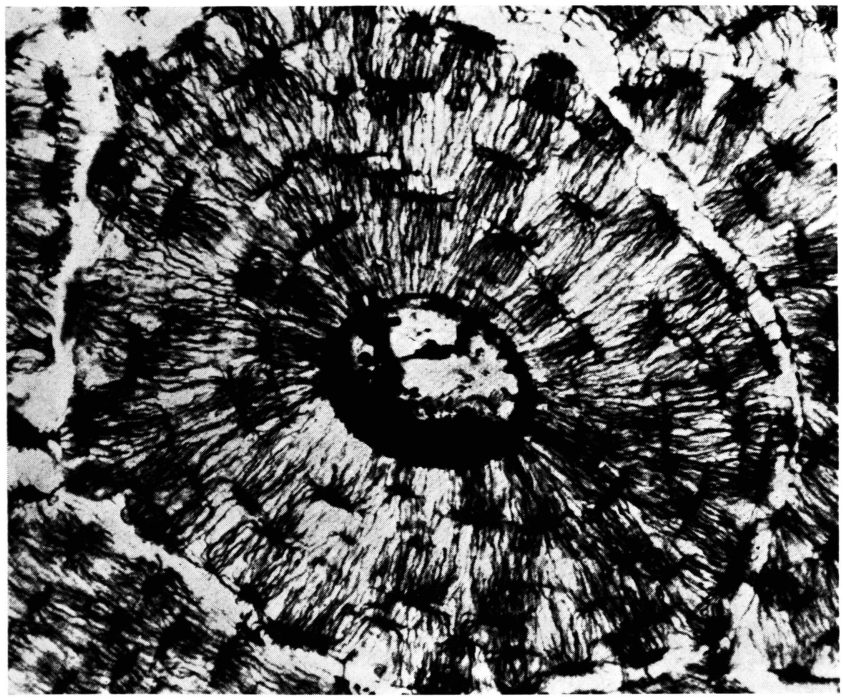

Figure 3. Ground section of human femur showing a typical Haversian canal surrounded by concentrically arranged lacunae and canaliculi. X300. (Courtesy of Dr. D. W. Fawcett from Bloom and Fawcett, 1975.)

Cancellous bone is a porous network of branching and anastomosing *trabeculae* of bone. This type of bone tissue fills the interior of the epiphyses and metaphyses with very little in the diaphyses. The thin trabeculae receive their nutrition from the surrounding blood vessels in the marrow spaces (see Fig. 4).

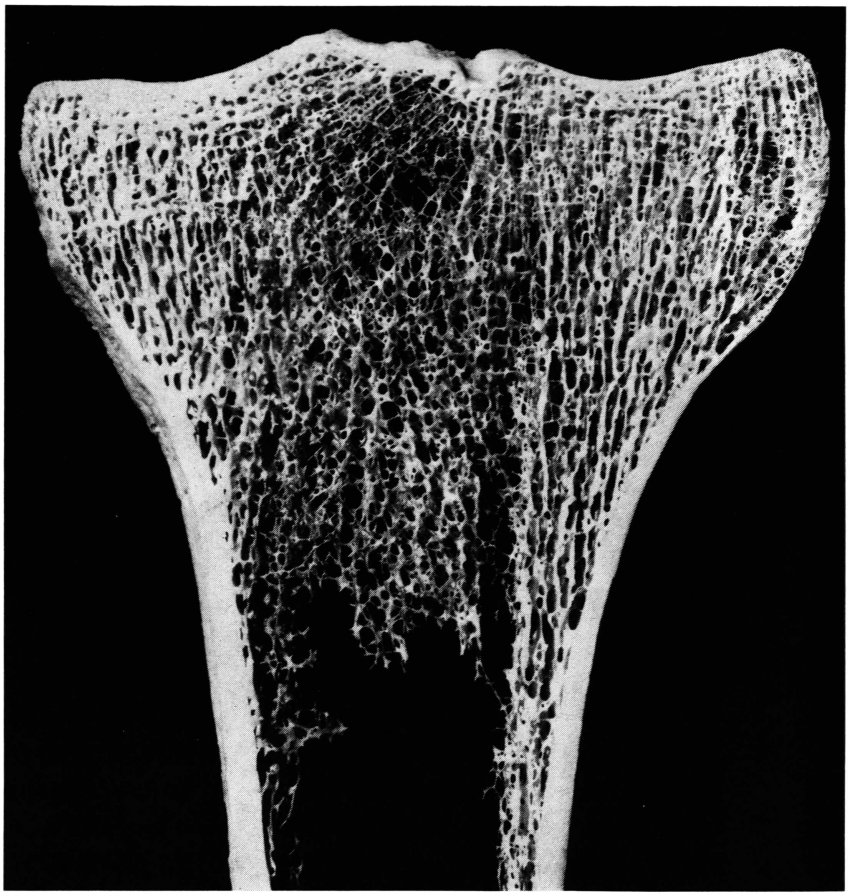

Figure 4. Longitudinal section of proximal tibia showing cancellous bone in the epiphyseal and metaphyseal regions. The cortex is composed of compact bone. (Courtesy of Dr. D. W. Fawcett from Bloom and Fawcett, 1975.)

BASIC BONE REACTIONS

Normal Bone Formation

Normal bone formation in the growing skeleton occurs by two different processes: intramembranous and endochondral. *Intramembranous ossification* is responsible for the formation of the frontal and parietal bones as well as enlargement of the other

bones by subperiosteal bone apposition. In the embryo the mesenchymal tissue gives rise to a primitive connective tissue membrane. Small groups of osteoblasts develop from the membrane to produce nonmineralized matrix or *osteoid* which is subsequently mineralized to form woven bone trabeculae. Gradually these trabeculae form a latticework of osseous tissue, and the primitive connective tissue membrane condenses on the surface of the bone layer to form the periosteum. The periosteal osteoblasts lay down new bone in the latticework of trabeculae to eventually form a solid layer of bone. Concomitantly, the woven bone initially produced is replaced by lamellar bone.

Endochondral ossification is the major process of bone growth until fusion of the epiphyses occurs. By this process mesenchymal cells differentiate into chondroblasts which form a hyaline cartilage mass roughly corresponding to the shape of the bone in the adult skeleton. Cartilage cells in the center of this "cartilaginous model" enlarge, and the surrounding cartilage matrix is resorbed. Then the cartilage cells die and are invaded by rapidly growing blood vessels. The invading blood vessels contain mesenchymal cells which can differentiate into osteoblasts and hematopoietic cells. Thus, a primary center of ossification develops and spreads from midshaft towards both ends of the bone. At a later time secondary centers of ossification appear in the cartilaginous epiphyses. These centers expand until only a thin plate of hyaline cartilage separates the epiphysis from the shaft. This is called the *epiphyseal plate,* and continued growth of the cartilage followed by bone replacement provides for growth in length of the bone until adult stature is attained.

Normal Bone Resorption

The creation of properly proportioned bones requires bone resorption as well as bone formation. Moreover, bone is a living tissue necessitating constant renewal by bone resorption as well as bone formation.

Bone resorption or deossification is the dissolution of both the organic matrix (osteoid) and its mineral content. The *osteo-*

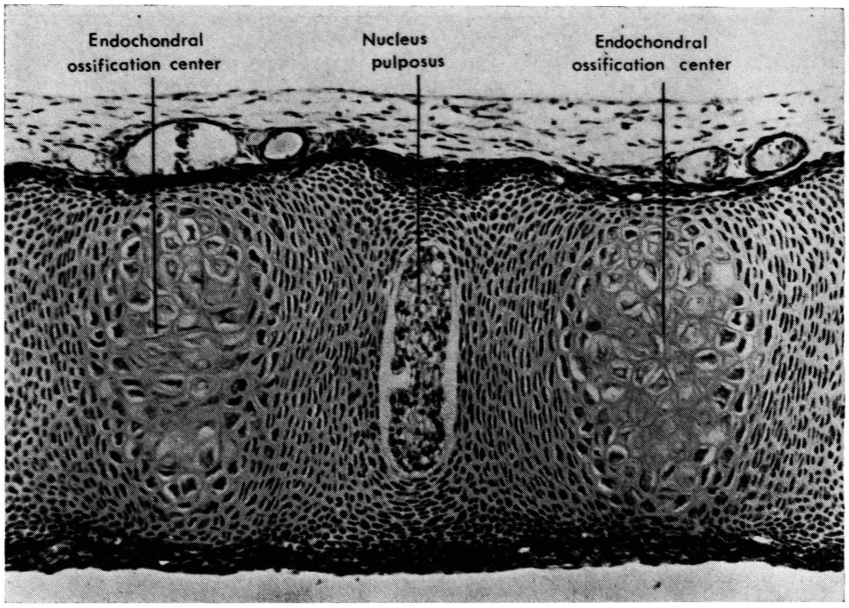

Figure 5. Photomicrograph of the cartilaginous vertebral column in a mouse embryo. In the center of each vertebra is an area of hypertrophied cartilage cells representing an early stage in the establishment of a center of endochondral ossification. (Courtesy of Dr. D. W. Fawcett from Bloom and Fawcett, 1975.)

clast appears to play the major role in bone resorption. This short-lived cell apparently arises through fusion of several mononuclear cells, but it is not known whether these cells were osteoblasts, osteocytes, macrophages, or less differentiated mesenchymal cells. Experimental evidence indicates that osteoclasts secrete both acidic substances which dissolve the bone mineral and lysosomal enzymes which depolymerize the organic matrix. This extracellular digestion releases minute bone fragments which are ingested by the osteoclasts and digested intracellularly.

BONE FORMATION AND RESORPTION IN PATHOLOGICAL CONDITIONS

Pathological conditions create an imbalance in the normal equilibrium of bone resorption and formation. Therefore, bone reacts to abnormal conditions by an increase or decrease in

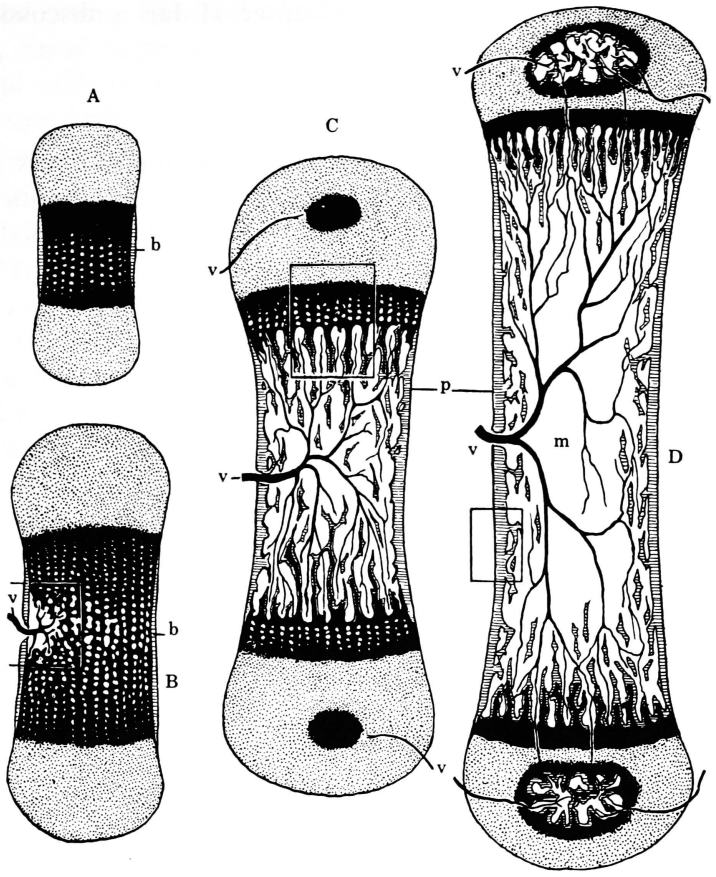

Figure 6. Diagrams of the ossification of a long bone: (A.) early cartilaginous stage; (B.) stage of eruption of the periosteal bone collar by an osteogenic bud of vessels; (C.) older stage with a primary marrow cavity and early centers of calcification in the epiphyseal cartilages; (D.) the condition shortly before birth with epiphyseal centers of ossification. Calcified cartilage in all diagrams is black; b, periosteal bone collar; m, marrow cavity; p, periosteal bone; v, blood vessels entering the centers of ossification. (Courtesy of Dr. W. F. Windle, 1955.)

the normal processes of bone formation, bone resorption, or a combination of the two processes at different locations in the bone. The repair of bone following a fracture is an excellent example of the response of bone tissue to abnormal or "super-

normal" influences. (Refer to Chapter II for a discussion of bone repair.)

Mechanical Stress

Mechanical stress is one of the most important factors affecting bone architecture. Osteoblastic activity is in some way stimulated by increased tension or compression so that new lamellae are laid down according to the lines of increased stress. Abnormally high stresses may be exerted on bones in certain pathological conditions such as rickets. In such cases the osteoblasts produce osteoid in the areas of stress, but no calcium salts are present to harden the soft osteoid. The weight-bearing bones, such as the femur, become bowed. With healing, the thick layer of osteoid present along the concave side of the femur becomes mineralized, and even more new bone may be laid down in response to the abnormal stress on the deformed bone.

The absence of stress on the bone causes a decrease in osteoblastic activity. For example, the bones of a limb immobilized by paralysis or severe fracture become atrophied as osteoclastic resorption continues without new bone replacement. The bone tissue loss results in *osteoporosis,* a general term denoting a reduction in the amount of osseous tissue (mineralized osteoid) per unit of bone volume. Disuse atrophy due to the absence of normal mechanical stress is further discussed in Chapter VIII.

Blood Supply

Both bone formation and bone resorption are active cellular processes requiring an adequate blood supply. Thus, it is inaccurate to state that an increase in vascularity (*hyperemia*) always causes an increase in bone resorption only. For example, increased vascularity is found in the metaphysis of growing bone and in a healing bone fracture. Moreover, active bone formation and resorption often occur at the same site such as in a single bone trabecula. Increased blood supply thus favors both processes.

Of even more importance is insufficient blood supply or *ischemia* which results in the death of all bone cells in the

affected area. Gradually blood vessels from the surrounding living tissue begin to invade the necrosed region, and mesenchymal cells of the vascular bud give rise to both osteoblasts and osteoclasts. Resorption of the dead bone and formation of new bone then takes place in the revascularized regions. The complete reorganization of a mass of dead bone depends both on the amount of dead tissue and the available blood supply. In certain anatomical regions, such as the head of the femur, the blood supply is very limited and aseptic bone necrosis will be long-lasting.

Inflammation

Inflammation is a normal response of tissue to an injurious agent such as an infection or fracture. The major feature of inflammation is the release of fluid, polymorphonuclear leukocytes, macrophages, and later mononuclear cells from the blood stream into the affected area. Blood vessel proliferation is also prominent. In purulent inflammations of bone, the increased pressure caused by the pus in the internal spaces of the bone leads to blood vessel occlusion and destruction. Thus, bone necrosis may be considerable in acute forms of inflammation where pus production is excessive.

New bone production is a prominent feature of inflammation. The exudative fluid and pus raise the periosteum from the bone, and this irritation of the periosteum stimulates exhuberant new bone formation. The increase in vascularity (granulation tissue) also favors the production of bone as well as the osteoclastic resorption of dead bone. In the final stages there is osteoclastic resorption of the primitive woven bone and replacement by highly structured lamellar bone.

Hormonal Imbalance

Hormones are very important in controlling bone growth, maintaining the equilibrium of bone resorption and formation, and mobilizing calcium salts from the bone to maintain a normal blood calcium level. No attempt will be made here to describe the response of bone to each type of hormone imbalance. In

general, an increase or decrease of the hormone involved causes an increase or decrease in the normal physiological response of the bone to that hormone. For example, an increased secretion of somatotropic hormone (STH) from the anterior lobe of the pituitary causes an increase in bone growth resulting in giantism or acromegaly. Hyperparathyroidism is an increased secretion of parathyroid hormone which increases the number of osteoclasts. The result is a generalized resorption of bone or osteoporosis.

Neoplasia

Bone invasion by primary or secondary tumors usually causes both bone formation and bone resorption. Bone resorption is the main feature of most invasive tumors and appears to be mediated by a stimulation of the osteoclasts by the tumor cells. Bone formation is prominent in some types of tumors, particularly those that are slow-growing. Thus, one often finds osteosclerotic metastases of bone in slowly developing prostatic carcinoma or massive bone production by a meningioma slowly invading the cranium. In both instances the bone is produced by osteoblasts which have been stimulated to lay down bone. Most of the new bone produced is of the woven type, but lamellar bone may eventually replace it in the slower growing tumors.

Tumor cells of primary bone tumors originate from the bone tissue and therefore may directly cause bone formation and resorption. Bone destruction is prominent in giant-cell tumor (osteoclastoma) due to the proliferation of malignant osteoclasts. Similarly, bone production is considerable in osteosarcoma. It should be emphasized however, that much of the bone produced in oesteosarcoma is the result of stimulating normal osteoblasts in the endosteum and periosteum.

SUMMARY

This chapter has hopefully corrected the common misconception that bone is an inert structural framework impervious to the external environment. Instead, it is composed of living cells

in a hard matrix which are quite sensitive to such influences as trauma, infection, mechanical stress, nutrition, and neoplastic growths.

With this general introduction to the gross anatomy of bone and its response to various pathological conditions, let us now examine specific types of gross bone pathology. The discussion will stress the gross features of bone pathology as these are of the greatest importance in diagnosing the specific disease entity from a dried bone specimen.

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