Bone in Clinical Orthopaedics

A STUDY IN COMPARATIVE OSTEOLOGY

Edited by

G. SUMNER-SMITH, B.V.Sc., M.Sc., F.R.C.V.S.

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FOREWORD

During the last two decades, surgical treatment of bone fractures and prosthetic replacement of diseased joints have gained widespread use in human and veterinary medicine. At the same time, a certain lack of knowledge of bone reaction to artificial environments became apparent.

Besides causing pain and limiting activity, fractures of the bone and degenerative changes of the joints result in a high toll on a purely economic level. As in Switzerland, in most countries fractures are observed in only a small percentage of the general trauma population. They nevertheless cause about half of the total costs of all trauma. The temporary or permanent loss of working capacity results in a multiple of the costs compared to rescue, therapy, and rehabilitation. Optimal treatment of the traumatologic bone disease has, therefore, a good chance to result in an important improvement for both the patient and the community.

A variety of different therapeutic approaches to the diseases mentioned rely on the use of supportive or replacement implants. All of these, while restoring early function of the limb, cause marked changes in the vascular, metabolic, and physical environment of the bone. This demonstrates the importance of a thorough knowledge of bone behavior, thus providing a sound basis for surgical and noninvasive treatment. This applies equally to the treatment of both acute bone trauma and chronic joint disease.

Geoffrey Sumner-Smith has masterfully activated and welded a team of experts to provide us with a broad and deep insight into the development, biology, and bioengineering of intact and diseased bone. This book brings together the knowledge of veterinary and human medicine. It is as well suited to be read from cover to cover by the student as it is to be used as a reference by the expert clinician.

STEFAN M. PERREN

PREFACE

The intent behind the production of this text is to form a bridge, or more properly a "firm callus," between the literature available for undergraduate students of orthopaedics and those intending to study bone in depth. Many texts already exist for the undergraduate and also for the student of advanced orthopaedic surgery. It is hoped that this book will set the stage for graduate training in orthopaedics for surgeons in the medical, dental, and veterinary fields. It is my belief that no one should undertake a detailed study of orthopaedics, as a subject, before he or she becomes familiar with much of the material presented herein. Some of it is in outline form, but in other areas the contributor has taken the theme to considerable depth. In some chapters the material is not of a nature that is often altered by new contributions to the particular subject being described. I hope this type of text permits recent findings to be put before the reader in a succinct form.

It is a matter of particular pleasure and pride to me, as the editor of this text, to be able to present such a galaxy of experts. The fact that each contributor responded, readily and with enthusiasm, to the invitation to contribute, convinced both the publishers and me that there is a need for such a text. The reception of the results of the contributions of so many people by you, the reader, will prove us right or wrong.

The production of a multiauthored text is not without its difficulties; naturally there is some overlap of subjects of one chapter with another. I believe this to be a healthy situation and, even though opinions may differ in minor details, it serves to show the serious student of orthopaedics that all is not quite so cut and dried as one might have been led to believe from undergraduate teaching.

G. SUMNER-SMITH

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To the editors at W. B. Saunders Co. I wish to extend my thanks for their faith in agreeing with my suggestion that this book be prepared. My initial company editor was Mr. R. W. Rheinhardt, whose place was later taken by Mr. R. Kersey. To these two gentlemen and their stalwart colleagues I extend my thanks. My sincere appreciation is also due to Elizabeth Peiper, who typed the final manuscript prior to submission to the publishers. Through the long gestation period she was patient and tolerant with me and produced an immaculate manuscript. Without her diligence and sensibility, the manuscript would still be on my desk.

Finally, it will be obvious to the reader that without the labors of the contributors the book would never have seen the light of day. To these people who had faith in my idea, my heartfelt thanks.

G.S-S.

CONTENTS

CHAPTER 1

DEVELOPMENT OF BONE

V. S. DEKLEER

1.1. INTRODUCTION

This chapter deals with bone as an entity in man and dog.

Two things must be made clear at the outset, although they will become self-evident as the reader progresses through this book. First of all, the subject matter is described as orthopaedics, but it is that and much more — it goes far beyond the original literal meaning of *'ορθόσ* (straight) and *παιδίον* (child). On occasion it is concerned with straight structures, but much of the study of bone development is concerned with normal arcs which may be along the plane of a bone, as in the frontal bone, along its axis, as in some long bones or the ribs, or in series, as in the spinal column, which shows a variety of curvatures throughout the mammalian class. Our study will progress from prenatal development through youth, adulthood, and into a discussion of geriatric changes. Straightness and childhood properly remind us of the classic origin of such studies so that we may better appreciate the advances that have been made over hundreds of years, most particularly during the last quarter of a century.

Secondly, although we are primarily concerned with bone, we must not, and indeed cannot, deal with pure tissue beyond that which may be contained in an individual Petri dish or on a slide. Blood and nerve supply, adjacent musculature, actual weight, the relative location to other body parts, even life style are some of the factors that constantly bear upon the structure of simple bones. By

its nature, bone withstands decay and subsequent destruction even when long dead, thus enabling us to analyze the skeletal remains of prehistoric creatures. According to Trueta (1968) the basic physiology of vertebrates has been virtually unchanged over 500 million years of their existence, and this suggests structural as well as functional continuity. Fossil records go back well over 60 million years to the Paleocene epoch, the beginning of the age of mammals. Fossil remains 2000 years of age provide invaluable information to the anthropologist. Yet, in spite of eons of time, bone is probably the last of the body tissues to have been developed. From an evolutionary point of view (Hancox, 1972) this occurred 400 million years ago. However, different bones do not have equal ability to withstand external physical and chemical forces. In man teeth, the hardest tissues of all, are most resistant and mandibles survive quite well, as do the femur, humerus, scapula, clavicle, tibia, and fibula. The rarest bones to find are those of the pelvis, distal extremities, vertebrae, and forearm (Walker, 1979). The petrous bone may be all that remain in the stomach of carnivores such as whales, giving us information, at least in part, about their diet and feeding habits.

Although it may be fascinating to study the histologic architecture of prehistoric animals, and to reconstruct their probable appearance from such evidence in spite of the fact that remains of bone reveal far more about the animal than its framework, these facts bring out the point that bone is not uniform. As we study development and the individual cells that serve as building blocks of this architecture, it may be assumed that all bone is alike wherever it is found in the body, but such is not the case.

Within the individual, from the time that a given bone may be recognized until shortly after the clinical death of the body in which it was housed, bone is continuously growing and/or changing. This involves processes not unlike modeling with clay — the constant addition of a bit here or there, balanced by pieces that must be eroded or carved away. Bone is never left alone but is subjected to chronic change, always being pestered at its ends, from the surface, and from the center of its shaft. It is highly specialized and yet capable of repair. The ancient statement that "the more a thing changes the more it remains the same" is apt.

Considering their individual environments and circumstances, there are remarkable similarities between bones of species that vary markedly in design. Mouse, man, and mastedon, creatures that are biped or quadruped, those that walk, gallop, jump, fly, or swim — all have comparable individual bones. There is a master pattern with a wide possibility for adaptation to need. Although skeletal structures have long been described as a mechanical system of levers ingeniously arranged, we must not forget that we are concerned with a plastic, living frame. Bearing all these archeologic and zoologic facts in mind we are better prepared to investigate the development of bone, cell by cell, process by process, in twentieth century mammals.

1.2. PROCESSES OF BONE FORMATION

Of the original fetal layers the **mesenchyme** is of prime interest, for it contains the potential for bone production. Although bone may develop in one of two ways, there is only one actual process of bone formation. Since cartilage initially occupies the space and form in many places that is to be taken over by true bone, we will consider it first. Often ambiguous and even erroneous statements are made which may lead one to believe that cartilage turns into bone. This never happens and, simply by investigating the blood supply, it is obvious that it cannot occur. The second of the two locations in which bone may develop is suitable for flat rather than long bones, and the bone that arises in these areas is known rather inaccurately as membrane bone.

Although two processes each lead to one formation of true bone, microscopic examination of adult tissue will not provide any clues as to where or how it originates. It must be emphasized that those terms which are in

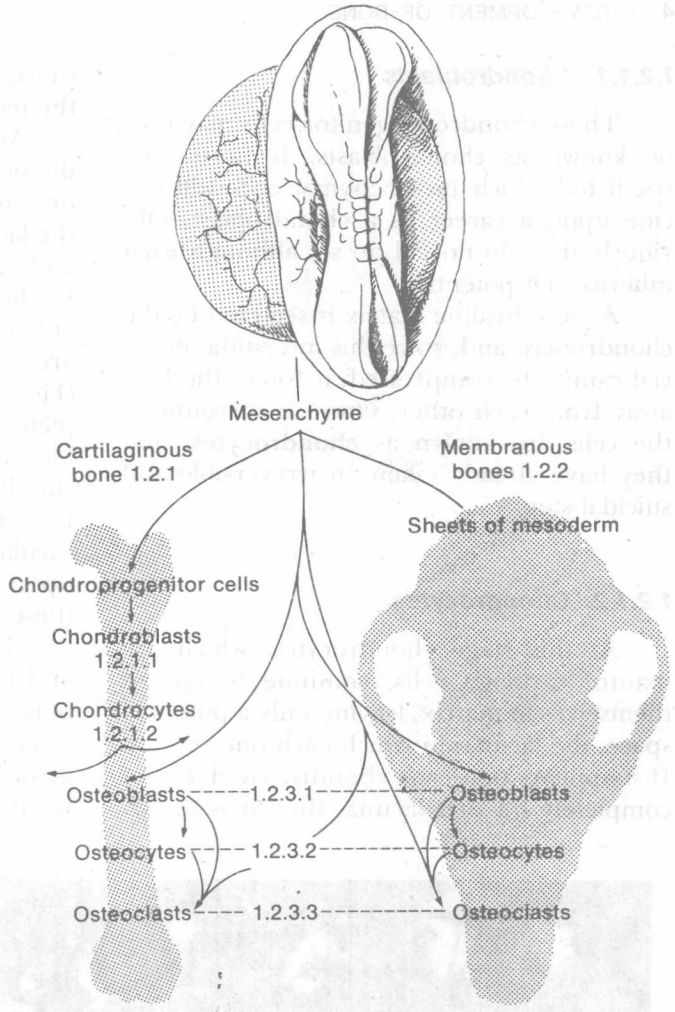

Figure 1–1. Origin of cartilaginous and membranous bone. Mesenchyme arising as an early fetal layer has the potential to develop in different ways, giving rise to true bone that has a cartilaginous or a membranous precursor. The numbers refer to sections in the text. (Original drawing by Mary Trafford, School of Art as Applied to Medicine, University of Toronto.)

common usage must be understood in the context of development, and not in terms of the finite product. Figure 1–1 will clarify this point.

1.2.1. Cartilaginous Bone

Cartilage, derived from **mesenchyme,** is the forerunner of this type of bone. There are in fact three types of cartilage, only one of which is of particular interest in the development of bone:

1. **Elastic cartilage** is not involved with osseous structures.

2. **Fibrocartilage** is limited in distribution. It is really an intermediate or transitional stage between cartilage and dense connective tissue. It is closely associated with the connective tissue of the joint capsule, and with ligaments. Although it may be found in some articular cartilage and in intervertebral discs, the pubic symphysis, teres ligament, and the attachment of some muscle to bone, it is not of concern to us here.

3. **Hyaline cartilage** is the type that predetermines the shape of many bones. In a typical long bone, such as the femur, the first indication that a cartilage site is being prepared is that the mesenchymal cells in the area look crowded (Ham, 1979). These centers of chondrification contain tightly packed cells that form a model of the bone that they preform. Under magnification (× 400) it is not possible to define cell boundaries nor, since they have been retracted, will cell processes be evident (Bloom and Fawcett, 1975).

1.2.1.1. Chondroblasts

These chondroprogenitor cells may now be known as chondroblasts. It cannot be predicted which mesenchymal cells will decide upon a career as a chondroblast. Obviously they do not all do so, although each inherits that potential.

A clear hyaline matrix is secreted by the chondroblast and, since this interstitial material cannot be compressed, it forces the cells away from each other. Once so surrounded the cells are known as **chondrocytes**, and they have already taken an irreversible and suicidal step.

1.2.1.2. Chondrocytes

At this stage chondrocytes, which are mature cartilage cells, continue to embed themselves in matrix, leaving only a potential space, the lacuna, in which each one resides. If it appears that each chondrocyte does not completely fill that lacuna, then it is an artifact. Such apparent cell shrinkage is due to the technique of preparation (Ham, 1979).

When the chondrocyte undergoes cell division once or twice more, the resulting two or four cells are temporarily trapped within the lacuna. The chondrocyte responds to this and, shortly after the division has occurred, a further partition is built up between the individual isogenous daughter cells so that they are held in isolation in secondary lacunae (Fig. 1–2). This solitary confinement is complete, not only from cells of their own kind, but there are no nutrient routes and no blood supply. This is most important to remember, for it is one of the prime differences between cartilage and bone and has a profound effect upon the difference in healing capabilities of these two tissues.

This activity constitutes *interstitial growth* and is seen in young cartilage but never in bone. Figure 1–3 shows both types of growth, interstitial and appositional. Interstitial growth which results in longitudinal growth is characterized by cell division within the

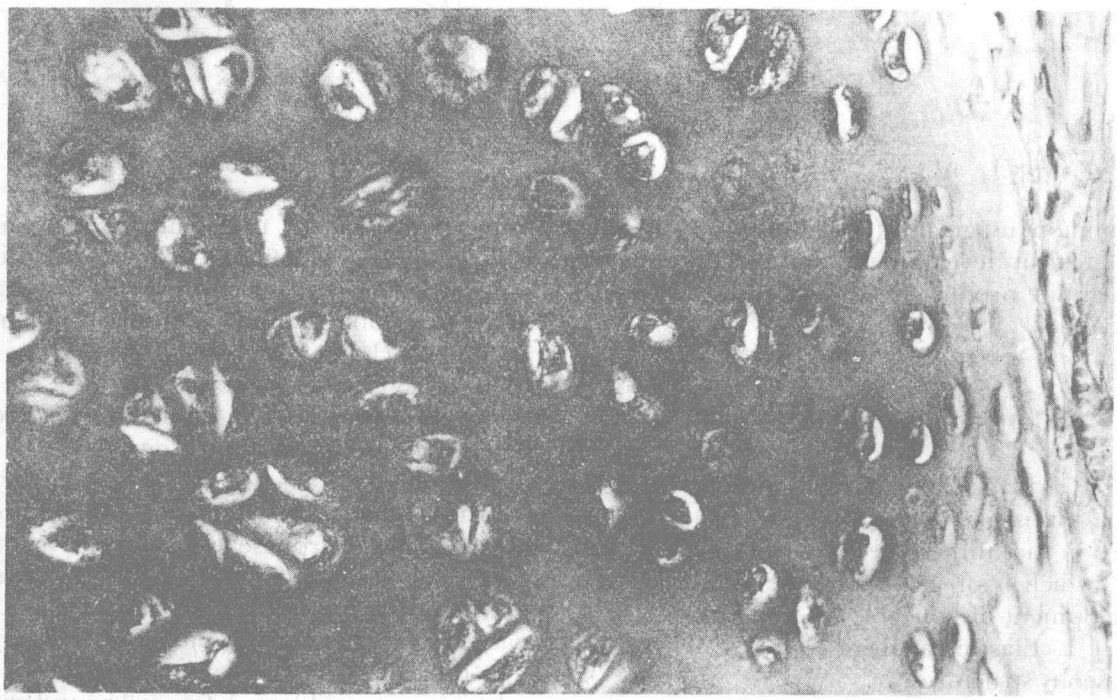

Figure 1–2. Hyaline cartilage from the trachea of a rabbit. Note the more intense staining area of territorial matrix around the groups of cells forming isogenous nests. The chondrocytes immediately under the perichondrium (top) are single and fusiform and represent appositional growth. Hematoxylin and eosin × 780. (Courtesy of Dr. M. K. Bhatnager, Department of Biomedical Sciences, Ontario Veterinary College.)

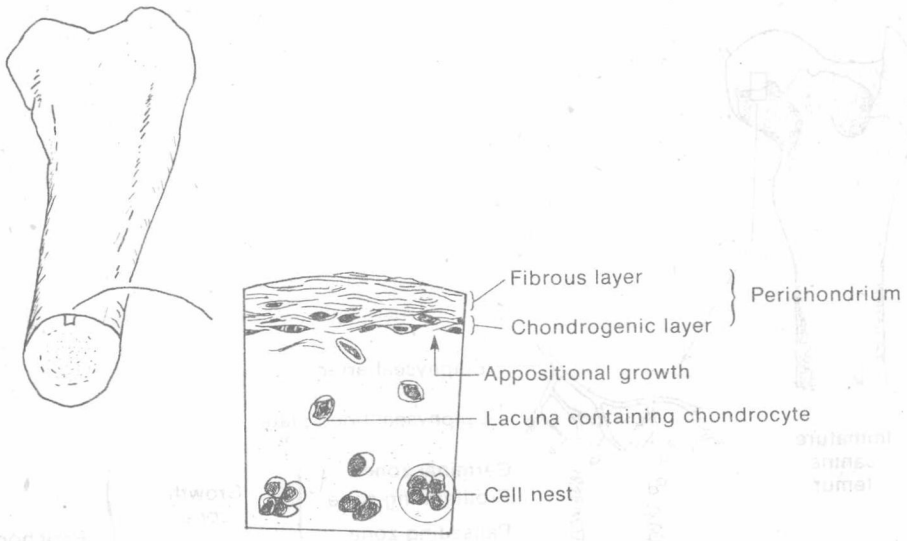

Figure 1–3. Cell division in hyaline cartilage. Diagram to illustrate the principles of appositional and interstitial growth. (Redrawn from Ham, 1978.)

primary lacuna. Because the cells divide in one plane they appear to be stacked (Fig. 1–4). Rang (1969) has described them as forming palisades. In spite of this multiplication each column maintains an approximately even height, since as fast as they are produced at one end the cells at the opposite end are removed (Rang, 1969). Obviously, if a bone is to be preformed in a rapidly growing embryo, this pattern of growth leads to a corresponding and required increase in length, necessary for correct adult proportion. A numerically identical multiplication of cells in all directions would lead only to globular enlargement. Appositional growth must supplement longitudinal advance in order for true proportion to be maintained and achieved.

A chondrogenic layer is seen, as before, but it encircles the cartilaginous bone model, forming a thick perichondrium. The deeper, interior cells of this sleeve remain undifferentiated to the extent that they can divide, and thus contribute to radial growth, building up much like the bark on a tree. The external or fibrous layer of the perichondrium surrounds the chondrogenic layer, but must itself permit growth.

The nucleus of each chondrocyte is described variously as irregularly ovoid (Ham, 1979) or large and symmetric (Rang, 1969). The number of **nucleoli** vary according to the species of animal from which the cell was removed. The Golgi apparatus is well developed, generally localized, and close to the nucleus, which it may indent (Ham, 1979). It enlarges when intercellular matrix is being produced. The saccules of the Golgi apparatus are dilated (Bloom and Fawcett, 1975), but this effect is reduced when the cartilage is less active and is not concerned with growth or repair. Cytoplasmic footlets are rather triangular processes that indent the matrix at several places around the periphery of the cell. Cartilage **matrix** looks homogeneous because the refractive indices of collagen and the ground substance in which it lies are similar. The walls of the vacuole that encompass the chondrocyte take a darker basophilic stain than the bulk of the interstitial substances and is known as capsular matrix, although it is not a capsule (Bloom and Fawcett, 1975) as we usually understand that term (such as when applied to the kidney or spleen). This increase in affinity for stain may be due to a higher proportion of mucopolysaccharides in the surface area (Fig. 1–2). This is a characteristic appearance in growing cartilaginous tissue.

The chondrocytes enlarge, vacuoles ap-

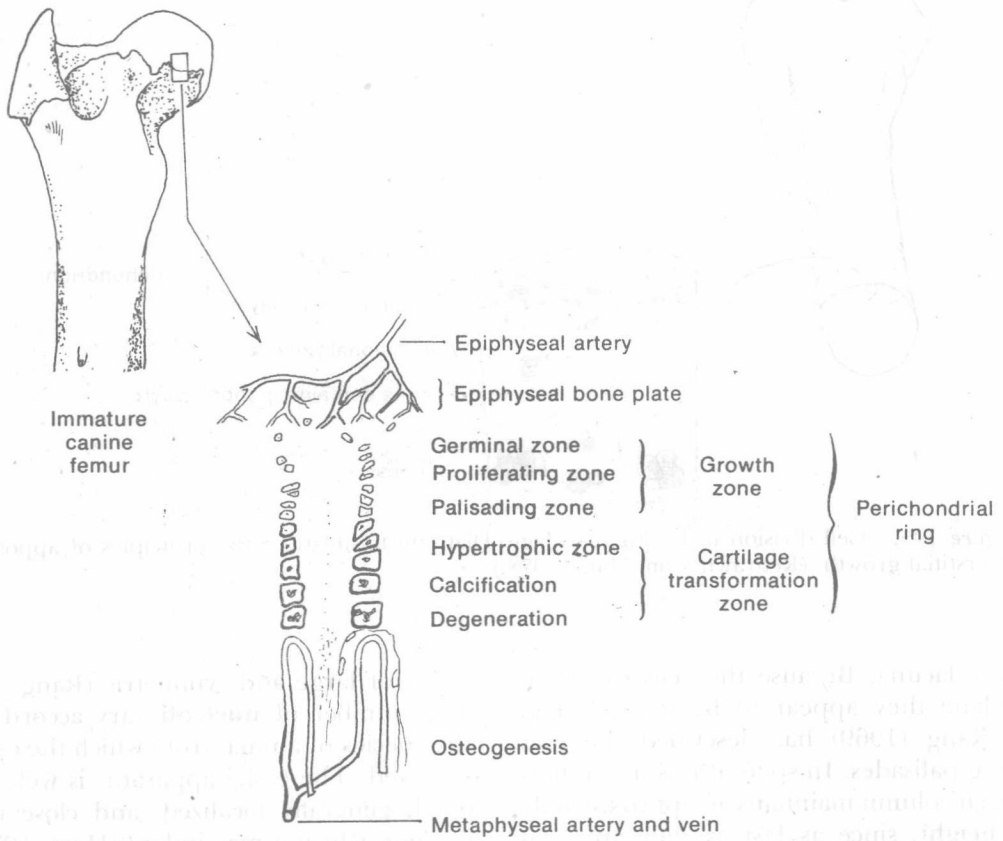

Figure 1–4. Typical growth plate. Diagram to show the zones involved in a growth plate. (Redrawn from Rang, 1969.)

pear in their cytoplasm, matrix is reduced, and the small amount of matrix that does remain may calcify. the cells themselves die and subsequently degenerate, having effectively cut themselves off from any supply of nutrition.

1.2.1.3. Regressive Changes in Cartilages

Although regression is generally understood as a backward and undesirable procedure, it is a necessary forward step in the growth of an individual. The steps followed are proliferation-maturity-hypertrophy-degeneration, which is essential since it permits replacement (Bloom and Fawcett, 1975).

Up to now, although there are no capillaries in the immediate area, some exchange may have taken place between the chondrocyte and its surrounding matrix since three quarters of the matrix is fluid, although grossly it does not appear to contain that much; its appearance is rather solid. Once crystallization occurs and there is calcification of the matrix, diffusion is impossible and the cells in the area subsequently die, and fairly quickly, owing to insufficiency or total lack of nutrients and waste removal. Replacement may then be made by true bone tissue.

1.2.1.4. Repair of Cartilage

Cartilage grows with facility but repair is a different matter, being both slow and limited. This is not surprising when one considers the meager or absent nutrient supply. Following degeneration of cells that have been traumatized, as well as damaged matrix, repair is

by means of connective tissue (**metaplasia**). Since damaged tendon and cartilage are so slow to undergo repair, the clinician may prefer to see an honest bone fracture than apparently lesser damage to soft tissue (this will be discussed fully in Chapter 11).

1.2.2. Intramembranous Ossification

The descriptive term "membrane bone" is frequently used in regard to development; however, intramembranous ossification is a more accurate way of describing the situation (Ham, 1979). Flat bones such as those of the brain case (frontal, parietal, occipital, and temporal bones) tend to have their "blueprint" laid down in membranous sheets, rather than being preformed in cartilage. Here there is cellular proliferation, as was the case with cartilaginous bone. Connective tissue in the area develops an excellent blood supply, and the individual cells contact each other by means of long processes (Fig. 1–5). These processes are of vital importance, since the canaliculi follow their route (Bloom and Fawcett, 1975).

Collagen fibers are scattered throughout the ground substance. Condensations in the matrix soon occur, and these take on an eosinophilic stain as a branched pattern. The processes of the surrounding cells become shorter, and the cells themselves become either cuboidal or columnar in type. These cells have an affinity for basic stain and are called osteoblasts at this stage.

1.2.3. True Bone

It should be noted here that bone never arises as a primary tissue. The predecessor of endochondral ossification is somewhat more complicated than that of many flat bones, in

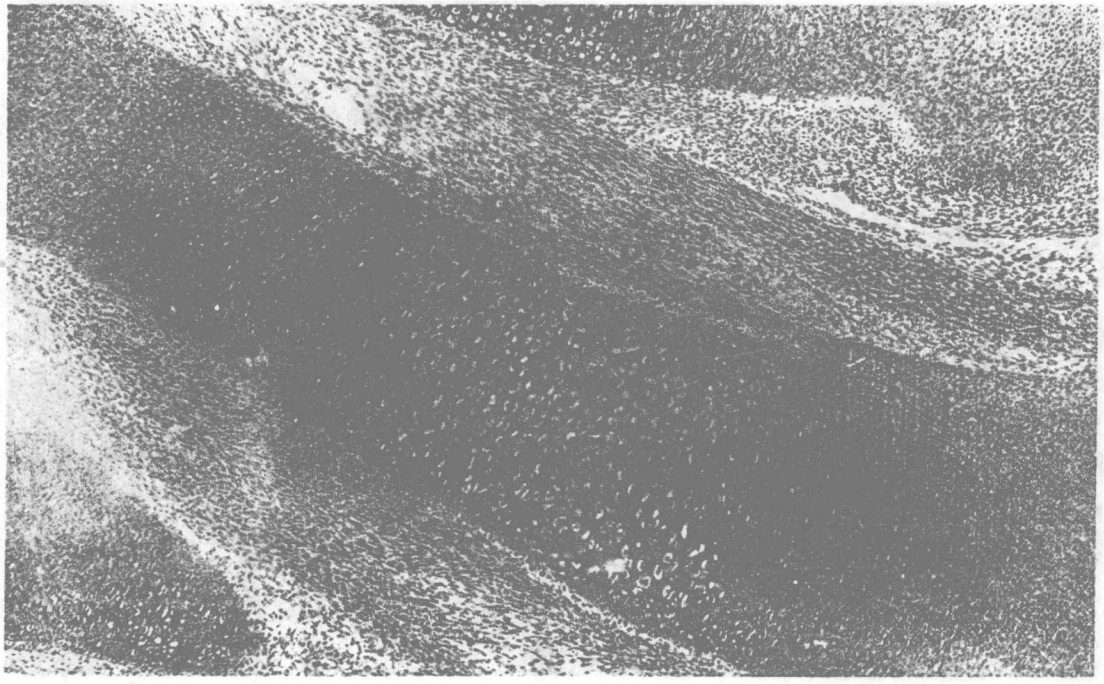

Figure 1–5. Endochondral ossification. Longitudinal section of the metatarsal cartilaginous primordia of a pig fetus. This is a very early stage of primary ossification center. The chondrocytes in the mid-portion of the shaft are hypertrophied. The perichondrium shows differentiation into an outer fibrous (Pf) and an inner cellular (Pc) layer (H and E stain; × 80.) (Courtesy of Dr. M. K. Bhatnager, Department of Biomedical Sciences, Ontario Veterinary College.)

which a simpler, less sophisticated connective tissue is replaced by bone. In either case the final method of building bone, regardless of the foundation, is the same. Osseous cells progress from immaturity to senility, and at each stage they may be recognized and identified. Bone can grow only by appositional, never by interstitial, growth (Ham, 1979). Almost as soon as it is formed bone matrix begins to ossify, and it cannot therefore expand within its own now solid surrounding matrix. All bone growth must be from a pre-existing surface which may be specialized, as is the growth plate.

1.2.3.1. Growth Plate

The longitudinal growth of bone, which is so rapid in the fetal mammal, is achieved by the highly specialized growth plate. Growth plate is a more accurate term than epiphyseal plate, since the epiphysis is moving away from the center of the bone, relatively, while growth occurs at the metaphysis. It is the shaft and not the extremity which grows (Figs. 1–6 and 1–7).

Since long bone is irregularly circular or oval on transverse section, so the growth plate is an irregular disc in shape. In fact, the diameter of the bone is greatest at the level of the growth plate. In limited areas, such as short bones, the growth plate may be spherical (Rang, 1969). At this stage the cortex and medulla are not differentiated; this occurs subsequently during remodeling, and simultaneously there is nearly always a decrease in the diameter of the bone.

1.2.3.2. Osteoblasts

The origin of osteoblasts, as for chondrogenic cells, is the multipotential mesenchymal layer. Such cells differentiate during the normal course of development. However, more may be induced to become osteoblasts owing to the pressure of stress or injury. By

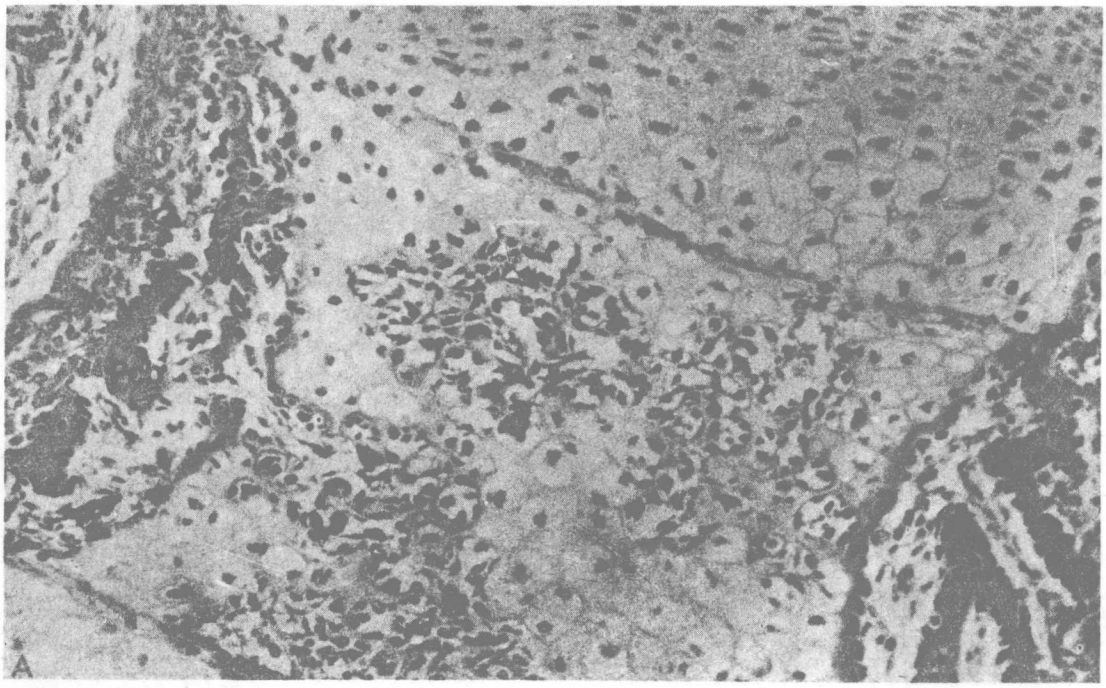

Figure 1–6. *A,* Endochondral ossification. Longitudinal section of the metatarsal primordia of a rabbit. Note the progression in the development of primary ossification center. The perichondrium (P) is well differentiated into two layers. The periosteal collar of bone (Pcb) is being laid down from the osteogenic cells from the periosteum. The central cartilage displays degeneration of chondrocytes and invasion of the lacunae with mesenchymal cells from the periosteum. (H and E stain; × 192.)

Illustration continued on opposite page