THE PATHOLOGY OF ARTICULAR AND SPINAL DISEASES

BY

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TO MY WIFE

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PREFACE

In this book I have sought to provide an up-to-date and adequately illustrated account of the morbid anatomy of the joints and spine. This is a branch of their science which pathologists, with a few notable exceptions, have tended to neglect and which is inadequately represented in most standard pathological text-books. In clinical medicine and surgery an increasingly large number of practitioners to-day specialise in the diagnosis, treatment and investigation of disorders of the skeletal system. Few of them have had the opportunity of studying these diseases in the post-mortem room or in the histological laboratory and much of their knowledge of the pathological processes involved is founded on radiology, which is only the shadow without the substance of morbid anatomy. Radiography is, of course, an indispensable tool in the study of bone and joint disease, but many excellent radiological atlases already exist and, since a particular purpose of this book is to supply illustrations of anatomical dissections and microscopical preparations, I have used only a few radiographs to demonstrate special points.

It is my hope that this book will appeal not only to pathologists, but also to orthopaedists, rheumatologists and radiologists.

The introductory chapters deal with the anatomy and physiology of the joint tissues and bone. Thereafter, I have arranged the subject matter in a conventional pathological sequence, as indicated in the table of contents, inserting descriptions of those diseases whose cause is still unknown in places which at the present time seem most appropriate. I have tried in this way to present a connected story in which the several diseases of joints and spine are related and compared with one another and orientated within the framework of general pathology.

It has been necessary to allude frequently to disorders of bone, but other books contain good descriptions of the general diseases and tumours of bone. To avoid redundancy I have compressed a short survey of bone pathology into a single chapter (Chapter III), written mainly from the standpoint of the clinician who has the X-rays of his patient in his hand. Tumours of bone, except where the spine is concerned, receive no more than mere mention.

The illustrations are all original. They have been made from material which I have myself prepared or to which I have been given access. Most of them are here published for the first time.

The lists of references at the end of each chapter include those which substantiate points raised in the text and which also should help the reader who wishes to pursue the further study of particular subjects. They do not constitute a full bibliography. Many more papers and books have been read and their essence distilled, and I offer an apology to any author or colleague not quoted by name who might recognise a thesis of his own which I have unconsciously adopted without acknowledgement.

This book is the outcome of fifteen years' special interest in bone and joint pathology. Many colleagues, during this time, have kindly helped me to extend my acquaintance with the subject from the laboratories to the wards of the general, orthopaedic and rheumatism hospitals at Leeds, Sheffield and Harrogate, in which my work has been carried out.

Finally, I have not shirked from speculating upon the aetiology of those diseases whose true cause is still obscure. Rheumatoid arthritis and rheumatic fever, for example, possibly result from the workings of pathological mechanisms which we cannot at present define. No conclusions as to the nature of these diseases can yet be drawn from the promising new investigations with adrenal and pituitary hormones, but references to this important work are included together with discussions of older theories. The major part of the book, however, is devoted to an objective record of pathological phenomena which are themselves unchanging even though our interpretation of them may change with advances in medical knowledge.

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Dr. Philip S. Hench of the Mayo Clinic, Rochester, Minn., has given me many important suggestions and the most helpful criticism of my plans. He has generously sent me books from his own library and a classified bibliography prepared from the thousands of papers which have passed through his hands as chairman of the editorial committee of the Rheumatism Reviews.

I owe a special debt of gratitude to Mr. J. Hainsworth, the Leeds Medical School photographer, who has prepared over 170 of the illustrations specially for this work and whose technical and artistic skill has revealed the salient features both of gross specimens and of microscopical sections.

To all of these and to many others too numerous to name I express my warmest thanks. Lastly, the book owes a great deal to my wife, not only for the continual encouragement she has given its author, but also for her great help with translations and her detailed editing and revision of the manuscript.

D. H. C.

CONTENTS

CHAPTER I

GENERAL ANATOMY AND EMBRYOLOGY OF THE JOINTS

ANATOMY

Types of Joints

Diarthroses (*Di-*, *dia-*, =through or across) are the freely movable true joints endowed with a synovial cavity, capsule and synovial lining.

Synarthroses (*Syn-*, =together) are the immovable unions between separate bones or between the epiphysis and shaft of the same bone. They play an important part in skeletal growth but, apart from certain developmental disorders, they are of no pathological interest.

Amphiarthroses (*Amphi-*, =both, of both kinds) are slightly movable joints combining some of the features of each of the former types. The simplest amphiarthrosis is the *syndesmosis* or ligamentous joint, such as the articulation between the lower ends of the tibia and fibula. A more complex variety is seen in the intervertebral disc and between the pubic bones. Here both bony surfaces are covered by hyaline cartilage but are separated by a mass of fibrocartilage instead of by a synovial cavity. This sort of joint is called a *symphysis* (=a growing together). The intervertebral discs are of peculiar origin and structure. Theirs is a strikingly individual pathology which will be dealt with in a later section (Chapter XIV). For the present we shall concern ourselves solely with the structure, function and diseases of the diarthrodial or true synovial joints.

The diarthrodial joints in man are all paired structures with the exception of the centrally placed pivot joint between the atlas and the odontoid process of the axis. Their total number can be variously computed but, having regard to the most usual arrangement of synovial compartments in the carpus and tarsus and counting each compartment as a distinct joint, the figure of 187 is arrived at. Of this number of joints, about one-half belong to the axial skeleton and one-eighth to each of the four extremities. Some joints include a number of bony articulations within a single synovial stratum. Eleven bones of the carpus and metacarpus interarticulate by means of about a score of interfaces within a single joint capsule. The knee, which is the largest joint in the body, is formed of three separate interfaces. Many of the small joints, however, such as those between the phalanges and those between the articular processes of the vertebrae, display only two opposing surfaces.

The anatomy of the individual joints is fully described in many standard text-books, and it is only necessary here to recall examples of the main types of diarthroses which are classified according to the kind of movement they permit:

Ginglymus or hinge joint	(e.g. humerus-ulna, interphalangeal)
Trochoid or pivot joint .	(e.g. upper radius-ulna, atlas-odontoid process)

Arthrodial or plane joint	(gliding movement—e.g. posterior intervertebral and most intercarpal and intertarsal joints)
Condyloid joint . .	(movement in two axes without rotation—e.g. radiocarpal)
Enarthrosis or ball and socket joint	(free movement around all axes—e.g. hip and shoulder joints)

This classification of joints according to their kind of movement is of little importance in itself to the pathologist, who must also consider other aspects of joint mechanics. The distance traversed by one surface moving on another, the points of contact at which cartilages are subjected to both pressure and movement, the situation of the capsular attachment, the extent of the synovial tissues and cavity and the presence of interarticular fibrocartilages and ligaments, these are the anatomical facts which show their influence upon the appearance, incidence and severity of morbid changes in different joints.

Components of a joint

Bone. The ends of the long bones and the short bones which participate in diarthrodial joints are of cancellous structure and possess only a thin shell of compact cortex and a subchondral plate of continuous bone, upon which the articular cartilage rests and to which it is firmly attached by a series of tooth-like insertions and by the continuity of the fibril system of the cartilage with that of the bone. Bone surfaces lying within the joint and not covered by cartilage are sheathed in fibrous tissue representing the blending of periosteum with reflected synovial membrane. The trabeculae of cancellous bone are normally arranged in such a way as to give great strength, but spongy bone is a labile structure in which movement of bone salts and resorption and regeneration of matrix are continually taking place. Normally the gross shape of the bone ends is maintained throughout life but, under certain circumstances, extensive reshaping can occur.

Articular cartilage. A plate of hyaline cartilage, varying in thickness from 2 to 5 mm. in different joints, covers every bony face on which movement is made. In health its surface is perfectly smooth and its margins merge with the synovial tissues attached to the bone edges. In infancy articular cartilage is a pale translucent blue but changes to an opaque creamy yellow in early adult life. This change in colour seems to accompany a change in physical properties, for the youthful cartilage is compressible and resilient, a character gradually lost with age. There is at the same time an alteration in the size and distribution of the cartilage cells and of the relative proportion of cells to matrix.

Interarticular fibrocartilages are found as menisci incompletely dividing the knee joint or as discs completely dividing the cavities of the temporomandibular, sterno-clavicular and, sometimes, the acromio-clavicular joints. A fibrocartilaginous disc of rather different form completes the

distal radio-ulnar joint and separates the ulna from the carpus. All these structures and all intra-articular ligaments and tendons are covered by a thin investment of synovial membrane.

The articular capsule is a sleeve of connective tissue enveloping each joint. The outer layer (*stratum fibrosum*) is of fibrous tissue, varying in density from loose areolar tissue to closely packed bundles of collagenous tissue recognisable as distinct ligaments in sites which are constant to each joint. The capsule is originally attached to bone at the level of the junction of epiphysis with diaphysis but later, as a result of movement and muscular traction, the attachment often shifts away from the epiphysial line. Part of the shaft may come to lie well within the capsule, or part of the epiphysis may appear outside the capsule. The lower end of the femur provides an example. Anteriorly much of the diaphysis lies within the joint but laterally and medially the capsule has moved on to the epiphysis.

The capsule is lined by an inner layer (*stratum synoviale*) which covers everything within the joint except the articular cartilages. This is the *synovial membrane*, a more cellular structure than the fibrous stratum upon which it rests and one that carries many blood vessels, lymphatics and nerves. It is closely attached to the ligamentous parts of the capsule, where it consists of little more than a single layer of flattened cells, but in the laxer parts it can be stripped away from the stratum fibrosum. Villous projections from the synovial lining are present in all joints, though sometimes requiring the use of a hand lens to be seen. In the larger joints some villi are easily seen. The synovial membrane may be said to begin at the articular margin. It clings to the periosteum as it passes down the bone to the line of the capsular attachment, then, sweeping round the joint cavity to its termination at the opposing articular margin, it sends out folds to cover any fatty pads, fibrocartilages or ligaments which project into the joint space.

Synovial fluid is a viscous, clear, yellowish liquid filling the joint space and serving the double function of a lubricant and a vehicle whereby the avascular cartilages are nourished.

Blood vessels are present in the stratum synoviale in great profusion. They are most evident to the naked eye around the bone ends near the joint margin where they form the *circulus vasculosus* of anastomotic channels, but a section of any part of the synovial membrane, except the most fibrous areas, will reveal numerous capillaries just beneath the lining layer of cells. The rich plexus of vessels can best be seen in an inflamed joint or after their injection, as William Hunter first demonstrated two centuries ago. No vessels penetrate adult articular cartilage.

Lymphatics are seen by appropriate technique to be abundant in the synovial tissues but do not appear to enter the villi.

Nerves. Modern studies (3) have confirmed Hilton's law that all the nerves which supply the muscles bringing about major movements of a joint send branches into that joint. The nerve supply to a large joint such as the knee, may, therefore, be derived from many different spinal

segments. Sensory nerve endings are present in the capsular and subcutaneous connective tissues and in the ligaments and tendons. After penetrating the capsule, the bundles of nerve fibres mainly follow the course of the blood vessels. They lie close to the arteries and decrease in size as the vessels decrease. The nerves in the synovial membrane are probably concerned largely with vasomotor control. There is some dispute about whether the synovial membrane is itself sensitive. Articular cartilage is devoid of nerves and is certainly insensitive.

EMBRYOLOGY

The limb buds appear very early in the human embryo. By the sixth week the sites of the major joints are shown by surface furrows which mark off the principal segments of the limbs. A central core of condensed mesoderm forms the skeletal blastema and before long centres of chondrification appear in it, spaced in such a way that each marks the mid-point of the diaphysis of a future bone. The growing cartilage expands at a greater rate than the still undifferentiated mesenchyme. Both increase by multiplication of cells, but the cartilage expands also by the enlargement of individual cells and by the laying down of bulky intercellular substance, and it compresses the surrounding mesenchyme into a kind of capsule which laterally becomes the perichondrium. At the ends of each chondroid shaft, the mesenchymal cells arrange themselves in loops and arches and, in a short time, two opposing systems of arcs meet and interlace in the region destined to become a joint. Fell and Canti (1), who observed this process in tissue cultures of embryonic chick limb buds, conclude that the separation of the articular surfaces is a mechanical result of differential growth caused by the resistance of the undifferentiated tissue of the joint region and perichondrium to the rapid expansion of the cartilage masses from each centre.

Histological definition of the joints as interzones formed from the remains of the skeletal blastema between the cartilages is first apparent in the 11 mm. embryo of man. The sequence of events thereafter may be summarised from the recent review by Haines (4). Condensations of the extra-blastemal tissue near the joints next appear. These are destined to form the fibrous joint capsules and they cut off and enclose portions of the general mesenchyme which are henceforth termed synovial mesenchyme. At the very early foetal age of seven or eight weeks, the mesenchymal tissues of the embryonic joint are already separated from the other tissues by a rudimentary capsule. Within the capsule, at either pole, lie those parts of the cartilaginous blastema which will become the epiphyses; centrally, there are the remnants of the blastemal interzone, and, laterally, the synovial mesenchyme, its vascularity distinguishing it from the avascular blastema. Synovial membrane, sub-synovial tissues and all intra-articular ligaments, tendons and fibrocartilages arise from synovial mesenchyme. Articular cartilages are formed partly from the blastemal cartilage, partly from the intra-capsular perichondrium and partly from

the interzonal tissue, which quite early shows areas of central softening, leaving a few laminae of flattened cells, eventually to be chondrified, on each cartilage surface. The joint cavity results from the liquefaction of the interzone and of the central parts of synovial mesenchyme. It does not appear all at once. There are secondary extensions, as the synovial pouches develop, and also secondary infoldings of the synovial membrane. It is often stated that a sleeve of perichondrium persists as the joint capsule. Haines has made it quite clear that this is not so and that the capsule is derived from tissues lying outside the blastemal axis. Some perichondrium is included within the capsule, parts of which persist throughout life, first as perichondrium to the epiphyses, later as periosteum, while other parts form the circumference of the articular cartilage.

The final and familiar shape of each joint is partly predetermined by a mysterious faculty for self-differentiation inherent in the cartilage model of each bone. Murray and Huxley (6) grafted part of a chick limb bud upon the chorio-allantois of an older embryo and saw a femur, devoid of all other skeletal parts, grow into a recognisable femoral form with a well-developed head and a normal diaphysial curve. Fell and Robison (2) and others have confirmed the observation that the crude shaping of the articular ends of bones is an intrinsic property of embryonic tissue. The final perfect fit of two or more bones articulating one with another must depend on their mutual adaptation under the influence of pressure and movement.

The subsequent history of the diaphysis of a long bone is complicated. Ossification starts with a collar of periosteal bone around the middle of the shaft. As this spreads upwards and downwards, the cartilaginous shaft is vascularised, and the primary marrow cavity is formed by resorption from within. Cartilage is replaced by embryonic bone which in turn is resorbed as final lamellar bone is laid down. The mammalian epiphysis, however, remains cartilaginous until the secondary centre of ossification appears in it. It eventually unites with the shaft, leaving only the articular surface unossified.

What are the lessons a pathologist may learn from studying the embryology of the joints? All components of a joint are mesenchymal in origin. Some, however, cartilage and bone, are derived from the skeletal blastema and others, synovial tissues, menisci, etc., from the outlying mesenchyme included within the capsule at a very early stage of foetal growth. The perimeter of the articular surface is where these two mesenchymal structures unite and where later, bone, cartilage, synovial membrane, periosteum and perichondrium all meet. Hyperplasia of cartilage, bone or synovial tissues is common in this zone in diseased joints and it may be that the tissues here possess special proliferative powers by virtue of their mixed parentage. It is difficult to believe that the presence of a rich vasculature in this region is, by itself, a sufficient explanation of this capacity for reactive growth. In former times, much has been written about perichondrium covering the surface of articular cartilage. It is now generally believed to be a bare surface in all but the

peripheral parts and embryological studies support this view. The flattened cells from the interzonal tissue, which are left upon the cartilages when the central tissue liquefies, disappear in man before birth, though they may still be seen for the first few days of life in some other animals. The cells become chondrified and incorporated in the cartilage. The joint space appears as a liquefaction of the blastemal and parts of the synovial mesenchyme. Excavation proceeds by stages, and Haines has noted that wherever the joint cavities are spreading rapidly, the synovial surface is ragged, but when extension is arrested, even temporarily, a smooth layer of cells demarcates the cavity. There can be no doubt that the synovial lining is an adaptation of simple connective tissue cells and that it is not a special endothelium. An analogous process may be observed in the formation or extension of bursae and synovial ganglia in adult life.

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CHAPTER II

HISTOLOGY AND PHYSIOLOGY OF BONE, CARTILAGE, SYNOVIAL TISSUES AND JOINT FLUID

Bone

Bones have permanence of form and structure only after death. Familiarity with macerated anatomical specimens leads many students to disregard the essential vitality of living bone. In the healthy adult, it is true, cortical bone changes little in outward form, but cancellous bone, and to a certain extent compact bone too, is constantly being remodelled. There is a continual exchange of calcium and other salts between tissue fluid and bone trabeculae, and the bone cells alternate between periods of rest and periods of activity. The movement of minerals to and from bone is not simply a humoral affair, a washing in and out of bone salts, for these salts are built into bone and are removed by resorption of bone substance. The operation of bone-forming and bone-destroying cells is necessary in these processes.

Alizarin red, or madder root extract, which John Hunter used in 1794 to show new bone deposition in growing animals, has been used to study the recalcification of adult bone after parahormone decalcification (3). Thereby it has been shown that new trabecular bone matrix is laid down at the same time as calcium is restored to the bones, and that the most readily available source of calcium is in the cancellous bone ends. The use of radioactive tracer elements has confirmed the fact that the freest interchange of bone salts takes place in cancellous bone, but it has also shown that the salts in compact bone are from time to time renewed. Hevesey and his colleagues (17) maintained a constant plasma level of radioactive P in frogs and rabbits and found that within 50 days 29 per cent of epiphysial bone and 7 per cent of shaft bone had been renewed. Traces of the labelled element were shown to have been built into bone within a few minutes of the substance being introduced into the body. Bone is the only important reservoir of calcium in the body and there is normally a continual movement of calcium in and out of bone, especially in and out of cancellous or spongy bone. This movement is always accompanied by resorption of bone or regeneration. In its minute structure, therefore, bone is constantly changing. It is only in disease, however, when there is accelerated or abnormal destruction or new formation of bone that we are best able to observe these structural changes in process, in a histological section. The great majority of the bone cells in a section of normal adult bone are to be seen at rest.

Bone consists of matrix, salts and cells. The exact chemical composition of bone matrix has never been determined. It is an organic substance, allied to the interstitial ground substance of collagenous connective tissue and cartilage. Although appearing homogeneous after the mineral salts

have been removed by an acid decalcifier, it is permeated by a system of argyrophil fibrils in much the same way as collagen. The inorganic salts, which constitute 60 to 70 per cent of the dried weight of a bone, are a mixture of calcium phosphate and carbonate with some magnesium phosphate, united in complex minerals, *apatite* or *hydroxyapatite*. In preparing bone for histological section it is necessary to remove the calcium by a dilute acid;[1] the pliable soft bone matrix which is left retains its cellular structure. This material is not *osteoid* but decalcified

FIG. 1. OSTEOBLASTS

Bone growth in periosteum near healing fracture. Above is original cortex thickened by layer of partly calcified osteoid. Outlining this and the new osteoid islets are numerous and prominent osteoblasts. Below are the outer fibres of periosteum. *H. and E.* ×156

bone, whereas *osteoid* is new bone matrix which has not yet been calcified. The two can usually be distinguished under the microscope even in a decalcified section. Osteoid, when subsequently calcified, becomes true bone, but the formation of osteoid is not always seen in ossification, since very often under normal conditions bone matrix is calcified as soon

[1] The following decalcifier allows excellent staining after formalin fixation and is reasonably rapid:

Citric Acid	1 lb. (450 g.)
Water	1200 ml.

after solution add

87 per cent Formic Acid	1200 ml.
Water	1200 ml.

Wash blocks in running water overnight before proceeding with dehydration

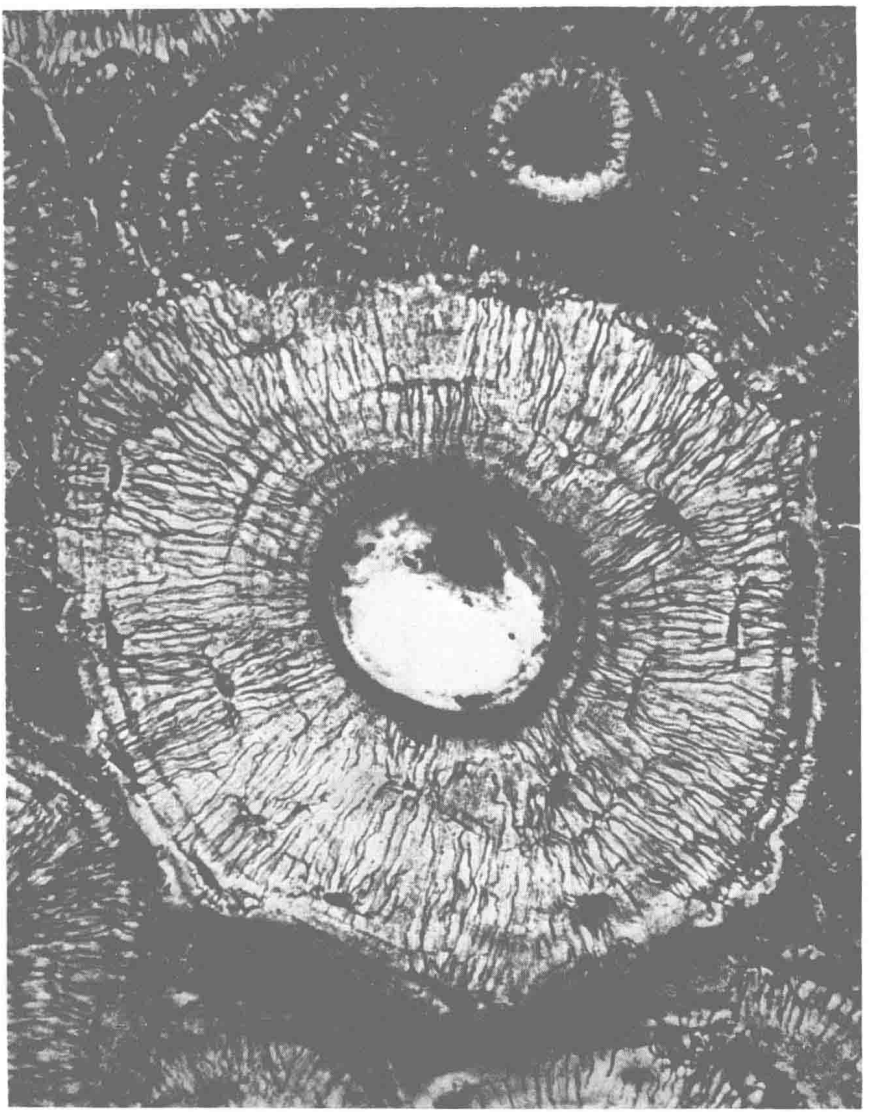

FIG. 2. NORMAL BONE

Transverse section of compact bone of femoral shaft. Haversian systems. Central canal containing blood vessel, concentrically disposed osteoctyes in lacunae, radial canaliculi. Lamellar rings are best seen in the upper system. *Celloidin section. Stained picro-thionin.* ×365