ORTHOPEDIC DISEASES

PHYSIOLOGY - PATHOLOGY - RADIOLOGY

Aegerter and Kirkpatrick

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BY

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Preface

THE "BASIC SCIENCE" aspect of bone disease has lagged behind that of other systems, not from lack of need or interest, but largely perhaps because of the very nature of bone, which until recently has defied investigation on the cellular and intracellular level. The rigors of salt removal by acid so distort the fine morphologic detail of cytoplasm and nucleus that conclusions have depended, until recently, mostly on interpretation of artefacts. Abnormal variations in mineralization were impossible to appreciate when the mineral had to be removed before the tissue could be examined. Knowledge of the healing of human bone was derived from that acquired by animal experimentation since even today we are denied serial sampling of the process as it occurs in our patients. Biopsies are usually relatively difficult and taxing to the donor so that the course of a disease process must be extrapolated from one or two known points in time.

Today there is feverish activity in the fields of bone morphology and function. New techniques and tools have become available. Histochemistry, immunologic chemistry and radioactive isotopes have allowed us to probe within the cell membrane. The ability to section undemineralized bone, microradiography and x-ray diffraction are telling us something of the tissue in its natural state. Phase, ultraviolet and electron microscopy reveal aspects of cell composition that heretofore were undreamed of. These new tools have set the anatomists, the chemists, the physicists and the physiologists to work hacking out new trails which will eventually converge in precise knowledge of the skeletal tissue cells, what they are and what they are doing in health and disease.

All this sound and fury has bewildered the busy physician who

must correlate the new material with one hand while he administers his knowledge with the other. It will take many books to sift the facts, to concentrate the verbiage, to reduce the product to the level of practicability. This book is meant as a starter. It looks at bone disease from the standpoint of its altered morphology and physiology, yet it tries to interpret these in terms of symptomatology and roentgenography. Diseases must be understood if they are to be successfully treated. This book is concerned with the understanding of the diseases which affect the musculoskeletal system. The diagnosis of orthopedic diseases necessitates an understanding of the pathology and physiology of bone; from these disciplines arise the clinical manifestations and the radiographic and laboratory findings.

Very little original material is recorded here. Almost all of it can be found in published papers if the searcher will spend the time and energy. The virtue of this volume, if such it has, is in the availability of this material. A consuming interest in the subject for nearly twenty years has given the authors the opportunity of selecting what they think the physician should know. There is no pretense at completeness, for such would remove it from the field of practical usage. It is intended for the clinician who wants to increase his diagnostic efficiency, for the radiologist who is perplexed by the meaning of an overwhelming array of radiographic nuances, for the pathologist who is distraught by his inability to interpret what he sees through his microscope, for the young specialist who wants to pass his board examinations and for the medical student who must acquire a certain amount of knowledge of orthopedic diseases in order to graduate.

The first four chapters are intended as a review of the complicated anatomy and physiology of the tissues of the skeleton. It is difficult to imagine how one can understand the disease process until one understands the normal. The fifth chapter is a simple histology of the tissues involved in orthopedic diseases. It is meant for the orthopedist and the radiologist who have forgotten rudimentary structural components of bone. It is not meant for the pathologist who reviews these basic principles in the first month of his residency. Chapter Six is a primer of bone radiology intended for the pathologist and the orthopedist who must know the fundamentals of radiology if they are to appreciate gross bone pathology. The remaining chapters are divided into three sections, each section grouping the skeletal diseases in a manner which should make them easier to find and to remember. Classification is stressed, perhaps overstressed, with the deliberate intention of presenting a concept of the whole before attempting the specific. There is no truer maxim in diagnostic medicine than "Before one can name the entity, one must know the possibilities."

When one attempts to acknowledge the help which is so important in writing any medical book, it is difficult to know where to start and where to end. First, perhaps, there were all our colleagues in other fields who unselfishly supplied cases and patiently instructed us in the fundamentals of their specialties. Principal of these is Dr. John Royal Moore, Chief of the Departments of Orthopedics at Temple Medical Center, Philadelphia Shriner's Hospital and St. Christopher's Hospital for Children. Most of the material in this book is derived from patients from his services. Many of the radiographs are from the Department of Radiology of Temple Medical Center and we are indebted to Dr. W. E. Chamberlain and his staff for permission to use them. Dr. Howard Steel read the entire manuscript and kept us thinking in terms of the patient.

Then there are our friends in our own specialties. Dr. James Arey of St. Christopher's Hospital read manuscript and offered many valuable suggestions. Dr. William Ehrich of Philadelphia General Hospital, Dr. E. B. D. Neuhauser of Children's Medical Center, Boston, and Dr. John Hope of the Children's Hospital of Philadelphia supplied unusual and hard-to-come-by cases. Grateful thanks are given our assistants in pathology and radiology, too many to list here, who did our work while we wrote.

Credit for the technique of the photomicrographs goes to Dr. E. S. Gault and for the rest of the illustrative material to Mr. William Taylor, both of Temple. Whatever faults there are in this field must be blamed on the authors who sacrificed perfection for punctuality.

An author gets the credit, favorable or adverse, for the words he puts down, but for every hour he writes, someone must spend at least an equal amount of time in transcribing it to a readable state. The authors happen to have the most talented, efficient and cheerful secretaries in the world. To them, Mrs. Hildegard Kates and Mrs. Dorothy McKee, this book rightfully should be dedicated. Our sincere thanks go to Jane Kirkpatrick, co-worker on the text, and to John A. Duross who skillfully arranged the index.

Finally, we owe a debt of gratitude to those wonderful institutions which housed us, nourished us, taught us and endured us while we worked, Temple Medical Center and its pediatric unit, St. Christopher's Hospital for Children. If any good comes of this labor let it reflect bountifully upon them.

ERNEST AEGERTER
JOHN A. KIRKPATRICK, JR.

Contents

Section I

A GENERAL CONSIDERATION OF CONNECTIVE TISSUES

Chapter 1

CELLULAR COMPONENTS OF CONNECTIVE TISSUE 3

Chapter 2

INTERCELLULAR COMPONENTS OF CONNECTIVE TISSUE 11

Chapter 3

NORMAL BONE PRODUCTION 23

Chapter 4

ESSENTIAL FACTORS IN NORMAL BONE PRODUCTION..... 31

Chapter 5

HISTOLOGY OF THE MUSCULOSKELETAL SYSTEM..... 46

Chapter 6

RADIOLOGY OF THE SKELETAL SYSTEM 78

Section II

DISTURBANCES IN SKELETAL DEVELOPMENT

Chapter 7

CONGENITAL DISTURBANCES 87

Achondroplasia 87
Morquio's Disease 96
Osteogenesis Imperfecta 101
Osteopetrosis 108
Miscellaneous Sclerosing Disturbances 116
Melorheostosis 116
Progressive Diaphyseal Dysplasia (Engelmann) 118
Hereditary Multiple Diaphyseal Sclerosis (Ribbing) 120
Osteopathia Striata (Voorhoeve) 120
Osteopoikilosis 121
Chondrodystrophia Fetalis Calcificans 123
Miscellaneous Developmental Deformities 123
Arachnodactyly (Marfan) 123
Acrocephalosyndactylism (Apert) 125
Cleidocranial Dysostosis 129

Chapter 8

DEVELOPMENTAL DISTURBANCES OF CHILDHOOD 133

Enchondromatosis 133
Hereditary Multiple Exostoses 139
Fibrous Dysplasia 145
Neurofibromatosis 155
Pseudarthrosis 161

Chapter 9

FUNCTIONAL DISTURBANCES OF THE RETICULOENDOTHELIAL SYSTEM 165

Hand-Schüller-Christian Disease 165
Eosinophilic Granuloma 172
Letterer-Siwe Disease 176
Gaucher's Disease 183
Niemann-Pick Disease 189
Lipochondrodystrophy 191

Chapter 10

DISTURBANCES IN VITAMIN METABOLISM 197

Rickets 197
Vitamin D Refractory Rickets 205
Hypervitaminosis D 209
Scurvy 213
Hypervitaminosis A 220

Section III

DISTURBANCES IN THE NORMALLY FORMED SKELETON

Chapter 11

THE REPAIR OF FRACTURES 227

Chapter 12

CIRCULATORY DISTURBANCES 240

Bone Infarct 240
Epiphyseal Ischemic Necrosis 246
Osteochondritis Dissecans 254
Aneurysmal Bone Cyst 259
Solitary Cyst of Bone 266
Hypertrophic Osteoarthropathy 273
Non-osteogenic Fibroma 277
Subperiosteal Cortical Defect 284

Chapter 13

METABOLIC DISEASES 290

Osteoporosis 290
Osteomalacia 297
Paget's Disease 304
Slipped Epiphysis 316
Bone Changes of Pituitary Dysfunction 322
Giantism 322
Acromegaly 326
Pituitary Dwarfism 328
Bone Changes of Thyroid Dysfunction 329
Hypothyroidism 329

Chapter 14

DISTURBANCES OF PARATHYROID FUNCTION 334

Hyperparathyroidism 334
Parathyroid Adenoma 334
Secondary Parathyroid Hyperplasia (Renal Rickets)..... 348
Renal Osteomalacia 353
Idiopathic (Primary) Parathyroid Hyperplasia 354
Hypoparathyroidism 357
Chronic Hypoparathyroidism 357

Chapter 15

INFECTIOUS DISEASES OF BONE 361

Acute and Chronic Suppurative Osteomyelitis 361
Brodie's Abscess 371
Tuberculosis of Bone 373
Syphilis of Bone 380
Miscellaneous Infections in Bone 385
Infantile Cortical Hyperostosis (Caffey) 390
Sclerosing Osteitis (Garré) 393
Villonodular Pigmented Synovitis 394

Section IV

TUMORS AND TUMOR-LIKE PROCESSES

Chapter 16

GENERAL CONSIDERATION OF TUMORS 401

Chapter 17

TUMOR-LIKE PROCESSES 418

Reactive Lesions in Bone 418
Osteoid Osteoma 418
Ossifying Fibroma 426
Non-osteogenic Fibroma 428
Subperiosteal Cortical Defect 429
Hamartomas 431
Osteogenic 431
Osteoma 431
Osteochondroma 433
Chondrogenic 435

Enchondroma 435
Collagenic 438
Angioma of Bone 438
Aneurysmal Bone Cyst 442

Chapter 18

OSTEOGENIC TUMORS 446

Benign Osteoblastoma 446
Osteosarcoma 449
Parosteal Sarcoma 469

Chapter 19

CHONDROGENIC TUMORS 477

Benign Chondroblastoma 477
Chondromyxoid Fibroma 482
Chondroma 488
Chondrosarcoma 494

Chapter 20

COLLAGENIC TUMORS 505

Giant Cell Tumor of Bone 505
Fibrosarcoma of Bone 519

Chapter 21

MYELOGENIC TUMORS 527

Plasma Cell Myeloma 527
Ewing's Tumor 541
Reticulum Cell Sarcoma 550

Chapter 22

TUMORS OF SOFT PARTS 559

Lesions of Mesothelial Origin 561
Synovioma 561
Benign Synovioma 563
Lesions of Lipoblastic Origin 567
Lipoma 567
Liposarcoma 569
The Muscle Tumor Group 570
Leiomyoma 570

Leiomyosarcoma 571
Rhabdomyosarcoma 571
Granular Cell Myoblastoma 573
Alveolar Cell Soft Part Tumor 575
Lesions of Connective Tissue Origin 576
The Fibromatoses 577
Desmoid 578
Fibrosarcoma 580
Lesions of Nerve Supportive Tissue Origin 582
Neuroma 582
Neurilemmoma 583
Malignant Schwannoma 583
Neurofibroma 584
Neurofibrosarcoma 584
Lesions of Supportive Structures of Vessels 585
Hemangioma 585
Hemangioendothelioma 586
Hemangiopericytoma 586
Hemangiosarcoma 587
Mesenchymoma 588

INDEX 589

SECTION

I

A General Consideration of Connective Tissues

CHAPTER

I

Cellular Components of Connective Tissue

Factors in Cell Differentiation

The various components of the connective tissues, i.e., fat, fibrous tissue, cartilage and bone, arise as differentiated elements from the primitive mesoderm. Differentiation of a cell from those of the totipotent cell mass to become the highly specialized unit of tissue structure is the result of a number of influencing factors some of which are as yet poorly understood. It is presently believed that the mechanism by which a mature cell reproduces by mitotic division resides in the chromatin constituents of the nucleus. This substance, largely desoxyribose nucleic acid, is supplied by the cytoplasm and, therefore, conditioned by enzymatic processes which take place in the cytoplasm of the cell. As long as the cytoplasmic metabolic processes are stable it may be assumed that the nuclear hereditary constituents will remain constant and reproduction may continue indefinitely, each cell a faithful duplicate, in both morphology and function, of its predecessor. We may call the combined agents which govern this exact duplication the primary heredity factor.

Obviously this type of exact reproduction can take place only among cells which are completely differentiated, i.e., cells whose enzymatic constituents are mature and stable, and in an environment which is absolutely unchanging. Thus the chondroblast may give rise to identical chondroblasts, osteoblasts to identical osteoblasts, and so on, as long as the medium in which they exist remains the same. If we introduce new elements into the cell environment, we may force the cytoplasmic metabolism to change and conceivably produce a

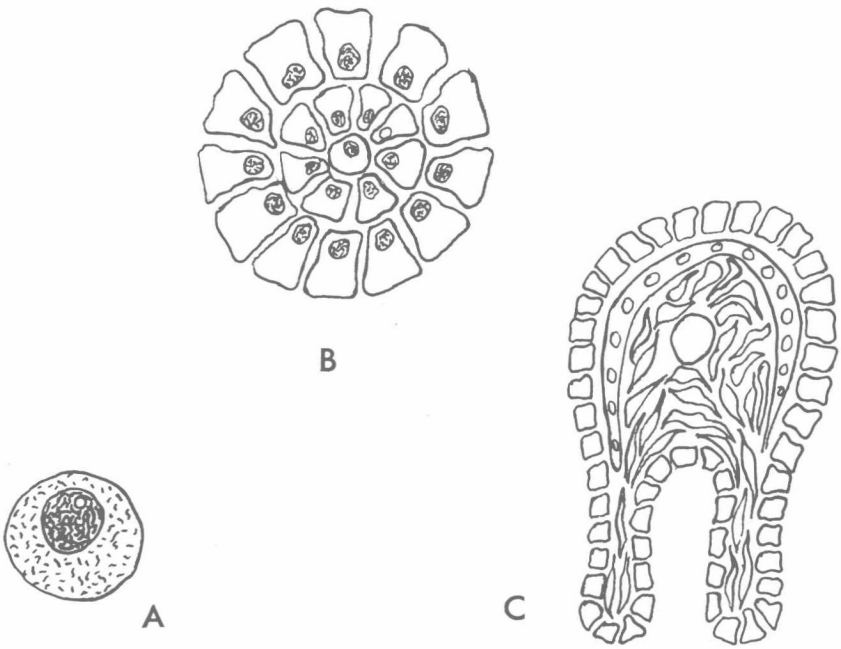

FIG. 1. Schematic drawing to illustrate cell differentiation. *A,* The unicellular organism ingests what it needs from its environment, processes it to form the plasmagenes in the cytoplasm and passes on the refined product, mostly desoxyribonucleic acid, to the nucleus. It discharges its wastes into its environment. *B,* In the multicellular organism the surface cells still have access to environmental resources but the central cells must accommodate their metabolism to the materials that can penetrate between the surface cells. These materials are contaminated by the catabolic products of the surrounding cells. *C,* The more complex the organism becomes, the greater the possibilities for differentiation. This drawing illustrates the effects of the secondary heredity factor upon the deep cells.

change in the molecular constituency or arrangement of the nuclear heredity apparatus. We may designate the environmental influence on a cell its secondary or somatic heredity factor.

It is probable that this latter factor is a potent agent in what we know as cell differentiation. It is obvious that in the development of a multicellular organism the environment of cells must change until a stage of stable maturity is reached. When the embryonic form consists of a single cell or a small group of cells each with at least some free surface, the cell may choose from its environment what it needs and discharge its metabolic refuse as it likes. As the organism grows and the early cells become surrounded by other cells (Fig. 1) the resources of the deep cells become more and more limited, since the environmental substances must penetrate between surrounding cells to reach them and in doing so must be conditioned by the catabolic products of the surrounding cells. So the environment of the deep cell is changed and thus its metabolic pattern must change if the cell

is to survive. Changes in the intracytoplasmic chemistry may result in changes in the nuclear hereditary apparatus and thus, by means of the somatic hereditary factor, differentiation and specialization are achieved to the point of stable maturity.

When the state of maturation is complete we may expect a cell to faithfully reproduce its kind in the normal, physiologic repair of necrobiosis. However, if its environmental resources are altered by changes in blood supply or changes in the selective dispersion of its surrounding, intercellular supportive components, one may find alterations in its heredity mechanism which result in offspring which differ from the mother cell. These differences may be forecalculated when the environmental changes are known. When the heredity alteration is reversible we call this change in morphology and function "metaplasia," a physiologic or near-physiologic process. Disturbances in cell health and growth patterns sufficient to induce tissue changes constitute the lesions of disease. It is the understanding of these lesions, a recognition of their morphologic and functional characteristics, that is the field of interest of the pathologist.

The Development of Cells

The primitive mesoderm is a pleomorphic tissue substance which arises in the very early stages of embryologic development. As one examines it through the microscope one sees a mound of stellate and reticulated cells in a gel matrix (Fig. 2). As the celomic cavity develops, the cells on the cavity surface, and thus the cells which come to line the cavity, swell and line up in a single layer. Their rounded free surfaces characterize their cell type which we may designate as primitive mesothelium. It is of interest that wherever mesodermal tissue or one of its derivatives borders on a natural space or cavity, such as the lumina of blood vessels, the pericardial, pleural or peritoneal cavities or the joint or tendon spaces, they tend to take this shape. These are known generally in their mature, differentiated state as endothelial or simply mesothelial cells. In the vessels they retain their name of endothelial cells. Lining the major body cavities they are called serosal cells and when they face joint and tendon spaces they are designated synovial cells (Fig. 57, page 76).

The reticulated portion of the mesoderm forms a syncytium. Deposits of this syncytium are best seen in the mature organism in the spleen, the lymph nodes and scattered lymph follicles, and in the bone marrow. Apparently the differences of environment condition its morphology and function, for though there are similarities, the functions particularly of marrow, spleen and lymph node reticulum appear to