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TRAUMA

MADE IN U.S.A.

Press of W. B. Saunders Company

Library of Congress Catalog Card Number: 59–5961

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PREFACE

THE PREPARATION of a monograph on Trauma is not an easy task, for the surgery of trauma embraces the entire science of surgery. Consequently, much potentially pertinent material must be omitted, and for this there is no apology, only a hope that the material included will prove to be of value to those for whom it was intended. With one exception the authors are or have been members of the Attending Staff of the Columbia-Presbyterian Medical Center in New York City, and of the Faculty of the College of Physicians and Surgeons of Columbia University. These busy surgeons have pre-empted time from their full schedules to write the following pages, not so much for the edification of other surgeons as for the innumerable medical students, residents, and practitioners who will not receive a complete training in surgery but who, nevertheless, will be called upon to cope with the great majority of all civilian accidents.

The first section of the book is devoted to a consideration of the local, regional, and systemic responses to injury, an enunciation of established principles in the treatment of trauma, and discussions of the problems of thermal and vascular trauma and of infection. The later chapters are limited mainly to a rationalization of treatment predicated upon a reconciliation of these fundamentals with the characteristic features, effects, and requisites of specific injuries.

Consideration of traumas of the specific regions is prefaced by a brief outline of the special features of surgical anatomy which may influence the choice and technique of treatment or predispose to complications in the management of common injuries. Uncommon injuries are also discussed in sufficient detail to forewarn the unwary of imminent pitfalls in their management and to suggest measures for their avoidance.

Trauma cuts across all branches of medicine with a superb disdain for the man-made boundaries of the different specialties. For this reason a small amount of duplication has been intentionally retained so that the reader may obtain information about certain injuries from multiple sources having different viewpoints and approaches. Fractures in and about the base of the skull, for example, are discussed in various chapters from the viewpoints of the neurosurgeon, the otolaryngologist, the ophthalmologist, and the maxillofacial surgeon.

The discussion of fracture treatment is inescapably dogmatic in appropriate situations. This should be interpreted not as reflecting the only, or even the best, solution of the problem at hand, but merely as presenting one form of treatment which has been employed widely and which, even when used with variable technical competence, has proved as efficient as and safer than others. An effort has been made to avoid the orthodox textbook approach to the treatment of fractures, which by categorical classification so oversimplifies this problem that the inexperienced practitioner soon is led to believe that a fracture is merely a broken bone, and that a roentgenogram and a book of instructions are the sole requisites for successful treatment.

Modern medical literature dealing with trauma, especially in the field of fracture treatment, is often so empirical as to mislead, and at times so controversial as to confuse any but an experienced surgeon. For this reason, and in keeping with the fundamental level which the authors have attempted to maintain, bibliographic citations have, for the most part, been omitted.

My sincere gratitude and appreciation are tendered to all the authors for their splendid contributions; to my good friend, Doctor Preston A. Wade, for his valuable suggestions and criticisms; to Miss Clara Barry, who typed the manuscript and prepared the text, and without whom this work could not have been completed; and to Mr. Alfred Feinberg, who has taken my amateur cartoons and made them into legible illustrations. The unflagging cooperation, assistance, and, above all, the patience of the publishers are acknowledged with thanks.

HARRISON L. MCLAUGHLIN, M.D.

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† Deceased.

CONTENTS

Part I

GENERAL CONSIDERATIONS

CHAPTER 1

THE RESPONSE TO INJURY

By HARRISON L. McLAUGHLIN, M.D.

REPAIR

AN INJURY damages and kills tissue cells. The resulting gap in the tissue is filled by blood which extravasates along all communicating tissue planes, often for a considerable distance. Repair commences almost at once and is marked by an orderly train of events, many of which may occur almost simultaneously, and each of which is somewhat dependent upon the others for consummation. The morphological aspects of these phenomena are demonstrable, but their biochemical interrelationships, especially in the case of bone repair, are not yet entirely clear.

Injury, Inflammation, and Repair

These three are inseparable, except in the case of avascular tissues, such as the cornea and hyaline cartilage, where injury is followed by neither inflammation nor repair unless the damaged tissues become vascularized. In all other tissues injury not only mobilizes the defense reaction of inflammation for the evacuation of cellular casualties, but also initiates a process of cellular proliferation for their replacement. In the early stages following injury the signs of inflammation prevail, but repair is proceeding apace. As time passes the evidences of healing become predominant, but a subsiding residuum of inflammation remains until repair is complete. The surgeon must recognize that inflammation and repair are coincident and not successive processes.

The Protein Cesspool

Suspended throughout the hematoma that occupies the tissue gap caused by the injury are red blood cells and dead and damaged tissue cells. The ensuing clot bridges the gap by a meshwork of fibrin strands in the interstices of which are these doomed cellular products of tissue damage. This refuse does not remain indefinitely, but must be catabolized, evacuated, and replaced by living cells. Its autolysis commences at once, and pro-

gressive decomposition produces a liquefying accumulation of the end-products of protein disintegration. The systemic effects of intestinal obstruction are illustrative of the toxic properties of some of these protein catabolites, but it has become increasingly evident that others serve a useful purpose in the healing process, possibly as building blocks for the reparative matrix which follows. It is logical, therefore, that the treatment of trauma should, among other things, be designed to minimize the total tissue damage and facilitate evacuation of its noxious products.

Concomitants of Cell Death

The permeability of the local capillaries is increased and, augmented by the markedly increased protein concentration of the extracellular fluids in the damaged area, results in an escape of fluid through their walls to produce edema.

Many tissue cells of the region become distorted by changes in both cytoplasm and nuclei. Edema and cell distention progressively obliterate the elasticity of all the regional structures so that they soon become internal splints which, augmented by rapid proliferation of fibrous tissue, effectively maintain displacement, or assist in the maintenance of reduction of the structures distorted by the injury. Early reduction of displaced tendon or bone fragments is, therefore, facilitated by elasticity of the regional structures, and their subsequent rigidity is a mechanical aid to whatever form of fixation is employed. By the same token delayed reduction becomes progressively more difficult as time passes, and its accomplishment entails considerable additional damage to the "jelled" regional musculature.

Margination of polymorphonuclear leukocytes along the walls of the capillaries in steadily increasing numbers is soon followed by their migration through the walls of the vessels into the area from which debris must be removed. These are the scavengers of the undigested protein refuse which cannot be evacuated by diffusion. Many of the leukocytes are destroyed by the toxic properties of the materials they ingest, and their decomposition releases additional enzymes which aid in the digestion of the tissue debris.

The pH of the tissue fluids drops sharply as the injured area becomes saturated in an acid bath by the total products of cell death mixed with serum, lymph, and extracellular fluid.

Local vasodilatation, due to liberated histamine and other products elaborated by dying cells, rapidly supersedes the primary but transient reflex vasospasm caused by the injury. Despite this constant reaction, it seems improbable that there is an increased flow of blood through the injured area. Undoubtedly there is an increased quantity of blood per tissue volume unit, but the equally constant regional vascular effects of an injury (page 9) militate for a state of engorgement and passive congestion rather than to increased blood flow at the injured area.

Local decalcification is peculiar to this state of vasodilatation and passive congestion when bone is involved by the injury or is adjacent to

the area involved by the ensuing inflammatory process. The surgeon, as he watches the pathologist decalcify bone specimens by acidification, is tempted to speculate that the acidity of the fluids bathing a fractured bone may contribute to this phenomenon. The calcium apatite emigrates from the bone fragments and diffuses throughout the liquefying ooze of the surrounding hematoma. Formerly it was thought to remain until it was redeposited in the bone-forming process, but recent tracer studies indicate that it enters the circulation and is lost in the urine. New calcium from the circulation is deposited and remains suspended within the scaffolding of the fibrin clot until some unknown biochemical reaction either changes it into a salt that can be utilized for new bone formation or elaborates a calcifiable matrix for its reception. There are some indications that both phenomena may occur.

Fibroplasia

Coincident with progressive evacuation of the products of cell death the fibrin clot is invaded from all sides by an army of young cells derived from all viable connective tissues, probably excepting cartilage, at the site of injury. These undifferentiated cells all are capable of eventuating as fibroblasts. Simultaneously, from surrounding blood vessels, sprout solid outgrowths of similar cells, which eventually become canalized and flattened to form a network of new vascular channels generally adhering to the pattern of the original fibrin clot. Gradually the fibrin scaffolding is replaced by collagen fibrils which form a matrix for accommodation of the invading cellular elements.

To recapitulate, certain products of cell death useful to the repair process remain at the site of injury. As the remaining useless and toxic products are removed, their place is filled by a mass of ingrowing young vessels and connective tissue cells, which adapts itself to the original fibrin scaffolding, and this in turn is dissolved and gradually replaced by a collagenous matrix. This is repair by granulation tissue as it occurs in wounds of all vascular tissues. In the absence of what, for want of a better term, might be described as a specific stimulus, the end-result is a mass of fibrous tissue. In the healing of a fracture it is clear that some influence produces a metaplasia into cartilage and bone as well as into fibrous tissue.

LOCAL EVENTS IN FRACTURE REPAIR

A working knowledge of the orderly sequences of bone repair is necessary to good fracture treatment, but the surgeon must realize that the factors pertinent to a fracture problem are not limited to the injured area. Secondary regional and systemic concomitants and products of the injury must be anticipated and reconciled with local factors before the best plan of treatment can be determined. Above all, the surgeon's concept of a fracture, the problems it presents, and his ultimate goal of treatment must remain in clear clinical focus.

Clinical Definition of a Fracture

A fracture is more than a broken bone, and its outmoded definition as "a solution in the continuity of a bone," which has been accepted by most past and by some contemporary surgeons, is incompatible with modern surgical philosophy. It was this false concept that engendered the equally fallacious dictum that the treatment of a fracture consisted of "reduction and immobilization until the bone healed."

The results of trauma are never limited to bone. A fracture involves primarily all the soft tissues in the injured area as well as the bone; secondarily, all the undamaged structures of the injured region; and, finally, the physical, economic, and emotional state of the injured person. Identical fractures cannot produce identical problems if for no other reason than that they occur in different persons. Consequently fractures should not be categorized as other medical and surgical lesions are, nor can fracture treatment be learned by rote. On the contrary, it must remain adaptable in detail and method to many factors, including the age, size, sex, occupation, and various other characteristics of the patient; the vulnerability of the uninjured regional structures to the penalties of disuse; a priority rating of the functional demands to be satisfied by the injured part after healing occurs; and, finally, the specific features of the local injury. To be of clinical significance the definition of a fracture must be stated as *a localized area of soft tissue and bone damage attended by secondary harmful effects upon adjacent regional structures and upon the patient as a whole.*

Theories of Bone Repair

The process of new bone formation remains an enigma. Confusion is increased by three popular theories purporting to explain the mystery. The advocates of each contrive to rationalize one concept at the expense of the others, but clinical common sense suggests a modicum of truth and error in all three.

The periosteal theory, which envisions periosteum and endosteum as the sole specific source of bone-forming cells, is impugned by the frequent formation of histologically normal bone in tissues devoid of periosteum. It is obvious to the surgeon that periosteum has both beneficial and detrimental effects upon the repair of fractures—beneficial in its shaping and confining influence upon reparative callus, but harmful when it is interposed between bone fragments or when it has been avulsed from its soft-part connections and insulates the bone from extra-osseous circulation. It is clear that cells derived from living periosteum participate in fracture healing, but only in concert with other cells derived from all adjacent mesodermal sources.

The osteoblast theory is also vulnerable to criticism. Its proponents have postulated that extra-skeletal new bone formation results from the activity of "wandering" or "resting" osteoblasts which, before becoming recognizable as such, cannot be differentiated from young connective tissue cells. On the other hand, microscopy reveals forming bone trabeculae to be

surrounded by a peripheral layer of cells which are very similar to the osteoblasts of the embryo.

The theory of pluripotentiality of the mesenchyme is consonant with embryonal development, clinical and microscopical observations, and the present state of biochemical knowledge. This theory envisions that, prior to a change in type or potential, an adult cell must be destroyed and replaced, or revert to its primitive state; that metaplasia is peculiar only to young undifferentiated cells; that osteogenesis depends upon reparative tissue matrix and some unknown biochemical influence, rather than upon the intrinsic ability of a specific cell to secrete bone; and that coincident with new bone formation undifferentiated mesenchymal cells undergo metaplasia to eventuate as osteocytes, even as in the embryo.

These and other theories may be allowed to tickle the intellectual curiosity of the fracture surgeon, but they will prove to be suitable mortar for his therapeutic bricks only so long as they remain in complete harmony with clinical events.

The Facts of Bone Repair

Bone heals by the same process as other vascular tissues, except for the superimposed enigma of osteogenesis. As stated earlier, it is clear only that some unknown factor or factors influence the process of repair by granulations to cause a metaplasia into cartilage and bone, as well as into fibrous tissue.

Chondroplasia. In the healing fracture nests of young cartilage cells are to be observed throughout the reparative mass of granulation tissue, and especially in the gap between the bone ends. Possibly the elaboration of these cells, most pronounced at the fracture gap, reflects a response of nature to motion, even as cartilage is elaborated at the bone ends in the embryo. Clinically there is no doubt that excessive mobility of certain fractured bones over too long a period may be an important factor, but not necessarily the sole factor, in a failure of bony union and the production of a primitive joint with cartilage-covered bone ends enclosed within a fibrous capsule.

Normally chondroplasia is a transient phenomenon. Cartilage cells, like their fibroblastic counterparts, disappear and eventually are replaced by bone. It seems unlikely that a direct transformation of chondrocyte into osteocyte takes place; it seems more likely that all cartilage is resorbed or reverts to its primitive mesenchymal form prior to or coincident with replacement by bone.

Calcium and calcification. The composition of the calcium salt in bone is unknown, and the dynamics of its removal and deposition remain a mystery. The inflammatory phase of bone repair is marked primarily by a movement of calcium ions from the local bone into the cavity to be obliterated by the healing process. Possibly at this stage either the calcium is in a form unsuitable for redeposition, or the matrix of the granulation tissue is as yet unprepared for its reception, or both. At any rate, there is evidence that the calcium enters the circulation and most of it is lost in the urine.