Sixth Edition

A TEXTBOOK OF SURGERY

By

John Homans, M. D.

Clinical Professor of Surgery, Emeritus

Compiled from Lectures and Other Writings
of Members of
The Surgical Department
of
The Harvard Medical School

With a Special Bibliographical Index
and
With Illustrations by Willard C. Shepard
and others

1945

CHARLES C THOMAS · PUBLISHER

301-327 East Lawrence Avenue · Springfield, Illinois

Published by CHARLES C THOMAS
at BANNERSTONE HOUSE
301-327 EAST LAWRENCE AVENUE, SPRINGFIELD, ILLINOIS

Published simultaneously in Canada by
THE RYERSON PRESS, TORONTO

First Edition, February, 1931
Second Edition, November, 1932
Third Edition, February, 1935
Fourth Edition, October, 1936
Fifth Edition, February, 1940
Sixth Edition, July, 1945

Printed in the United States of America

A TEXTBOOK OF SURGERY

PREFACE TO THE SIXTH EDITION

THIS is a war edition—not in the sense that wars are responsible for new principles which must be incorporated into a surgical textbook, but rather that they introduce problems and confirm or disprove the worth of many procedures. During the present world conflict, for example, the value of chemo-therapy for most of the known infections has been affirmed and the relative effectiveness of individual drugs against particular bacteria is being worked out; the advantage of the complete immobilization of wounded limbs has been supported; the lifesaving effect of very prompt treatment of the seriously wounded individual by skilled surgeons, near to or on the field of battle, has been proved, and transfusion of whole blood into the exsanguinated and shocked individual has been found superior to the use of even the best of the blood substitutes or fractions. Yet none of this is really new, and much remains to be amplified and re-examined.

Rather to my own surprise, it has seemed better to continue the system of deletions, corrections and substitutions, used in previous revisions, than to rewrite considerable portions of the book. Thus the pagination remains almost unaltered and to the casual reader this revision may seem less rigorous than is actually the case. However, one or two chapters have been rearranged within the exact space originally occupied; one or two old illustrations have been done away with and several new ones by Miss Piotti have been introduced.

In this work, Dr. Robert S. Myers, Instructor in Surgery at the Harvard Medical School and Associate in Surgery at the Peter Bent Brigham Hospital, has been of great assistance. From the point of view of the youthful teacher, he has given warning of much outworn material, has permeated the book, as far as possible, with modern drug therapy, and has offered such suggestions as should make it acceptable to the student and practitioner of today. Where space has not been available for some details of clinical conceptions and exact methods of treatment, reference is made to authors of easily available monographs dealing with such subjects.

The names of the original contributors no longer appear upon the title page. They are gratefully recorded here—Arthur W. Allen, David Cheever, Edward D. Churchill, Harvey Cushing, William P. Graves, Robert B. Greenough, Gilbert Horrax, Daniel Fiske Jones, William E. Ladd, George A. Leland, Howard A. Lothrop, Richard H. Miller, Edward H. Nichols, Robert B. Osgood, Charles Allen Porter, Tracy J. Putnam, Lyman G. Richards, Edward P. Richardson, Channing C. Simmons, J. Herbert Waite, Wyman Whittemore, and Philip D. Wilson. Of this group, Harvey Cushing, William P. Graves, Robert B. Greenough, Daniel Fiske Jones, George A. Leland, Howard A. Lothrop, Edward H. Nichols, Charles Allen Porter and Edward P. Richardson are no longer living.

I am greatly indebted for much intelligent and pungent criticism to Dr. Daniel C. Elkin, who, as a keen and active professor of surgery, has tried to prevent me from misleading his students.

Dr. Ralph T. Knight, Associate Professor of Surgery and Anesthesia at the University of Minnesota Medical School, who kindly permitted me to use his *Outline of Anesthesia*, which first appeared, somewhat modified, in the Second Edition (1932) has made available a more recent form of this very useful guide.

To anyone interested in the origin of the book, the preface to the first edition, which will be found in all five editions previous to the present but which is now discarded, is available. Mr. Thomas has continued his helpful and generous attitude and the Banta Press has cooperated most intelligently in accurately fitting in fresh material.

In the Bibliographical Index, some old names have been dropped and many new ones added but, so far as possible, those of interest from an historical standpoint have been retained.

John Homans

Boston, June, 1945

A TEXTBOOK OF SURGERY

CONTENTS

CHAPTER I

REPAIR

HISTORICAL.—REPAIR OF WOUNDS: REPAIR OF ASEPTIC CLOSED WOUNDS; *The Exudate, Inflammation, Granulation Tissue, The Scar.*—GENERAL CONSIDERATIONS AFFECTING REPAIR.—VARIATIONS UPON THE NORMAL PROCESS OF REPAIR.—THE REPAIR OF VARIOUS TISSUES: BONE; CARTILAGE; INTESTINE; MUSCLE; TENDON; BLOOD VESSELS; NERVE.—THE INFLUENCE OF AGE ON REPAIR.

Physic and surgery, today the great divisions of the healing art, were once joined together in the parent stem of medicine. But already before medieval times, it was hardly possible for any individual to master the complexities of both branches and at the beginning of the Dark Ages the physician and the surgeon were called into being—the first of the specialists. The splitting apart was good for neither branch. The physician practised internal medicine. His attitude toward disease was intellectual, aloof, and he would not see what was before his eyes, preferring the sterile, speculative Galenic philosophy to the profitable Galenic experimental method. The surgeon was the craftsman who treated, in rather brutal ways, the surface of the body, sitting down under Avicenna's degrading dictum that his art was inferior to that of the internist. Such at least was the custom, though there were physicians observant and dextrous, and surgeons who did not relish exchanging the knife and ligature for the red hot rod and boiling oil.

But time has changed all this, and though specialties have continued to multiply, medicine and surgery have in the last few centuries been growing together again. The surgeon is no longer the barber and craftsman; he must be an internist as good as any, and in view of the hazardous nature of the procedure he has come to employ, he must have a better knowledge of the structure and function of the body than most. Surgery, in short, is now distinct from physic only because of the peculiar therapeutic measures it uses, for as a rule it treats injury and disease by manual operation or corrective apparatus. And success in the exercise of such measures depends before all else upon an understanding of the processes by which the tissues, when injured or diseased, are repaired.

The power of repair is an inherent and persistent quality of living matter. This power, always exerted toward the restoration of structure, is best exhibited in those tissues, such as fascia, tendon, cartilage and bone, which offer themselves as a means of transmitting physical force. It is least effective in those which transform chemical energy, as, for instance, the muscles, brain, secreting glands, the kidneys and liver. Expressed most simply, the less elaborate the function of a tissue, the greater its capacity for repair. The skin presents a special case. From its basal cells, which constantly renew its epithelial layers, regeneration is effectual and rapid. With this exception, repair is now believed to proceed from undifferentiated, mesenchymal cells. From these, the fibroblasts of connective tissue develop, and out of this material, purely local influences may evolve specific structures such as tendon, cartilage and bone. Lacking such influences, the result is a scar.

REPAIR OF WOUNDS

The most simple and characteristic repair is that of a wound upon the body's surface. Some tissue is always destroyed; the least, by division with a sharp knife; the most, by a burn or crushing violence. The very injury sets in motion the reparative process. There is first called forth an exudate, whose amount is proportional to the extent of the damage. This joins divided surfaces, fills gaps and becomes the scaffold for repair, being replaced by new tissue. Replacement, that is, the active period of repair, depends for its perfection upon certain local factors, notably, a minimum of destruction, a good blood supply, freedom from foreign material, especially bacteria, and, less directly, upon many general considerations (Pages 5–7) among which are preeminent, rest, an adequate blood supply, a normal fluid and electrolyte balance, both dehydration and overhydration being harmful, sufficient blood proteins and a reasonable supply of vitamins (especially C). Ideal repair occurs in bacteria-free, that is, aseptic, accurately closed wounds and is called healing by *First Intention*, or in the vernacular, "on the first try."

Repair of Aseptic, Closed Wounds: Healing by First Intention.—*The Exudate*, which fills the interstices of a wound, when hemorrhage has ceased and divided surfaces have been united, is derived from blood, lymph and tissue fluid. It includes red corpuscles, fibrin, evolved as a coarse meshwork by contact of blood with injured tissue, and leucocytes which migrate to the

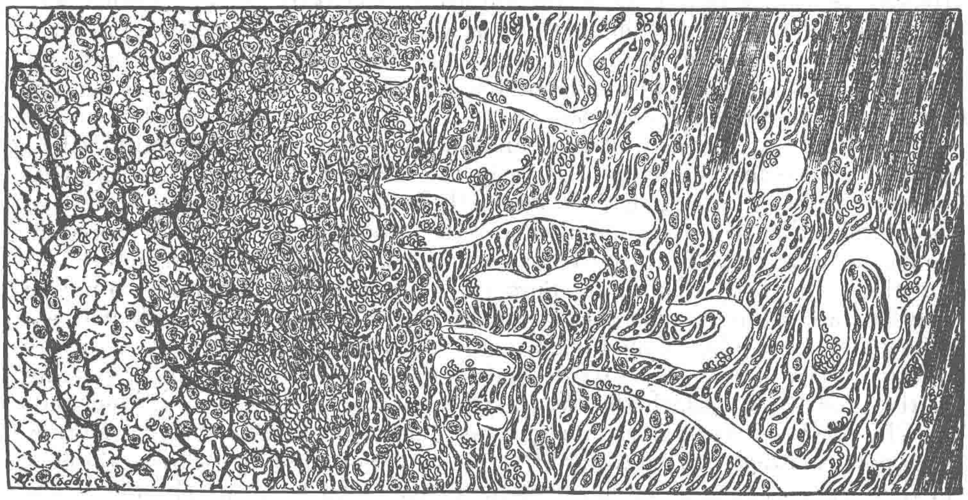

Fig. 1. A Granulating Wound.—A diagrammatic sketch of one surface of an incision in the rabbit's tongue, on the 5th day of healing. (E. H. Nichols Collection.) Nearest the muscle on the right is loose young granulation tissue showing large capillary loops. In the central part of the wound, on the left, the exudate is only beginning to be organized and the fibrin meshwork filled with red blood corpuscles and leucocytes can still be seen.

scene from adjacent vessels. These elements spread into contiguous parts as far as the last injured cell or fiber, uniting opposing surfaces and filling gaps by forming an adhesive coagulum, while on the surface they dry to form a scab. In association with this normal and purposeful reaction, near-by blood vessels are dilated.

Granulation Tissue: The Lag and Active Periods.—A lag of about four days, lengthened by necrosis of tissue, lack of blood supply, the presence of foreign bodies, infection and want of rest, is occupied, first, by autolysis and phagocytosis of dead tissue, the work mainly of mononuclear phagocytes (monocytes) and, second, by ameboid movement of fibroblasts, that is, wandering cells of uncertain origin, derived perhaps from the reticulo-endothelial system. The beginning of the end of the lag period is marked by the sudden growth of these primitive mesenchymal cells, which, supported by capillary loops, fan out centrifugally into the wound spaces and elongate along fibrin strands. The formation of collagen fibers by the fibroblasts ends the lag period and begins to give the wound strength. The process has an orderly embryonic quality and continues with decreasing velocity until the 12th to 14th day. The strength of the wound follows a regular curve (Howes and Harvey) being one-third completed on the 6th day and two-thirds on the 8th. Experimental evidence harmonizes with clinical observation and with Carrel's earlier studies of wound healing. Proliferation of the surface epithelium, by contrast, may under favorable circumstances, be immediate.

The Scar.—As its growth slackens, granulation tissue undergoes a gradual shrinkage. Its cells become spindle-shaped; its newly formed intercellular collagen fibers contract and squeeze the capillary loops out of existence. At first red, it gradually pales. Two weeks from the time the wound was made may see the end of this gross change, yet the scar continues to contract for months, for a year perhaps. The new layer of epithelium covering its surface, however narrow the wound, always differs from the surrounding skin. It has little tendency to form papillae and contains no hair or glandular tissue. Red at first, like the granulations, it fades in the course of many months to white.

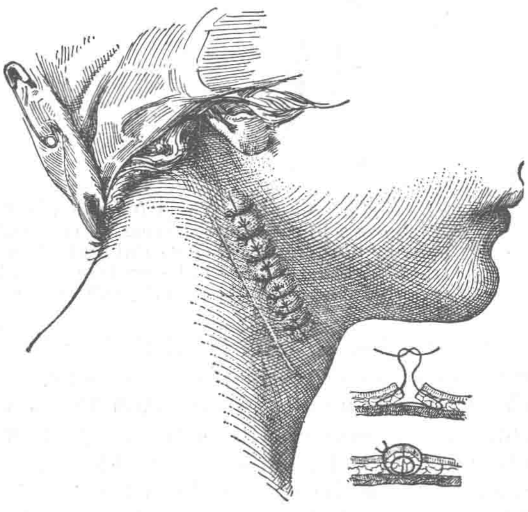

Fig. 2. Building up the edges of an incision to allow for shrinkage of a scar in depth. The cut has been made in the direction of the elastic fibers and the natural folds. (See Figure 4.) Since the wound is greatly supported by buried stitches, the external row can be tied loosely and removed early, lessening the injury done to the skin by the suture material.

The contraction which any scar must finally undergo is roughly proportional to its extent, and acts in three directions—length, breadth and depth. Incised wounds cleanly made and gently closed, result in scars of such infinite narrowness that though visible always, their shrinkage causes no noticeable deformity. Such perfection is seldom attained, and contraction must always be reckoned with in the management of injuries involving the skin. If a wound has any degree of depth, the contraction which takes place in this direction will be so considerable as to draw the scar below the level of the adjacent surfaces, but such a depression may be avoided, in suturing a wound, by building up its edges into a low ridge. As healing takes place and the granulation tissue contracts, the ridge is gradually brought down to a normal level. Shrinkage of a wound in length almost inevitably occurs, and if the amount of granulation tissue has been considerable, this factor will be more noticeable than any other. A scar may actually be crippling on this account alone. If, for instance, an incision is made following the edge of the pectoralis major muscle along the anterior border of the axilla, the resulting scar is likely to restrict elevation of the arm; whereas a cut across the axilla, even though it should undergo considerable shortening, is innocuous.

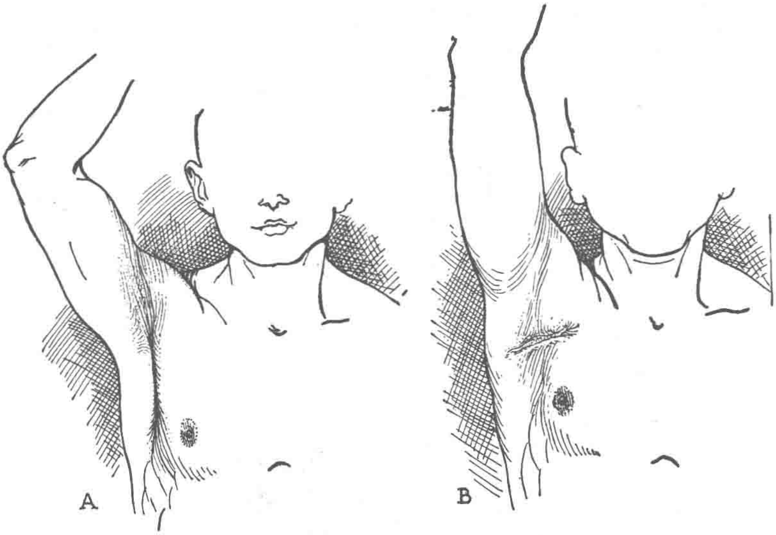

Fig. 3. A and B. The deforming effect of a wound improperly placed. A, shows a linear scar resulting from a cut made parallel to the anterior axillary border. It prevents full abduction of the arm. B, shows, by contrast, a rather contracted scar properly placed perpendicular to the anterior axillary fold. It permits full abduction of the arm.

Contraction of scars in width is influenced by yet other factors. Since the skin possesses elastic fibers and is forever in motion, the lateral pull of the elastic skin is usually greater than the power of the inelastic fibrous scar to shrink. In various parts of the body the prevailing general direction of the elastic fibers is known and the width of a scar in these regions will depend upon whether the cut has been made across their path or parallel with it. This is particularly true of the neck and axilla. In other regions, muscular contractions as well influence the appearance of the scar. In the face, for instance, where the skin is constantly thrown into folds by the play of the emotions, a scar will be the less noticeable the more exactly it follows an habitual fold, and the more unsightly, in so far as it crosses the direction of a muscular pull. Such considerations may, however, be overruled as, for instance, by the presence of branches of the facial nerve in the field of the incision: it may be essential to make the cut parallel to its fibers to avoid any risk of injury to them.

Though the large majority of scars contract normally, a few, for no clear or consistent reason, undergo hypertrophy and build themselves up above the surface. Undoubtedly constriction and new growth are simultaneously taking place, for these scars, which are known as *keloids* and which may take on the appearance of true tumors, have a very poor blood supply and tend to undergo necrosis. This process will be more fully described in Chapter X.

It might appear from the foregoing account that dense, contracted scars should have unusual strength and stability. Such is not actually the case. There are occasions, of course, when a great sheet or a heavy band of cicatricial tissue is permanently deforming and can not be stretched. A deep burn or an infected wound often results in a situation of this sort: the lower lip or an eyelid becomes everted, or the chin is drawn down upon the breast, and it is mechanically impossible to relax the scar to any appreciable degree. But the unyielding quality of such a scar lies partly in the impossibility of applying a correcting force, for when such a force can be applied, the deformity may often be relieved. For instance, heavy cicatrices closing the axilla and binding down the arms can not be stretched by voluntary effort, but the contraction due to an extensive wound which crosses the palmar surface of the elbow may often, by muscular exertion plus gravity, be made to loosen its grip. In a general way, indeed, scars do tend to soften and stretch with time, and when subjected to constant tension of considerable strength, as in the case of wounds of the abdominal wall, they give way in a remarkable manner. Consequently when an abdominal incision has healed, not by first intention but after suppuration, leaving a broad cicatrix, it may later become so thinned by intra-abdominal pressure as to allow a breach, or hernia, to be formed.

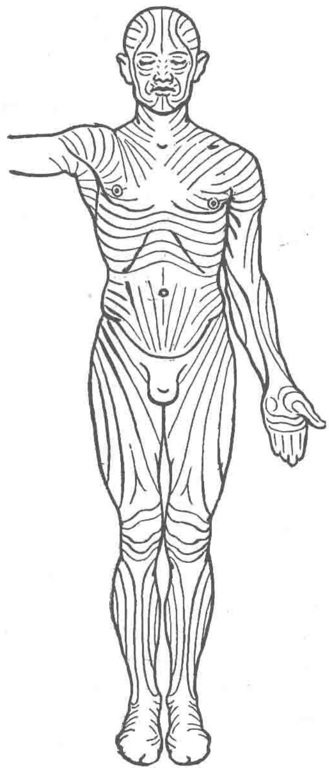

Fig. 4. The direction of the lines of elasticity in the skin. By following the directions of these lines, wounds will result in the least noticeable and deforming scars. Other anatomical considerations may in some regions override this rule which is of greater value in the neck, axilla and about the knee than elsewhere.

General Considerations Affecting Repair.—The presence of bacteria in a wound, particularly of the "pyogenic" sorts, delays and materially alters repair. The wound is then septic. Toxins formed by the bacteria cause tissue necrosis and a responsive exudate, a process which is practically unlimited so long as living micro-organisms are present. The exudate is liquid, abundant and loaded with polynuclear leucocytes. The formation of granulation tissue occurs as usual but fails to secure healing of the wound until the destructive action of the invading bacteria is brought to an end. The polynuclear leucocytes, which give a white color to the exudate, are spoken of as *pus cells.* The exudate is a purulent one, that is, pus, and the inflammation which results from bacterial injury is distinguished as *suppurative inflammation.* As compared with aseptic healing, the repair of a wound subject to suppurative inflammation is delayed, and since destruction of tissue is greater, the scar must necessarily be more extensive. Such a process is described below as healing by second intention.

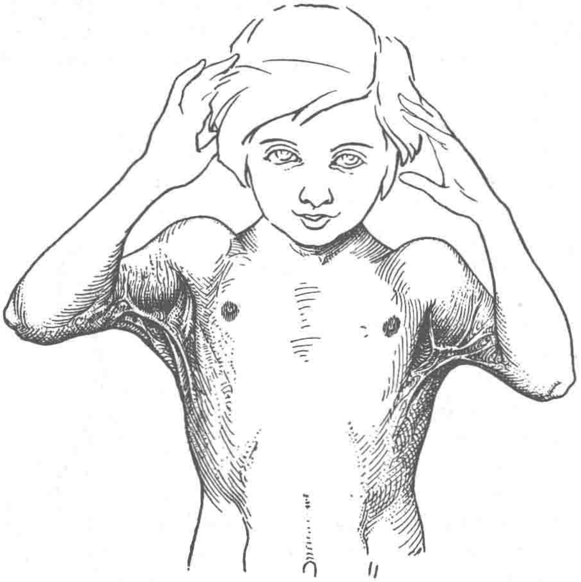

Fig. 5. Cicatrices in both axillae following a deep burn. Gravity tends to keep the elbows at the sides and the muscles abducting the arms are at a great mechanical disadvantage. Therefore such scars can not be stretched.

Foreign material, whether inert or irritating, absorbable or indestructible, interferes with repair. The presence of any foreign body adds to the stimulation of injury and leads to excessive exudation and granulation, an attempt on the part of the tissues to isolate a substance of which they are intolerant. In most instances, unless the object is too large or too rough, it becomes buried in fibrous tissue. Healing will then have been somewhat delayed and the extent of the scar increased. Small metallic fragments, lead shot or even bullets may remain enclosed and innocuous for an indefinite period. Large or rough bodies are seldom tolerated to this degree, and since they are likely to have carried bacteria into the tissues, are usually a source of actual suppuration. Indeed a foreign body of any sort or size is more harmful in this respect than in any other, and the suppurative inflammation caused by the bacteria it harbors is incurable as long as the focus of infection—the foreign body itself—is present. Even ligature material in infected operative incisions is not infrequently a cause of obstinate infection of this sort, and must be removed before healing can occur. In the absence of bacteria, however, unabsorbable material, such as silk, may be enclosed in wounds with little consequent reaction. Absorbable material must be dissolved or destroyed before healing

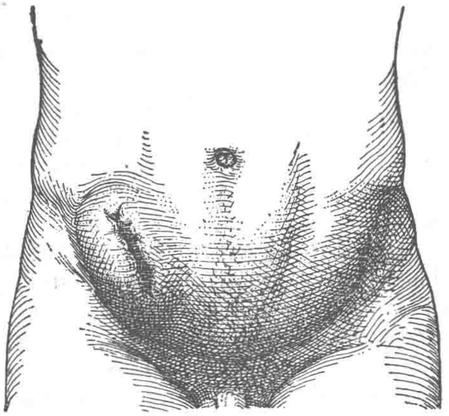

Fig. 6. The stretching of a broad operative scar in the abdominal wall has allowed the muscular and fascial edges of the wound to separate widely, and the abdominal viscera bulge out beneath the skin—a post-operative ventral hernia.

is completed. Naturally, injury to the tissue by too tight ties and stitches delays absorption or enclosure of suture material and retards healing.

A third important influence upon repair is rest. Hemorrhage and exudation are the more limited and the amount of replacement by new tissue is correspondingly lessened, the more perfectly the wound is approximated by stitches and immobilized by dressings, bandages and, as occasion demands, splints. This principle applies to tissues such as the intestine and requires that means must be taken to stay, for the time, the motile function of the injured part.

A fourth and really more vital factor is the preservation of a normal blood supply. Anemia may result from too tight dressings or too great tension upon the approximated tissues. Passive congestion increases exudation and causes failure to remove waste products. Thus a part, naturally dependent, such as a foot, must often be elevated by confinement of the patient to bed and a hand may have to be supported by a sling.

Fluid and Protein Balance.—Both dehydration, by loss of fluid and electrolytes, and edema impede repair, but protein deficiency, less readily noticed than gross fluid changes, is more disturbing to healing. For prolonged starvation or loss of protein by severe or repeated hemorrhage, that is, hypoproteinemia, draws fluid into the intercellular spaces. The resulting edema produces soggy wound edges and delays fibroplasia. The hematocrit records blood concentration, and the falling drop method, the specific quality of the blood.

Vitamins A, D and C are believed to contribute to the healing of wounds. Lack of vitamin C, which controls the formation of the essential intercellular substance, collagen, by the fibroblast, may cause weak union in ill-nourished infants and children, perhaps adults as well. The scurvy need not be outspoken. Lanman and Ingalls suggest that when the ascorbic acid of the blood plasma falls below .45 mg. per 100 cc., a want of vitamin C is evident and an asymptomatic scurvy, sufficient to cause a wound to lack collagen, is present. They propose the administration of ascorbic acid in five to ten 200 mg. doses. However, Lund and Crandon find that, in an adult, a low plasma level of ascorbic acid alone is no proof of a dangerous want of Vitamin C and that only a prolonged deficiency will result in failure of healing. They believe that in such a deficiency a lack of other vitamins, notably the B complex (thiamin, nicotinic acid) is decidedly a factor. Vitamins A and D in the form of cod liver oil favor healing when applied to open wounds.

Healing by Second Intention.—If much tissue is destroyed, whether by violence or infection, repair, though following the same plan as in ideal healing, must be delayed. Suppose a large area is lost from a flat surface. An exudate will be poured out and granulation tissue will form. Now, however, it is exposed and will present the red granular appearance from which its name is derived. Skin grows in from its edges and may in time fully cover the surface, but if the wound is too extensive, the covering will long be incomplete. In that case, the granulation tissue often rises above the level of the surrounding skin, forming "exuberant granulations" or "proud flesh." From the exposed surface there continue to exude serum and blood corpuscles, which, unless absorbed by dressings, form a scab or crust. Finally if the new epithelium reaches the limit of its growth without covering the gap, and as contraction of the deeper, older parts of the wound restricts the vascular exchange with the parts above them, the healing process stops. Passive congestion at the surface takes place, swelling of the superficial granulations occurs, and there is left a pale edematous, granulating area, known as an *ulcer*.

There is then a limit, not exactly to be defined, to the area of superficial loss of tissue which can be healed without artificial aid. The stimulus calling for the new growth of skin will not continue to act if the area to be covered is too large. If, however, in the midst of an area too wide to be overgrown by new skin from the edges of the wound, some small epithelial islands are preserved, the inhibition, if that expression may be used, is removed, for not only will epidermization proceed from the islands but these little areas seem to exert a stimulating influence upon the distant marginal epithelium.

Thus the mere size of a wound alters very considerably the course of repair. The process may be further complicated, even under aseptic conditions, by the amount of dead tissue left by the injury. Such necrotic material must be cast off or absorbed before completion of the granulating process can occur. If the necrotic tissue is in the form of one large, continous layer, the granulations formed by the healthy tissue beneath it will finally cast it off. If the dead material lies in small, scattered, buried areas, it may be dissolved and carried away by phagocytic cells. According to the time required for its disposal, healing is retarded. *In general, the greater the delay in healing and the deeper and more extensive the granulations, the more dense and contracted will be the resulting scar.*

Another cause of delay in normal repair results from the presence of large amounts of blood in a wound. In tissue having good vascular connections, a part of the clot is quite rapidly absorbed. The fluid portion soon disappears in this way, but the solid portion must slowly be disintegrated by the action of phagocytes and is eventually replaced to a greater or less extent by scar. When an unyielding cavity must be filled, this process may be a useful one, as, for instance, in destructive disease of bone. Even in extensive injuries of the soft parts, the organization of a clot may offer the only available means for filling a gap. In general, however, the process is slow, deforming and therefore undesirable.

Still another variation upon normal repair, seldom seen in the uncomplicated healing of wounds, is the union of two granulating surfaces, sometimes spoken of as healing by "third intention." This is likely to result from the unnatural separation of two surfaces, either because the wound has been kept open for some useful purpose or because of suppurative inflammation. The two already granulating surfaces are capable of uniting, the growing cells from the two sides blending and becoming organized into one scar. Union of such surfaces may be made by suture. Many of the great infected shell wounds of the late war (1914–1918) were closed in this way, and the procedure has become known as "secondary suture."

THE REPAIR OF VARIOUS TISSUES

Bone

Anatomy and Physiology.—A long bone consists of a hollow shaft, or diaphysis, filled with marrow, and two ends, the epiphyses, comprising spongy or cancellous tissue and the cartilaginous articulating surfaces. At each end of the diaphysis is its growing cartilaginous portion and only after growth ceases are the epiphysis and shaft firmly united. There is considerable cancellous bone in the ends of the diaphysis, its architecture adding strength in the region where the shaft flares and its wall becomes thin.