Synopsis of SURGERY

RICHARD D. LIECHTY
ROBERT T. SOPER

FOURTH EDITION

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To

Valerie and Hélène

Preface

For each of our first three editions we have quoted the following editorial:

> In all probability, the present type of medical textbook will eventually disappear, to be succeeded by two quite different types. There will be the textbook carefully designed for a particular audience (the student, whether still in school or 15 years out of school). . . . Quite distinct will be the reference books which discuss specific topics in substantial detail. . . . Whoever tries to mix these two types is, at the present complex level of medicine, performing only a disservice. (JAMA **197:**133, July 25, 1966)

With each edition, the writer's thoughts have become more prescient. New information has forced the larger textbooks to expand threefold in less than two decades. The senior editor of one of the large surgery textbooks confessed to us: "I no longer know what to teach medical students. How much can they absorb in the allowed ten weeks?" He laments, as do the students, that neither the human brain nor the time allotted to surgery has kept pace with the information explosion.

The student will find new material in our *Synopsis of Surgery:* radioimmunoassays, computerized axial tomography, and 1,25$(OH)_2$ vitamin D, for example. Nevertheless, we have tried to keep the material digestible to ease the way for the student, whether a junior ward clerk or a seasoned physician who needs only a brief update.

We welcome our new authors and hope that our efforts will help the student make the transformation from information to knowledge to wisdom—the goal of all education.

Richard D. Liechty, M.D.
Robert T. Soper, M.D.

Synopsis of

SURGERY

Contents

CHAPTER 1

Origin of surgical disease

RICHARD D. LIECHTY
ROBERT T. SOPER

Each day of the year our medical school libraries add the equivalent of three new *volumes* of medical literature to their already extensive collections. As in other scientific fields, the medical profession, and especially the medical student, face an "information crisis." The volume and scope of medical literature dramatically emphasize the diversity of specialization. However, common bonds do exist across the specialty fields. Obstruction is still obstruction whether in the lacrimal duct, ureter, or spinal canal. We would like to begin by emphasizing some of these common concepts that link one specialty to another.

All somatic diseases, regardless of the specialty fields treating them, have their origins in the following six basic pathological processes:

1. Congenital defects
2. Inflammations
3. Neoplasms
4. Trauma
5. Metabolic defects and degeneration
6. Collagen defects

Four phenomena that result from these fundamental pathological processes are responsible for almost all surgical diseases and for many nonsurgical diseases as well. These phenomena are (1) obstruction, (2) perforation, (3) erosion, and (4) tumors (or masses).

OBSTRUCTION

Cerebrovascular disease (strokes) and coronary heart disease (coronaries) are two of the leading causes of death in the United States. Both result from obstruction of vital arteries carrying blood to the brain or to the heart muscle. Glaucoma, one of the two leading causes of blindness in our country, also results from obstruction, in this case obstruction to the outflow of fluid from the anterior chamber of the eye.

Free flow of blood, urine, cerebrospinal fluid, lymph, and other fluids, as well as air, is essential for health. Note in Table 1-1 the wide variety of diseases that result from obstruction.

PERFORATION

Perforation, similarly, is the direct cause of many surgical diseases. Perforation is often such an intensely dramatic event that few medical students will forget the "boardlike" abdomen of the patient with a ruptured peptic ulcer or the shock that overwhelms the patient with a ruptured aortic aneurysm. Examples are given in Table 1-2.

EROSION

Erosion is a "partial perforation," a slower process of ulceration (i.e., a break in the continuity of a tissue surface). Examples of erosion are given in Table 1-3.

TUMORS

The most subtle of these four phenomena is a tumor or a mass. This explains in large measure why cancer is so often detected only after it induces one of the previous three processes; e.g., we occasionally see tumors of the breast that have grown to astonishingly large size. Because no vital flow is obstructed and perforation or erosion of the skin occurs very late, symptoms and consequently diagnosis are delayed, often tragically.

These four phenomena, *obstruction, perforation, erosion,* and *tumors,* are the underlying direct causes of most surgical diseases. Like the theme of a symphonic work, they recur in many

Table 1-1. Diseases resulting from obstruction

System	Disease	Nature of obstruction
C.N.S.	Hydrocephalus	Congenital obstruction of C.S.F.
E.N.T.	Middle ear infection	Eustachian tube obstruction
Eye	Glaucoma	Obstruction of aqueous humor
Lung	Atelectasis	Mucous plug in bronchus
Biliary tract	Cholecystitis	Cystic duct stone
G.I.	Appendicitis	Fecalith, appendix
G.U.	Prostatism	Prostatic hypertrophy
Extremity	Intermittent claudication	Arteriosclerosis

Table 1-2. Examples of perforation

System	Disease	Nature of perforation
C.N.S.	Cerebral hemorrhage	Rupture of central nervous system artery
E.N.T.	Perforation of tympanic membrane	Infection with pressure
Lung	Spontaneous pneumothorax	Rupture of bleb
Biliary tract	Rupture of gallbladder	Obstruction, distension, necrosis
G.I.	Duodenal ulcer	Perforation of ulcer
G.U.	Ruptured bladder	Obstruction and distension
Vascular	Aortic aneurysm	Rupture of aneurysm

Table 1-3. Examples of erosion

System	Disease	Nature of erosion
C.N.S.	Meningitis	Erosion of abscess wall; mastoiditis
E.N.T.	Pharyngeal carcinoma	Bleeding; erosion into blood vessels
Lung	Tuberculosis	Bleeding; granulomatous erosion into blood vessels
G.I.	Duodenal ulcer	Bleeding; ulcer erosion into blood vessels
G.U.	Bladder stone	Bleeding; erosion of bladder wall
Extremity	Raynaud's phenomenon	Digital ulceration; ischemic erosion of skin

different forms. Sometimes they appear unmistakably loud and clear; at other times soft, muted, and elusive. The able physician will learn to recognize them. The core and the concern of this text are to aid the student in his recognition and understanding of these processes.

CHAPTER 2

Wounds, wound healing, and drains

DAVID W. FURNAS
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Although the healing of wounds is a vital part of surgery, it also plays an important role in other medical fields. For example, the fibrous healing of myocardial infarcts often leads to life-threatening arrhythmias or ventricular aneurysms, and fibrous vegetations threaten embolization from rheumatic valvular disease. In posthepatitic patients scar tissue infiltrates the liver and in some cases fatally encases the regenerating liver cells or produces portal venous hypertension. In these examples fibrous tissue healing in its exuberant, sometimes misdirected, growth may eventually prove fatal. Wound healing, the surgeon's constant concern, is of more than casual interest to other physicians as well.

Healing by regeneration in man is limited to simple tissues, such as epithelium, and one compound organ, the liver. All other organs (skin, bowel, heart, brain) heal by merely sealing or patching of the wound. Paraplegia, for example, results from transection of the upper spinal cord. Scar tissue joins the severed cord ends but blocks all nerve impulses; the distal neurons, separated from their cell nuclei, degenerate and die. Unfortunately man has, in his evolutionary past, virtually lost the ability to regenerate compound tissues. There remains, however, this remarkable process of sealing or patching that man depends on to survive his hostile environment.

Tissues heal by three main processes: *epithelialization, fibrous tissue synthesis,* and the powerful force of *contraction.* Many surgical decisions depend on a clear knowledge of these extraordinary phenomena. When to remove sutures, where to make incisions, when to release a postoperative patient for normal activities, when to splint a wound, when to primarily close a wound, and when to leave it open are practical applications that the student should keep in mind as he studies the fundamental aspects of wound healing.

We first discuss the healing of *incised wounds, avulsed wounds,* and *contaminated wounds.* Pathological wound healing, wound complications, placement of incisions, suture materials, wound drainage, and drainage tubes complete this chapter.

INCISED WOUNDS AND SUPERFICIAL WOUNDS

A simple *clean incised wound* heals by *primary intention* after accurate surgical closure *(primary closure).* Within the first few hours of injury the cut edges of the wound are coapted by a fibrinous coagulum, which serves as a scaffold for granulation tissue to form. During the first day, leukocytes, mast cells, and macrophages enter the area to dispose of local debris and bacteria. The *epithelial cells* of the neighboring epidermis dedifferentiate, flatten out, multiply, migrate into and across the wound, and redifferentiate. Within 24 hours the epidermal surface is intact in an incised and sutured wound. This same sequence of fibrin deposition, granulation tissue, and epithelialization serves to replace and heal the surface of broader wounds, such as second-degree burns or light abrasions, within a few days or weeks.

During the first few days that an incision is healing, the *inflammatory phase,* almost no tensile strength is gained. Meanwhile, *capillary buds* begin to sprout from the wound edges and differentiate into functioning networks, and *fibroblasts* migrate into the wound area, probably from nearby loose connective tissue. These fibroblasts form *collagen,* the material that knits the wounded dermis and deeper structures and gives strength to the wound. First the fibroblasts secrete *tropocollagen,* which aggregates into large *procollagen* fibers. These herald the *collagen phase,* the earliest evidence of tensile strength. Procollagen, through polymerization and cross linkages, becomes collagen, and from the fifth through the fifteenth days there is a rapid gain in tensile strength.

The young collagen fibers mature, link with one another, and orient along lines of stress. The wound reaches almost its full strength within 6 weeks. Although a slight gain continues over a number of months, the scar seldom, if ever, becomes stronger than the surrounding skin and fascia.

The *rate of healing* is accelerated by a rich blood supply and perhaps by warmth of the wounded part. Thus the face heals rapidly and sutures may be removed in a few days. In contrast, sutures must be left for 10 to 14 days in wounds of the lower leg because of its poorer blood supply.

As *wound maturity* progresses, the fibroblasts and capillaries greatly diminish in number, and the resultant scar is composed chiefly of collagen connective tissue, capped with epithelium. This progress is observed clinically as an initial red, raised, hard *immature scar* that molds into a flat, soft, and pale *mature scar* over a period of 3 to 12 months or more, as collagen molecules and cross links rearrange.

An excised wound or defect closes more slowly but in identical fashion, except that contraction of the wound edges plays the principal role. The edges of the defect advance into the defect probably from the action of contractile myofibroblasts. These recently described cells resemble *smooth muscle cells* and can be inhibited in experimental animals by smooth muscle antagonists. Wound contraction is a consistent, powerful force that all experienced surgeons respect (Figs. 2-3 and 2-4).

"EXCISED" OR AVULSIVE WOUNDS

If a wound cannot be primarily closed, it must heal by *secondary intention* by means of the mechanisms of contraction and epithelialization. Examples are wounds that are excessively contaminated, wounds in which treatment has been delayed, or burns and wounds that involve necrosis of large skin surfaces. In a few days the raw, exposed area becomes filled with *granulation tissue* ("proud flesh") (Fig. 2-1) composed of sprouting capillaries and fibroblasts. The wound edges creep toward each other by *contraction* and *epithelial migration.* If a mantle of necrotic skin clings to the surface of the defect, it is called an *eschar.* Formed of coagulated collagen and debris, it is much thicker and tougher than the scab of a superficial wound (Fig. 2-2). Tightly attached at first, it eventually separates from the underlying granulation tissue and falls away.

Healing is speeded by removing dead tissue, debris, and secretions by surgical excision (débridement) and by applying intermittent dressings moistened with antibacterial solutions to the wound. The capillarity of the dressings drains away bacterial exudate. Dead tissue adherent to

Fig. 2-1. Granulation tissue. Red, moist bed of fibroblasts and capillaries covering surface of leg that sustained full thickness burns 6 weeks before.

the dressings is removed when the dressings are changed. There soon emerges a clean granulating surface that resists reinfection. Wound closure can be hastened by *secondary closure* (if the wound edges can be apposed) undertaken a few days after injury. Sutures are used to appose the wound edges, usually after first excising granulation tissue. Larger wounds are closed with split skin grafts. If the defect is *too* large, an unstable scar may result.

In many instances healing by *secondary intention* is convenient and desirable (Fig. 2-3). However, when the wound is located on the face or over a joint (where mobility of the part favors excessive displacement), contracture is likely and it results in diminished motion and sometimes

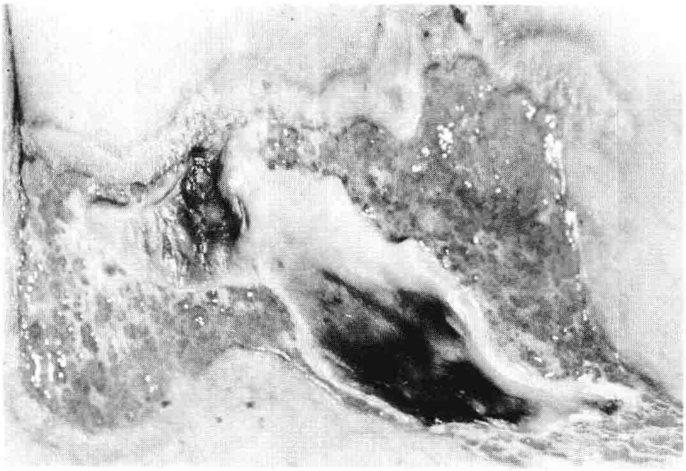

Fig. 2-2. Eschar. Deep burn coagulated full thickness of skin several weeks before. Eschar is in process of separating from underlying bed of granulation tissue. Copious exudate from local bacterial activity speeds process.

Fig. 2-3. Useful wound contraction. **A,** Wide removal of carcinoma of skin with electrocoagulation leaving open 3 × 4 cm. defect. **B,** Defect several months later after spontaneous closure by contraction and epithelialization. (Courtesy Department of Dermatology, University of Iowa Hospitals.)

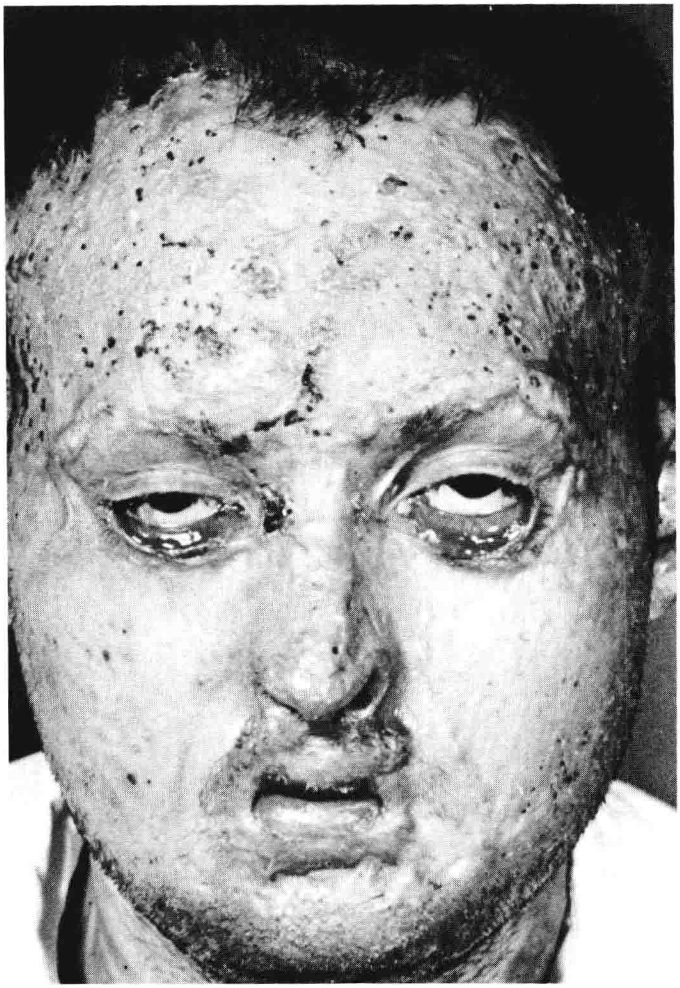

Fig. 2-4. Inimical wound contraction. Ectropion of eyelids and stenosis of mouth resulted from spontaneous closure of burn wounds of face by contraction and epithelialization.

grotesque deformity (Fig. 2-4). This is prevented by early closure of the wound with skin grafts or pedicles before contraction occurs. In addition, splints and physical therapy may help prevent skin grafts from contracting.

CONTAMINATED WOUNDS

Wounds received outside of the operating room are contaminated wounds. They may be *grossly clean* or *dirty, neat,* or *ragged* and contused (*"tidy"* or *"untidy"* in British parlance).

A "golden period" of approximately *6 hours* was cited several decades ago as the optimum time to close a contaminated wound, after which the wound should be left open to prevent infection. This concept should not be entirely ignored, but more important is the answer to the question: Can this *contaminated wound* be converted into a surgically *clean wound,* or is this a *contaminated wound* in which bacterial activity is already so advanced that it *cannot* be converted?

We now have antibiotics and more refined surgical techniques so that, *with an excellent blood supply, we can take liberties with the golden period.* A 2-day-old wound of the foot that shows no sign of infection can be closed with appropriate preparation (not simply "putting in stitches") with small risk of infection. A grossly clean, neat wrist laceration, 12 hours old, can be repaired safely. However, a 3-hour-old wound of the