Fundamentals of SURGERY

SIXTH EDITION

Richard D. Liechty

Robert T. Soper

IE INTERNATIONAL EDITION

FUNDAMENTALS OF SURGERY

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Preface

The first edition of this book was published in 1968, a tumultuous year. It marked the assassinations of Dr. Martin Luther King, Jr. and Robert Kennedy, the capture of the Pueblo by North Korea, the rioting at the Democratic Convention in Chicago, the announcement that President Johnson would not seek reelection, and the election of Richard Nixon as President. During the subsequent 20 years the world changed dramatically.

To the clinician, medical science has also changed dramatically. New scanning devices—computed tomography and magnetic resonance imaging—have revolutionized diagnosis. In some institutions, cardiac and liver transplantation have become routine procedures, and intensive care units have provided vital new dimensions to patient care.

Through all this evolution we have maintained our philosophy of writing for those students who are just beginning their clinical studies. Our coauthors have readily accepted the concept of ". . .a textbook carefully designed for a particular audience, the student, whether still in school or 15 years out of school. . ." (JAMA 197:133, 1966).

Although we have resisted the constant pressures to include more data, to make this a book for all readers, *Fundamentals of Surgery* has inevitably grown. We hope, nevertheless, that this sixth edition will continue to give students a broad grasp of surgical principles yet allow them some spare moments to think about other things—to gain perspective. "For perspective," said Will Durant, "is the secret of philosophy." And it well may be the secret to understanding this world of rapid change.

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

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I

Surgical Principles

1

Origin of Surgical Disease

Richard D. Liechty
Robert T. Soper

Obstruction
Perforation
Erosion
Tumors

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, faces 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 the common concepts that link one specialty to another.

All somatic diseases, regardless of what specialties treat them, have their origins in the following six basic pathologic processes: congenital defects, inflammations, neoplasms, trauma, metabolic defects and degeneration, and 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, respectively. Glaucoma, one of the two leading causes of blindness in the United States 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

Table 1-1. Diseases Resulting from Obstruction

System	*Disease*	*Nature of Obstruction*
CNS	Hydrocephalus	Congenital obstruction of cerebrospinal fluid
ENT	Middle ear infection	Eustachian tube obstruction
Eye	Glaucoma	Obstruction of aqueous humor
Lung	Atelectasis	Mucus plug in bronchus
Biliary tract	Cholecystitis	Cystic duct stone
GI	Appendicitis	Fecalith, appendix
GU	Prostatism	Prostatic hypertrophy
Extremity	Intermittent claudication	Arteriosclerosis

Table 1-2. Examples of Perforation

System	*Disease*	*Nature of Perforation*
CNS	Cerebral hemorrhage	Rupture of CNS artery
ENT	Perforation of tympanic membrane	Infection with pressure
Lung	Spontaneous pneumothorax	Rupture of bleb
Biliary tract	Rupture of gallbladder	Obstruction, distension, necrosis
GI	Duodenal ulcer	Perforation of ulcer
GU	Ruptured bladder	Obstruction and distension
Vascular	Aortic aneurysm	Rupture of aneurysm

Table 1-3. Examples of Erosion

System	*Disease*	*Nature of Erosion*
CNS	Meningitis	Erosion of abscess wall; mastoiditis
ENT	Pharyngeal carcinoma	Bleeding; erosion into blood vessels
Lung	Tuberculosis	Bleeding; granulomatous erosion into blood vessels
GI	Duodenal ulcer	Bleeding; ulcer erosion into blood vessels
GU	Bladder stone	Bleeding; erosion of bladder wall
Extremity	Raynaud's phenomenon	Digital ulceration; ischemic erosion of skin

(CSF), lymph, and other fluids, as well as air, is essential for health. Table 1-1 shows 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 mass. This explains in large measure why cancer is so often detected only after it induces one of the 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 different forms. Sometimes they appear unmistakably loud and clear; at other times they are soft, muted, and elusive. The able physician will learn to recognize and understand them. Such recognition and understanding are the chief concern of this book.

2

Wounds and Wound Healing

David W. Furnas
Richard D. Liechty

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 post-hepatitis 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 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 humans have, in their evolutionary past, virtually lost the ability to regenerate compound tissues. There remains, however, this remarkable process of sealing, or patching, on which humans depend to survive the 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 understanding 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 close a wound primarily, and when to leave it open are practical applications that the student should keep in mind as

he or she 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 that serves as a scaffold for formation of granulation tissue. 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. In an incised and sutured wound, the epidermal surface is intact within 24 hours. 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 day 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, 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 owing to 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 closed primarily, it must heal by *secondary intention,* by means of the mechanisms of contraction and epithelialization. Examples are wounds that are excessively contaminated, wounds whose treatment has been delayed, and burns and other wounds that involve necrosis of large areas of skin. 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*. A mantle of necrotic skin that clings to the surface of the defect is called an *eschar*. Formed of coagulated collagen and debris, it is much thicker and

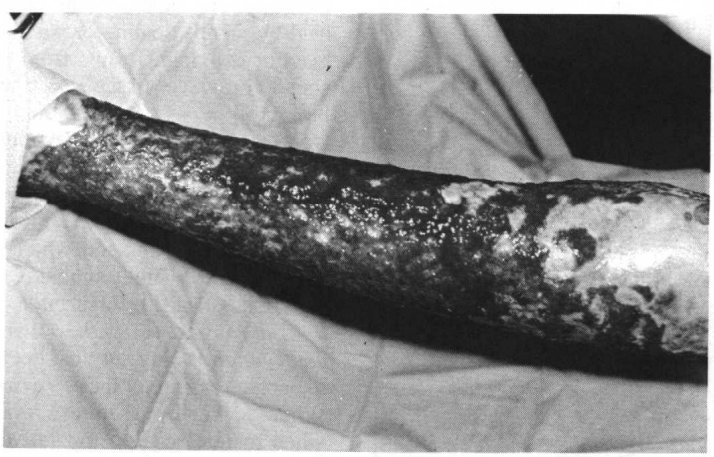

Fig. 2-1. Granulation tissue. Red, moist bed of fibroblasts and capillaries covering surface of leg that sustained full thickness burns 6 weeks before.

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 removal of dead tissue, debris, and secretions by surgical excision (débridement) and by intermittent application of dressings moistened with antibacterial solutions. The capillarity of the dressings drains away bacterial exudate. Dead tissue that adheres to the dressings is removed when the dressings are changed. There soon emerges a clean granulating surface that resists reinfection. If the wound edges can be apposed, wound closure can be hastened by *secondary closure* undertaken a few days after injury. Sutures are used to appose the wound edges, usually after the granulation tissue is first excised. 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 which can result in diminished motion and sometimes 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 optimal time during which to close a contaminated wound; if closure was not accomplished by 6 hours, 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 given *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 little risk of infection. A grossly clean, neat wrist laceration, 12 hours old, can be repaired safely. However, a 3-hour-old wound of the lower leg received from a dirty barnyard source should probably be left open.

A contaminated wound is always converted to a *surgically clean wound* before early closure, by the following steps:

1. Take culture; start antibiotics if wound is large; administer tetanus prophylaxis.
2. *Clean* all foreign material and loose debris by use of syringes, scrub brushes, and curets; avoid traumatic tattoos.
3. Accomplish *hemostasis.*
4. *Irrigate* with several liters of sterile solutions (saline, hydrogen peroxide, benzalkonium chloride) to dilute the number of bacteria remaining in the wound and to carry away microscopic debris.
5. *Match* the landmarks of the wound so that a tentative plan for débridement, shifting of tissue, and closure can be made.
6. *Débride*—excise with scalpel ragged wound edges, all dead or questionably viable tissue, and tissues that contain embedded foreign material.
7. *Close* with sutures, grafts, or pedicle, *or* if there is heavy contamination or missing tissue.
8. *Dress* frequently with moist antibiotic dressings and carry out delayed closure several days later *or* dress and await secondary healing.

PATHOLOGICAL WOUND HEALING

At times an excessively hard, raised, red, itching, unsightly *hypertrophied scar* (Fig. 2-5) may result from excessive tension on the wound, unfavorable site, inaccurate wound closure, or unknown factors. Exuberant

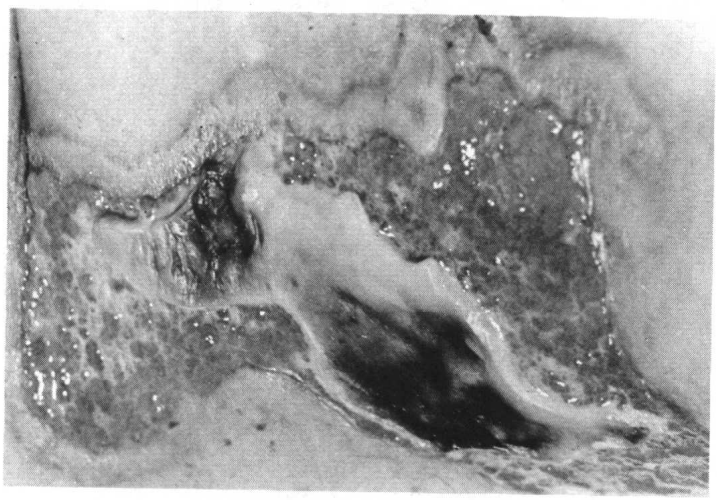

Fig. 2-2. Eschar. Deep burn coagulated full thickness of skin several weeks before. Eschar is in process of separating from underlying bed of granulation tissue. Copious exudate from local bacterial activity speeds process.