MANUAL OF SURGICAL THERAPEUTICS

FOURTH EDITION

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Departments of Surgery
The Medical College of Wisconsin
and University of Illinois

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NOTICE

The indications and dosages of all drugs in this manual have been recommended in the medical literature and conform to the practices of the general medical community at The Medical College of Wisconsin and at the University of Illinois. The medications described do not necessarily have specific approval by the Food and Drug Administration for use in the situations and the dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

PREFACE

The *Manual of Surgical Therapeutics* was originally developed to fill a pocket —both figuratively and literally—in the armamentarium of the surgical student and house officer. Before its conception there was no practical guide outlining the essentials of daily care of the surgical patient, no collection of readily available specific information for the person on the firing line. The worth of this manual has been attested to by our colleagues who have reviewed it, and we thank them. But more important, the value of this reference book has been affirmed by its large and wide readership—even senior surgeons and physicians have made it a part of their libraries.

In keeping with our original intention to make this a handy, even indispensable, volume we have retained the outline format as well as continued to present the general principles involved in the pathophysiological, pharmacological, and nonoperative aspects of the care of the surgical patient. The small atlas of minor surgical techniques, appended to the third edition in response to requests from our readers, remains, as do the suggested reading lists that follow most chapters.

For this fourth edition, the manual has been extensively revised and updated. Many of the chapters and sections have been completely rewritten, including those on chest trauma, urological trauma, cardiac arrhythmias, cardiac arrest, upper gastrointestinal hemorrhage, intestinal obstruction, aspiration pneumonia, cancer chemotherapy, and cutaneous and subcutaneous tumors.

The *Manual of Surgical Therapeutics* is the work of house officers and attending staff from the departments of surgery at the University of Illinois and at the Medical College of Wisconsin. Two exceptions must, however, be noted: Ronald L. Nichols and Robert M. Barone are, at this writing, at Tulane University and the University of California, San Diego, respectively; they were a part of our staffs until relatively recently.

We take this opportunity to express our gratitude to our collaborators, whose diligence and expertise are manifest in the pages that follow. We thank Ms. Carole Russell, who did most of the artwork, and Mr. George Spuda, Chief of the Medical Illustration Service at the Wood Veterans Administration Hospital, for their aid in enhancing the utility of this manual.

Mrs. Ann Haddick Napoletan once again made herself indispensable in the process of manuscript preparation and proofreading; our appreciation and gratitude for her capable and devoted assistance continues. We also wish to thank newcomers to this edition, Ms. Myrna Schlegel and Ms. Catherine Judge, for their aid in similar editorial duties. To Mr. Fred Belliveau of Little, Brown and Company, his skilled editorial staff, and, in particular, Ms. Christine Ulwick, our thanks and continued admiration.

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CONTENTS

1. RESUSCITATION FROM SHOCK AND TRAUMA

I. GENERAL PRINCIPLES

A. Initial Management

1. **Priorities** The primary objective in the care of an injured person is preservation of life. Treatment of airway obstruction, shock, or cardiorespiratory failure often must be started without exact knowledge of the cause of these disorders. Diagnostic and therapeutic measures are carried out concurrently rather than sequentially. Once the patient's condition is stable, etiological evaluation can be undertaken as a secondary objective.

   a. Establish and maintain an airway

   (1) Look for signs of obstruction: stridor, retraction, wheezing, or cyanosis. Sweep a finger deep into the oropharynx to remove clotted blood, mucus, vomitus, and any loose teeth or dentures.

   (2) Insert an **orpharyngeal airway** if the patient is obtunded or if unstable facial fractures cause obstruction. Ventilatory assistance with a mask is now possible. If a patient makes a violent effort to sit up, allow him to do so, since this action usually is a reflex response to maintain an open airway.

   (3) In comatose patients an **endotracheal tube** reduces aspiration and facilitates respiratory support. In a patient alert enough to make endotracheal intubation impractical, close observation and frequent suctioning are essential.

   (4) Examine for evidence of sucking chest wounds, flail chest, tension pneumothorax, or pulmonary injury. Treat airway obstruction (suction and intubation) and respiratory insufficiency (oxygen and assisted ventilation). Direct injury to the larynx or trachea may make endotracheal intubation impossible. In such rare instances, be ready to perform an emergency tracheostomy.

   (5) **Position** is important. The possibility of cervical or spinal cord injury necessitates immobilization and negates other positional considerations. Otherwise, keep the patient semi-

prone. The patient should not lie or be restrained flat on his back.

b. Assess and support cardiopulmonary function

(1) Hypoxia and acidosis can cause **cardiac arrest.** If arrest has occurred, mouth-to-mouth ventilation and closed chest massage are started immediately. Ventilation with an oral airway and a mask is sufficient in most patients (see Chap. 4). Time should not be wasted attempting a difficult endotracheal intubation during the early stages of resuscitation from cardiac arrest. If the chest wall is unstable (flail chest) or rigid (advanced emphysema) and closed chest massage is not effective, thoracotomy and direct manual cardiac decompression may be necessary.

(2) **Metabolic acidosis** should be anticipated in every patient with hypoperfusion and corrected with sodium bicarbonate given IV. Early arterial blood gas analyses are helpful. Electrocardiographic (ECG) monitoring is essential for the diagnosis of specific arrhythmias (see Chap. 3).

c. Control hemorrhage

(1) External hemorrhage is best controlled by **direct pressure over the site of bleeding.** Pressure is maintained until proximal control can be obtained in association with definitive treatment in the operating room. The use of tourniquets or of blind clamping in the depths of a wound is not advisable, since further injury may ensue.

(2) Internal hemorrhage may be identified by **thoracentesis** or **paracentesis.** Peritoneal lavage, interpreted by an experienced observer, increases diagnostic accuracy. Blood loss due to fractures, even without major vessel injury, can be appreciable.

d. Treat shock

(1) Hypovolemic shock is best treated by **blood replacement.** Blood loss of less than 1500 ml may be replaced by crystalloid solutions, but a greater blood loss should be replaced by blood. Patients with a loss of more than 45% of blood volume become severely hypotensive and hypoxic and will progress to cardiopulmonary arrest if not resuscitated vigorously. Clinical guides to assessment of blood loss are given in Table 9-4.

(2) The **essential steps in the management of hemorrhagic shock** are:

(a) Insert intravenous catheters Several catheters should be inserted using sterile techniques. At least one catheter

should be in the central venous pool. Vessels potentially involved by trauma are avoided. The clinical situation dictates which veins to use—almost always above the nipple line.

(b) **Draw blood samples for typing and cross matching** before giving plasma expanders. Determinations of arterial blood gases and hematocrit at this time provide baseline values and insight into the volume of bicarbonate buffering needed and the efficacy of respiratory support.

(c) **Administer IV fluids** Isotonic crystalloid solutions are administered very rapidly at first, with the rate of infusion slowed as the blood pressure and central venous pressure (CVP) begin to rise.

(d) **Transfuse** as soon as compatible cross-matched blood is available. In extreme cases in which crystalloid solutions are not adequate for resuscitation, or when the hematocrit is below 15%, type-specific or universal donor (O-negative) packed red blood cells may be given to maintain minimal oxygen-carrying capacity until cross-matched blood is available (see Chap. 15).

(e) **Monitor CVP**

(1) Fluctuation of the saline column in the manometer with respiration is a good indication that the tip of the central venous catheter rests in the central venous pool. The catheter tip should be within the thorax but outside the pericardium. Pulsation coincidental with the heartbeat indicates placement in the right ventricle, and the catheter should be withdrawn to an appropriate position. All catheters should be checked for position by chest x-ray when this becomes clinically feasible.

(2) Monitoring the CVP requires **serial observations of changes** with fluid replacement. The absolute value of the CVP measured in a hypovolemic patient is less meaningful than the change in CVP with therapy. A rise in CVP paralleling that of systolic blood pressure is seen with adequate volume replacement. A low CVP persisting after volume replacement may indicate continued occult bleeding. A significant elevation of CVP in the face of continued hypotension suggests cardiac tamponade, myocardial infarction, or congestive heart failure. Initial placement of a pulmonary artery catheter (Swan-Ganz) is not necessary, although such a catheter may be helpful later in treating patients who develop cardiac failure or myocardial infarction.

(f) **Catheterize the bladder with a Foley catheter** Measure urine output hourly. Decreased urine output with elevated specific gravity (above 1.030) or osmolality (above 700 mOsm/kg) reflects hypovolemia. Later, low urine output with low specific gravity and osmolality concurrent with normal vital signs may reflect renal tubular damage (see Chap. 11).

(g) **Immobilize fractures** "Splint 'em where they lie" remains the best rule. Protection of associated soft tissues, especially neurovascular structures, is of prime importance (see **VII**).

(h) **Dress soft tissue wounds** Elevation, compression for hemostasis, and sterile dressings protect against further injury and contamination. Definitive therapy can be carried out when anesthesia is available for adequate debridement and repair. Tetanus prophylaxis and antibiotics should be given when appropriate.

(i) **Establish the cause of shock** Inability to restore blood pressure and circulating volume by adequate blood volume replacement indicates that a source of uncontrolled hemorrhage is present. Hematuria, hemoptysis, bloody nasogastric suction, or rectal bleeding suggests internal sources of blood loss. Thoracentesis, paracentesis, peritoneal lavage, pyelography, or endoscopy may define the hidden bleeding site. Pelvic fractures frequently account for occult loss of as much as 10 units of blood. Continuing major hemorrhage usually requires operative exploration. *Prolonged preoperative attempts to stabilize the circulation in such patients may result in failure of resuscitation.*

2. **Immediate follow-up**

a. **Make repeated examinations of the patient,** with special attention to pulse rate, pulse pressure, skin temperature and color, and state of consciousness. A rapid pulse, narrow pulse pressure, blanched cool skin, and combativeness are signs of recurrent hypovolemia and hypoxia.

(1) After successful resuscitation, the patient enters a critical phase, requiring continuous observation and general evaluation to note changes in the level of consciousness, spontaneous motion of the extremities, chest excursions, and abdominal habitus. A nasogastric tube is inserted, the aspirate inspected, and the stomach emptied and kept on intermittent suction.

(2) **All patients must be completely disrobed.** A rapid assessment of the extent of injuries is performed by gentle but **firm palpation of all body parts,** especially those areas

where injuries are suspected. Obvious deformities, asymmetry, lacerations, and contusions require special attention. Palpation of the scalp, facial bones, trachea, and vertebral column, as well as gentle compression of the thorax, pelvis, and extremities, usually will elicit signs of hidden fractures or dislocations. A careful **abdominal examination** is mandatory; particular attention is paid to the presence of tenderness and the character of bowel sounds. **Rectal examination** is performed in all patients, with particular attention to the presence or absence of blood. Peripheral pulses and the neuromuscular status of the extremities are assessed. Chest auscultation and percussion may demonstrate signs of pleural collapse, rub, or effusion, as well as cardiac changes consistent with injury.

b. **Obtain as thorough a history as possible.** Previous medical problems, allergies, and medications used, as well as conditions concerning the accident, are important. Sources other than the patient may be necessary for an adequate history. During this period, serial determinations of blood gases and hematocrit and indicated laboratory and x-ray evaluations are carried out.

B. Pathophysiology of shock

1. **Definition** Shock is a state of hypoperfusion secondary to decreased effective circulating blood volume. Sympathetic neural responses, diverting blood so that perfusion to vital tissues is maintained, produce physiological and metabolic changes in poorly perfused tissues that eventually have profound general effects.

2. **Classification** Shock associated with trauma is primarily hypovolemic; associated problems may induce other forms of shock concurrently or subsequently.

   a. **Hypovolemic shock** is due to loss of blood volume, either from hemorrhage of whole blood or from third-space losses of plasma seen in peritonitis, pancreatitis, intestinal obstruction, and burns.

   b. **Endotoxic shock** is the result of peripheral pooling of blood in capacitance (primarily venous) vessels, decreasing the effective circulating blood volume without actual volume loss. Direct myocardial and peripheral effects add significantly to the complexity of the clinical picture.

   c. **Cardiogenic shock** represents pump failure with inadequate output and therefore inadequate tissue perfusion, in spite of normal blood volume.

   d. **Neurogenic shock** is caused by loss of sympathetic control of resistance vessels, with resultant dilation of arterioles and ven-

ules; the decrease in effective circulating volume produces shock. Hypotension due to spinal anesthesia is an example of this phenomenon.

3. **Endocrine response**

   a. Release of adrenocorticotropic hormone (ACTH), antidiuretic hormone (ADH), and aldosterone during hypotension results in renal retention of sodium, chloride, and water, with potassium loss and a decreased urinary volume.

   b. Release of epinephrine and norepinephrine from the adrenal medulla produces peripheral vasoconstriction, maintaining blood pressure by decreasing the volume of the vascular space as well as by mobilizing intravascular fluid from peripheral tissues to the central pool. With prolonged peripheral vasoconstriction, anaerobic metabolism results in accumulation of acidic metabolites (Fig. 1-1).

   c. Hyperglycemia develops during shock and generally has been attributed to the glycogenolytic properties of corticosteroids and epinephrine. Recent studies demonstrating depression of insulin secretion in shock imply more intricate relationships.

4. **Metabolic effects.** A normally perfused cell utilizes glucose in the glycolytic and citric acid pathways to form energy through adenosine triphosphate. Without oxygen, pyruvate is transformed anaerobically to lactic acid (see Fig. 1-1), which then accumulates and results in acidosis. Amino acids and fatty acids that normally would enter oxidative pathways for energy production also accumulate in shock, compounding the metabolic acidosis. Oxygen deficit and acidosis eventually interfere with cell membrane func-

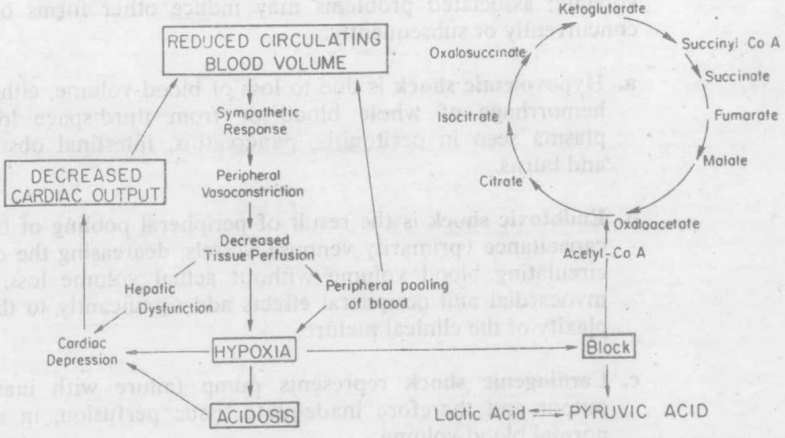

Figure 1-1. Shock leads to tissue hypoxia, with blockage of normal aerobic metabolism. Lactic acid accumulates, resulting in tissue acidosis.

tion. Intracellular potassium is lost; sodium and water move into the cell, producing cellular edema.

5. **Cardiorespiratory response** The intense sympathetic response during shock increases cardiac output by augmenting the rate and force of cardiac contraction in addition to increasing peripheral resistance. Since myocardial perfusion occurs primarily during diastole, tachycardia depresses myocardial perfusion, resulting in myocardial acidosis with prolonged shock. Although metabolic acidosis is compensated initially by increasing ventilation to augment carbon dioxide elimination, profound acidosis combined with hypoxia (both primary and secondary to decreased myocardial perfusion) results in myocardial depression, irritability, and susceptibility to arrhythmias.

C. Choice of replacement fluids in hypovolemia

1. **Crystalloid solutions** Initial volume replacement with normal saline or Ringer's lactate provides effective intravascular expansion. There are theoretical advantages to a buffered salt solution such as Ringer's lactate; despite concern about accumulation of lactate, it has not proved to be clinically important. Diffusion of noncolloid salt solutions out of the intravascular space is relatively rapid, so that a volume of up to four times the amount of blood lost must be infused as crystalloid solution to restore circulating blood volume.

   It is desirable to maintain the hematocrit above 30%. At these values, oxygen-carrying capacity can protect against further hypoxia. Thus, dilution of hemoglobin is the limiting factor in the volume of crystalloid solutions to be administered. **There is no place for salt-free crystalloid solutions (dextrose in water) in primary resuscitation.** When these solutions are given in acute posttraumatic states, water is retained because of the action of ADH, and severe hypo-osmolality may result.

2. **Albumin** Human serum albumin in various concentrations is readily available and free of the risk of hepatitis. It is an effective plasma expander and also has other advantages; e.g., it is easily metabolized and spares body muscle proteins from catabolism. However, transudation of albumin into the pulmonary interstitium has been implicated in adult respiratory distress syndrome when large volumes of albumin-containing solutions are administered during resuscitation. Delineation of problems in this area remains somewhat obscure.

3. **Plasma and plasma protein fraction** (Plasmanate) may be given without cross matching. Their use should be especially considered when plasma losses are anticipated, as in burns, peritonitis, and pancreatitis. The use of fresh-frozen plasma provides coagulation factors for patients with clotting abnormalities. There is a risk of hepatitis transmission by plasma. This risk is reduced by the use of single donor units, but cannot be entirely eliminated. Plasma pro-

tein fraction carries no risk of hepatitis and is preferred for most clinical situations.

4. **Dextrans** Both clinical dextran and low molecular weight dextran are effective plasma expanders; they also decrease blood viscosity and improve microcirculatory flow. Dextrans may interfere with platelet functions. The use of dextrans prior to exploration of soft tissue injuries is ill advised. Although often effective in preventing renal failure, low molecular weight dextran also can be responsible for renal failure due to "tubular burn" resulting from the very hyperosmolar urine formed if dextran is excreted in an inadequate volume of urine. No more than 1 liter of dextran solution should be given each day.

5. **Blood transfusion**

   a. Major losses of whole blood may be replaced with whole blood or packed red blood cells with a plasma substitute (albumin, Plasmanate, fresh-frozen plasma). It is generally possible to provide adequate volume expansion initially with crystalloid solutions until cross matching can be accomplished. The use of matched blood minimizes reactions due to blood group incompatibilities and isosensitization.

   b. Banked blood becomes progressively acidic and hyperkalemic, and there is a rapid loss of many coagulation factors (see Chap. 15).

   c. In addition to the high incidence of transfusion reactions with massive transfusion (> 20 units), it also carries the potential of causing the development of "wet lung" due to pulmonary entrapment of fibrin and other aggregates in the blood. The use of a microaggregate filter is advisable.

6. **Buffer therapy** The development of metabolic acidosis during shock can be countered by administration of buffer agents such as sodium bicarbonate. Generally, **sodium bicarbonate** is a sufficient adjunct to volume replacement in the treatment of shock. Tromethamine (THAM) is an effective intracellular and extracellular buffer and may be used if acidosis is severe (arterial pH below 7.2). The side effect of central respiratory arrest has limited the clinical use of THAM. Rapid changes in pH may be induced and cause cardiac arrhythmias, so that patients receiving rapid infusions of buffers should have continuous ECG monitoring.

D. Adjunctive therapy in shock

1. Oxygen therapy and ventilatory support

   a. All patients who have dyspnea or tachypnea should be given oxygen. The most important mechanism of oxygen transport (over 95%) is the hemoglobin in erythrocytes. A normal

hemoglobin of 15 gm/100 ml provides transport for 20 vol% of oxygen; a hemoglobin of 7 gm/100 ml (hematocrit 21%) carries only 10 vol%, which is the critical reserve level of oxygen consumption for most tissues, especially the myocardium and brain.

b. Oxygen given by a properly applied mask or nasal catheter at 8 liters/min will increase inspired oxygen concentrations by 10% and improve hemoglobin saturation and oxygen delivery to the tissues. Oxygen administered through a T-adapter and an endotracheal or tracheostomy tube will increase the inspired oxygen concentration to 40%. Oxygen delivery in concentrations above 50% is unnecessary in the management of most patients. Pulmonary oxygen toxicity may result if 100% oxygen is administered continuously for 24 hr.

c. It should be remembered that cyanosis, a generally reliable clinical sign of inadequate oxygenation, may be absent if blood loss has reduced the circulating hemoglobin concentration below 5 gm/100 ml.

d. Ventilatory support is indicated whenever voluntary respiratory volumes are inadequate for specific conditions, such as in cardiopulmonary arrest, mechanical chest-wall dysfunction, atelectasis, or pulmonary edema (see Chap. 12). Repeated examinations for hemothorax or pneumothorax prior to and during ventilatory support are especially important. Previously undetected injury may be responsible for the development of a tension pneumothorax when positive-pressure ventilation is employed.

e. High inspiratory pressure, continuous positive pressure, and the use of expiration retarding devices all may contribute to increased resistance to pulmonary blood flow. The positive pressure is transmitted to the alveolar space, pulmonary capillaries are compressed, and blood flow through the pulmonary circulation is diminished. Decreased return to the left side of the heart causes a fall in cardiac output and may prolong or increase shock, especially in a hypovolemic patient.

2. **Thermal support** Hyperpyrexia can contribute to the deleterious cellular metabolic effects of shock and appears frequently in endotoxic shock. The patient should be kept at normal temperature by means of a hypothermia blanket and administration of acetaminophen, aspirin, or sodium salicylate.

3. **Antibiotics** Massive trauma and shock are associated with depression of reticuloendothelial function. During the early posttraumatic period, the ability of the body to clear bacteria from the circulation is depressed. Prophylactic antibiotic therapy frequently is indicated in such circumstances. The choice of antibiotic therapy depends on the type of injury involved. A cephalosporin is usually