HANDBOOK OF SURGERY

SIXTH EDITION

Edited by

THEODORE R. SCHROCK, MD

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University of California, San Francisco

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PREFACE

Handbook of Surgery is a concise, portable first reference for the student, resident, and nonspecialist physician. It is intended for use on the scene to give the reader some idea of what questions to ask in taking the history, what physical signs to elicit, what tests to order, what other diagnostic possibilities to consider, and what treatment to institute immediately. This handbook is not a substitute for standard textbooks or original literature. Discussions of diagnosis and treatment are sufficient to help the nonspecialist handle problems on the spot, but it is assumed that the reader will take the earliest opportunity to consult references which discuss the topics more thoroughly.

Handbook of Surgery was edited by Dr. John L. Wilson and published by Lange Medical Publications through five editions over the past 18 years. This, the Sixth Edition, has a new editor and a new publisher. Most of the text has been rewritten by new contributors; authors in previous editions have revised and updated their material extensively. The contents have been reorganized, the format altered, and the type reset.

I am indebted to the contributors for submitting authoritative material despite the constraints imposed upon them by the style and objectives of this book. I am grateful to my publisher, Richard C. M. Jones, for his patience when the expected flood of edited manuscript proved to be a mere trickle. Both Mr. Jones and I are warmly appreciative of the close cooperation and support of Dr. Jack Lange in bringing this project to completion.

Theodore R. Schrock

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SHOCK & TRAUMA

Donald Trunkey, MD

I. SHOCK

Shock is defined as peripheral circulatory failure causing tissue perfusion to be inadequate to meet the nutritional requirements of the cells and remove the waste products of metabolism. In the simplest terms, therefore, shock is **inadequate tissue perfusion.**

Shock may be classified as hypovolemic, septic, cardiogenic, neurogenic, or miscellaneous (e.g., anaphylactic reactions and insulin shock).

A. HYPOVOLEMIC SHOCK is the result of decreased blood volume due to acute and severe loss of blood, plasma, or body water and electrolytes. Hemorrhage, burns, bowel obstruction, peritonitis, and crush injuries are some of the common causes. A fall in venous pressure, a rise in peripheral vascular resistance, and tachycardia are characteristic of hypovolemic shock.

Factors that make a patient especially susceptible to hypovolemic shock include: **age** (the very young and the elderly tolerate loss of body water or plasma poorly); **chronic illness** (such patients often have a reduced blood volume and relatively small acute losses may precipitate shock); **anesthesia** (paralysis of vasomotor tone may cause shock in a patient who has compensated for a reduced blood volume); **adrenal insufficiency** (profound hypotension may be induced by minimal stress if corticosteroids are not supplied during and after trauma, operation, or illness).

1. Pathophysiology Events in the microcirculation progress in phases.

a. Compensation phase The first response of the circulation to hypovolemia is contraction of the precapillary arterial sphincters; this causes the filtration pressure in the capillaries to fall. Since osmotic pressure remains the same, fluid moves into the vascular space with a corresponding increase in blood volume. If this compensatory mechanism is adequate to return blood volume to normal, the capillary sphincters relax and microcirculatory flow returns to normal. If shock is prolonged and profound, the next phase is entered.

b. Cell distress phase If vascular volume has not been restored, the precapillary sphincters remain closed, and arteriovenous shunts open up to divert arterial blood directly back into the venous system, thus maintaining circulation to more important organs such as the heart and brain. The cells in the bypassed segment of the microcirculation must rely on anaerobic metabolism for energy. The amount of glucose and oxygen available for the cell decreases, and metabolic waste products such as lactate accumulate. Histamine is released, resulting in closure of the postcapillary sphincters, and this mechanism serves to slow the remaining capillary flow and hold the red blood cells and nutrients in the capillaries longer. The empty capillary bed constricts almost completely; very few capillaries remain open.

c. Decompensation phase Just before cell death, local reflexes (probably initiated by acidosis and accumulated metabolites) reopen the precapillary sphincters while the postcapillary sphincters stay closed. Prolonged vasoconstric-

tion of the capillary bed damages endothelial cells and results in increased capillary permeability. When the capillaries finally reopen, fluid and protein are leaked into the interstitial space, the capillaries distend with red blood cells, and sludging occurs. Cells become swollen, they are unable to utilize oxygen, and they die.

d. Recovery phase If blood volume is restored at some point in the decompensation phase, the effects on the microcirculation may still be reversible. Badly damaged cells may recover, and capillary integrity may be regained. The 'sludge' in the microcirculation is swept into the venous circulation and eventually into the lungs where these platelet and white cell aggregates are filtered out and produce postshock pulmonary failure (see Chapter 2). Other capillaries may be so badly damaged and filled with sludge that they remain permanently closed; cells dependent upon these capillaries die.

Table 1-1. Clinical classification of hypovolemic shock

Mild shock (up to 20% blood volume loss)
Definition: Decreased perfusion of nonvital organs and tissues (skin, fat, skeletal muscle, and bone).
Manifestations: Pale, cool skin. Patient complains of feeling cold.

Moderate shock (20–40% blood volume loss)
Definition: Decreased perfusion of vital organs (liver, gut, kidneys).
Manifestations: Oliguria to anuria and slight to significant drop in blood pressure.

Severe shock (40% or more blood volume loss)
Definition: Decreased perfusion of heart and brain.
Manifestations: Restlessness, agitation, coma, cardiac irregularities, ECG abnormalities, and cardiac arrest.

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2. Diagnosis Clinical assessment permits classification of hypovolemic shock as mild, moderate, or severe (Table 1-1). The compensatory mechanisms act to preserve blood flow to the heart and brain at the expense of all others; thus, in severe shock, there is marked constriction of all other vascular beds.

3. Treatment Shock is an acute emergency: **act promptly!**

a. Keep the patient recumbent—do not move the patient unnecessarily.

b. Establish and maintain an airway.

c. Place one or more large intravenous catheters.

(1) Do a cutdown in the long saphenous vein at the ankle; this method is rapid and safe.

(2) Do a cutdown on the basilic vein in the antecubital space so that central venous pressure can be monitored.

(3) Percutaneous insertion of subclavian or jugular catheters is not recommended because the veins are collapsed in hypovolemic shock. Femoral vein catheters may be placed percutaneously in unusual circumstances, e.g., when a single physician is available for resuscitation.

d. Parenteral fluids Begin **immediately** to restore blood volume. In mild or moderate shock, it makes little difference which fluid is used (Table 1-2). In severe shock, the choice of fluid is important because endothelial permeability may be increased throughout the body, resulting in 'capillary leak' which compounds the problems if colloid is given.

(1) *Crystalloids* are preferred in the initial treatment of shock. They are readily available and effectively restore vascular volume for brief periods. Crystalloids also lower blood viscosity and enhance resuscitation of the microcirculation. Balanced salt solutions plus judicious amounts of sodium bicarbonate correct the acidosis which invariably is present in shock. Serial blood pH measurements are a guide. Overcorrection of acidosis is more harmful than the opposite.

Table 1-2. Fluid resuscitation of shock

I. Crystalloids
  A. Isotonic sodium chloride
  B. Hypertonic sodium chloride
  C. Balanced salt solution
    1. Ringer's lactate
    2. *Ringer's acetate*
    3. Normosol, Plasmolyte, etc.
II. Colloid
  A. Blood
    1. Low-titer O negative blood
    2. *Type-specific*
    3. Typed and crossed
    4. Washed red cells
    5. Fresh red cells
  B. Plasma and its components
    1. Plasma—fresh frozen
    2. Albumin
    3. Plasmanate
  C. Plasma substitutes
    1. Clinical dextran (M.W. 70,000)
    2. Low molecular weight dextran (M.W. 40,000)

(2) *Colloids*

(a) *Blood* is available in emergencies as low-titer O negative or type-specific. O negative blood has the theoretical disadvantage of isoimmunization or difficulty with typing and crossmatching later; this is probably not a major consideration. Type-specific blood can be used until crossmatched blood becomes available (about 45 minutes).

If shock persists after 2 liters of crystalloid have been infused, or if shock recurs after the patient initially responds, whole blood should be transfused immediately.

(b) *Plasma* and albumin solutions are detrimental in prolonged severe shock. These substances leak through capillary membranes taking water with them, thus exacerbating the interstitial edema. Plasma and its components should be withheld until capillaries regain their integrity (about 24 hours).

(c) *Plasma substitutes* (dextrans) interfere with function of the reticuloendothelial system and depress the already impaired immune mechanisms in shock patients. Clinical dextran coats red cells, making typing and crossmatching difficult; low molecular weight dextran coats platelets and may contribute to bleeding.

e. The underlying cause of shock should be investigated and treated while resuscitation is underway. Failure of resuscitation almost always reflects persistent massive hemorrhage, and definitive operative treatment offers the only chance for survival.

f. Evaluation of treatment The amount of fluid that a patient should receive is governed by the patient's response; there is no rigid formula. Constant close monitoring is essential (see Table 1-3). Atrial filling pressure and urine output are the most useful signs.

Table 1-3. Variables frequently monitored in shock

Measurement	Typical normal values	Typical values in severe shock
Arterial blood pressure	120/180	< 90 mm Hg systolic
Pulse rate	80/minute	> 100/minute
Central venous pressure	4-8 cm saline	< 3 cm
Hematocrit	35-45%	< 35%
Arterial blood:		
pH	7.4	7.3
pO_2	95 mm Hg	85 mm Hg
pCO_2	40 mm Hg	< 30 mm Hg
$-HCO_3$	23-25 mEq/liter	< 23 mEq/liter
Lactic acid	12 mg/100 ml	> 20 mg/100 ml
Urine:		
Volume	50 ml/hour	< 20 ml/hour
Specific gravity	1.015-1.025	> 1.025
Osmolality	300-400 mOsm/ kg water	> 700 mOsm/ kg water

(1) *Left atrial filling pressure* is rarely measured directly, but the pulmonary artery wedge pressure is a useful approximation, and it should be monitored in critical patients. Central venous pressure is sufficiently accurate in the majority of patients. In mild or moderate shock, resuscitation may be permitted to raise atrial filling pressure as high as 24 torr without risk. In severe shock, however, atrial filling pressure must be kept at or near normal (3-8 torr) because higher pressures aggravate interstitial edema.

(2) *Urine output* should be monitored; this usually requires a urinary catheter. Urine output greater than 0.5 cc/kg/hour is a good index of visceral blood flow, specifically renal blood flow.

(3) Additional signs of successful resuscitation include an alert, oriented patient and adequate peripheral perfusion as judged by clinical criteria.

(4) Blood pressure, pulse rate, and respiratory rate should be recorded every 15-30 minutes.

(5) Hematocrit should be measured every few hours if continued bleeding is suspected. The hematocrit usually falls gradually over a period of 24-48 hours because of hemodilution even if bleeding has stopped.

(6) Blood gases should be determined repeatedly (see Table 1-3).

(7) Other measurements, obtained in certain circumstances, include cardiac output and oxygen consumption.

g. Failures of resuscitation

(1) If both atrial filling pressure and urine output are increased, too much fluid is being given, and the infusion rate should be slowed immediately.

(2) If both atrial filling pressure and urine output are below normal, more volume is required.
(3) When atrial filling pressure is elevated and urine output is low, measurement of cardiac output is useful.
   (a) High atrial filling pressure, low urine output, and high cardiac output indicate deficient renal function. (i) Give mannitol (12.5-25 gm IV) followed by infusion of mannitol 50 gm in 500-1000 ml of balanced salt solution. No more than 75-100 gm of mannitol should be given. (ii) If there is no response to mannitol, give small doses of furosemide (10-20 mg IV) or ethacrynic acid (50 mg IV). These diuretics may cause vasodilatation and redistribution of blood flow within the kidney.
   (b) High atrial filling pressure, low urine output, and low cardiac output suggest that an inotropic agent is needed (Table 1-4). (i) *Dopamine hydrochloride,* 200 mg in 500 ml of sodium injection USP (400 μg/ml), is given initially at a rate of 2.5 μg/kg/minute. These doses stimulate both the dopaminergic receptors, which increase the renal blood flow and urine output, and the beta-adrenergic cardiac receptors, which increase the cardiac output. Higher levels stimulate alpha receptors to cause systemic vasoconstriction, and doses above 20 μg/kg/minute reverse the vasodilatation of the renal vessels achieved at lower levels. (ii) *Isoproterenol,* a beta-adrenergic stimulator, increases cardiac output by its action on the myocardial contraction mechanism, and it also produces peripheral vasodilatation. Give 1-2 mg in 500 ml of 5% dextrose in water IV. Isoproterenol should not be used if the heart rate is greater than 100-120/minute lest cardiac arrhythmias develop. (iii) *10% calcium chloride* (10 cc) may be administered directly IV over 2-3 minutes provided there is continuous cardiac monitoring for arrhythmias. Although calcium may produce an instant inotrophic effect, it is usually not sustained, and repeated doses are required. Measurement of ionized calcium levels are prudent in such instances.
(4) There is no convincing evidence that corticosteroids or ganglionic blocking drugs are of value in hypovolemic shock.

B. SEPTIC SHOCK is most often due to gram-negative septicemia, although infection by gram-positive bacteria can also cause shock. Trauma, diabetes mellitus, hematologic diseases, corticosteroid therapy, immunosuppressive drugs, and radiation therapy increase susceptibility to infection and thus predispose to septic shock. Precipitating events are often operations on the urinary, biliary, or gynecologic systems.

1. Pathophysiology

a. Gram-negative septicemia causes a generalized increase in capillary permeability, loss of fluid from the vascular space, and pooling of blood in the microcirculation. All of these mechanisms contribute to hypovolemia. There may also be a direct toxic effect on the heart, with depression of myocardial function. Peripheral vascular resistance usually is lowered as the result of arteriovenous shunting.

b. Gram-positive septicemia occasionally produces hypovolemia, but the loss of fluid from the vascular space usually is limited to the area of infection.

c. Disseminated intravascular coagulation (DIC) may develop in septic shock (see pages 49 and 84).

2. Diagnosis

a. Symptoms and signs (1) The inciting infection may be obscure. (2) Confusion and restlessness are early indications. (3) The skin is warm and the

Table 1-4. Adrenergic drugs used in hypotensive states.
(Effects graded on a scale of 0–5.)

Drug	Vasomotor Effect		Cardiac Stimulant (Inotropic Effect)	Cardiac Output	Renal and Splanchnic Blood Flow
	Vaso-constriction	Vaso-dilatation			
Alpha-adrenergic					
Phenylephrine (Neo-Synephrine)	5	0	0	Reduced	Reduced
Mixed alpha- and beta-adrenergic					
Norepinephrine (Levophed)	4	0	2	Reduced	Reduced
Metaraminol (Aramine)	3	2	1	Reduced	Reduced
Epinephrine (Adrenalin)	4	3	4	Increased	Reduced
Dopamine (Intropin)*	2	2	2	Usually increased	Increased
Beta-adrenergic					
Isoproterenol (many trade names)	0	5	4	Increased	Usually reduced

*Claimed to have a special (dopaminergic) receptor.

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pulses full initially; vasoconstriction develops later. (4) Pulmonary hypertension and hyperventilation. (5) Urine output is normal at first, then it slows rapidly.

b. Laboratory tests (1) Inability to metabolize glucose (glucosuria, hyperglycemia) is an early finding. (2) Respiratory alkalosis. (3) Hemoconcentration is common. (4) Early leukopenia followed by leukocytosis; usually the leukocyte count is 15,000 or more with a shift to the left. (5) Identification of the

Table 1-5. Biochemical and metabolic effects of corticosteroids

1. ↑ Hepatic glucose output
2. Hyperaminoacidemia
3. ↑ Secretion of glucagon
4. Inhibition of lipogenesis–selective
5. Induces negative calcium balance
6. Blocks ↑ capillary endothelial permeability
7. Markedly inhibits exudation of inflammatory cells
8. May maintain plasma membrane integrity
9. Suppresses T helper cell
10. Stabilizes lysosome membrane
11. Exhibits myocardial inotropism

responsible organism(s) is urgent. Obtain cultures on samples of blood, sputum, urine, drainage fluid, and any other suspicious site. A gram-stained smear of infected fluid may suggest the origin of the problem and guide emergency therapy.

3. Treatment As in other forms of shock, the objective of treatment is to improve tissue perfusion. In addition, the underlying infection must be treated.

a. Volume replacement The initial fluid should be balanced salt solution; colloids are particularly prone to leak from capillaries and aggravate interstitial edema in septic shock. Fluid volume is adjusted by close monitoring as described for hypovolemic shock.

Table 1-6. Acute complications of corticosteroid therapy in the shock patient

1. Peptic ulceration
2. Intestinal perforation
3. Pancreatitis
4. Sodium and water retention
5. Impaired wound healing
6. Suppression of the immune response

b. Antibiotic therapy Large doses of specific antibiotics should be given if the organism is known; if not, a 'best guess' should be made as to the responsible bacteria, and antibiotics are given accordingly (see Table 4-1).

c. Surgical drainage If an abscess or other accessible focus of infection is identified, it should be drained, debrided, or decompressed promptly. Antibiotics and fluid resuscitation will not salvage the patient if the source of infection is not found and drained.

d. Supportive measures Close attention should be paid to maintenance of ventilation. Accompanying disorders must be treated. If the patient continues to deteriorate, cardiovascular support with inotropic agents may be required as in hypovolemic shock.

e. Corticosteroid therapy Corticosteroids have both beneficial and deleterious effects in septic shock (Tables 1-5 and 1-6). Because the disadvantages outweigh the advantages, the use of corticosteroids in septic shock cannot be recommended.

f. Treatment of DIC (see page 84) If hemorrhagic manifestations of DIC develop, **heparin** should be given (100 units/kg IV initially, then 1000–3000 units/hour by continuous IV infusion). Response to heparin is indicated by improvement of bleeding and a rise of factors 5 and 7 and fibrinogen within 12 hours. Platelets may increase at a slower rate. Discontinue heparin therapy when the cause of DIC has been corrected and coagulation factors have returned to hemostatic levels.

C. CARDIOGENIC SHOCK Some degree of cardiac failure, usually left ventricular, can be detected in 20–50% of patients with acute myocardial infarction.

1. Diagnosis Clinical findings are often absent or minimal. Dyspnea, pulmonary rales, diastolic gallop, accentuated pulmonary second sound, pulsus alternans, and pulmonary venous congestion on chest x-ray may or may not be present. The radiographic changes take time to develop and are slow to resolve, so they are not very helpful acutely. Hypotension is often the first sign that cardiac failure is more severe than suggested by the other parameters.

2. Treatment

a. Treatment of *mild left ventricular failure* consists of oral diuretics (e.g., hydrochlorothiazide 50–100 mg), oxygen, and limitation of sodium intake.

b. More aggressive treatment is required for *severe left ventricular failure.* Such patients should have monitoring of arterial pressure, pulmonary artery wedge pressure, and cardiac output. The stroke work index can be computed, and rational therapy is based on the specific hemodynamic abnormality found.

(1) Low left ventricular filling pressure (less than 12 torr), normal cardiac output, and low arterial pressure indicate hypovolemia. Replace volume, beginning with 100 ml of saline or balanced salt solution. If cardiac output does not increase as left ventricular filling pressure rises to 15–20 torr, stop volume replacement to avoid pulmonary edema which may occur abruptly.

(2) Elevated left ventricular filling pressure, normal cardiac output, and normal blood pressure suggest that vigorous diuresis should be attempted with large doses of furosemide. Avoid volume depletion from excessive diuresis.

(3) Normal left ventricular filling pressure, normal cardiac output, and low arterial pressure reflect a failure of compensatory peripheral vasoconstriction. Give epinephrine or dopamine to stimulate beta-adrenergic receptors. These drugs should be infused slowly to avoid tachycardia, hypertension, and ventricular arrhythmias. The goal is to maintain blood pressure but not increase the stroke work index.

(4) Elevated left ventricular filling pressure (more than 20 torr), low cardiac output, and arterial blood pressure at or above 90 torr, is a pattern for which vasodilator therapy can be given. Drugs such as sodium nitroprusside, phentolamine, or nitroglycerine infused slowly IV, decrease the impedance to left ventricular ejection. Re-

duced left ventricular volume and filling pressure may improve the left ventricular stroke work index, lower the myocardial oxygen consumption (MVO_2), and improve perfusion to the brain, heart, and kidneys. The arterial blood pressure should be 90 torr or more before vasodilators can be given safely; if vasopressors cannot be used to raise blood pressure without elevating left ventricular filling pressure and aggravating cardiac failure, aortic balloon counter pulsation may be useful as a temporary aid to make vasodilator therapy possible.

D. NEUROGENIC SHOCK is due to a failure of arterial resistance from nervous or psychic stimulation (e.g., sudden pain or fright), vasodilator drugs (nitrites), spinal anesthesia, or spinal trauma. Blood pools in dilated capacitance vessels, and blood pressure falls. Cardiac activity increases to fill the dilated vascular bed and preserve tissue perfusion.

Prodromal symptoms and signs are pallor, cold sweat, weakness, light headedness, and occasionally nausea. Fainting is accompanied by transient hypotension and bradycardia.

Neurogenic shock is self-limiting. Resting in a recumbent or head-down position with the legs elevated for a few minutes is usually sufficient. If the patient is sitting down, and reclining is not possible, have him bend forward with his head between his knees. When faintness or prostration persists, other types of shock must be considered.

High spinal anesthesia induces neurogenic shock by paralyzing the vasoconstrictor nerves. Treatment consists of placing the patient in the head-down (Trendelenburg) position and administering a vasopressor agent. Acute traumatic paraplegia or quadriplegia causes neurogenic shock; however, associated injuries are common in these patients, and hypovolemia must be assumed to be responsible for shock until proved otherwise.

E. ANAPHYLACTIC REACTIONS These catastrophic allergic reactions may occur within seconds or minutes after the parenteral administration of animal sera or drugs; rarely, anaphylaxis develops after oral ingestion of drugs or foods. Anaphylaxis represents hypersensitivity induced by previous injection or ingestion, although occasionally no history of earlier exposure can be obtained.

1. Diagnosis The most conspicuous clinical feature may be laryngeal edema, bronchospasm, or vascular collapse. Symptoms and signs include apprehension, generalized urticaria or edema, a choking sensation, wheezing, cough, or status asthmaticus. In severe cases, hypotension, loss of consciousness, dilatation of pupils, incontinence, convulsions, and death occur suddenly.

2. Treatment Anaphylaxis is a life-threatening emergency. **Act immediately!**

a. Position the patient for comfort and ease of respiration.

b. Establish an airway and maintain oxygenation. If respirations have ceased, give artificial respiration by the mouth-to-mouth, mask, or endotracheal tube technics (see Chapter 3).

c. Drug therapy Epinephrine is the drug of choice for emergency use. It may be necessary to give intravenous antihistaminics, steroids, and aminophylline also.

(1) *Epinephrine hydrochloride:* give 1 ml of 1:1000 solution IM; repeat dose in 5-10 minutes and later as needed. For more rapid effect, give 0.1-0.4 ml of 1:1000 solution in 10 ml of saline slowly IV.

(2) *Antihistaminics:* give diphenhydramine hydrochloride (Benadryl) or tripelenamine hydrochloride (Pyribenzamine) 10-20 mg IV if the response to epinephrine is not prompt and sustained.

(3) *Steroids:* give hydrocortisone hemisuccinate (Solu-Cortef) 100-250 mg or prednisolone hemisuccinate (Meticortelone Soluble) 50-100