PRINCIPLES OF Trauma Care

THIRD EDITION

G. Tom Shires, M.D.

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Lewis Atterbury Stimson Professor and Chairman
Department of Surgery
Cornell University Medical College
and
Surgeon-in-Chief
The New York Hospital-Cornell Medical Center
New York

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PRINCIPLES OF TRAUMA CARE

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List of Contributors

WILLIAM D. ARNOLD, M.D.
Professor of Clinical Surgery (Orthopedics), Cornell University Medical College, New York; Director, Combined Fracture Service, The New York Hospital-Cornell Medical Center and Hospital for Special Surgery

YONG HO AUH, M.D.
Associate Professor of Clinical Radiology, Cornell University Medical College, New York

CHARLES R. BAXTER, M.D.
Frank H. Kidd, Jr., Professor of Surgery, The University of Texas Southwestern Medical School, Dallas

DAVID A. BEHRMAN, D.M.D.
Clinical Instructor in Surgery, Cornell University Medical College, New York; Assistant Attending Oral Surgeon, The New York Hospital-Cornell Medical Center

STANLEY J. BEHRMAN, D.M.D.
Clinical Professor of Surgery (Oral), Cornell University Medical College, New York; Attending Oral Surgeon-in-Charge, The New York Hospital-Cornell Medical Center

C. JAMES CARRICO, M.D.
Professor and Chairman, Department of Surgery, University of Washington School of Medicine, Seattle

W. KEMP CLARK, M.D.
Professor and Chairman, Division of Neurological Surgery, The University of Texas Southwestern Medical School, Dallas

PETER DINEEN, M.D.
Professor of Surgery, Cornell University Medical College, New York

G. B. DONNAN, M.B., B.S., F.F.A.R.A.C.S.
Visiting Assistant Professor of Anesthesiology, The University of Texas Southwestern Medical School, Dallas

RONALD L. FELLMAN, M.D.
Glaucoma Associates of Texas, Dallas; Clinical Instructor of Ophthalmology, The University of Texas Southwestern Medical School, Dallas

WILLIAM A. GAY, Jr., M.D.
Professor and Chief, Division of Cardio-Thoracic Surgery, Cornell University Medical College, New York

A. H. GIESECKE, Jr., M.D.
Jenkins Professor, Chairman, Department of Anesthesiology, The University of Texas Southwestern Medical School, Dallas

CLEON W. GOODWIN, M.D.
Johnson & Johnson Associate Professor of Surgery, Cornell University Medical College, New York; Director of the Burn Center, The New York Hospital-Cornell Medical Center

LEONARD D. HUDSON, M.D.
Professor and Chief, Division of Respiratory Disease, Department of Medicine, University of Washington School of Medicine, Seattle

ELIAS KAZAM, M.D.
Associate Professor of Radiology, Cornell University Medical College, New York

LARRY G. LEONARD, M.D.
Associate Professor of Surgery, University of Utah School of Medicine, Salt Lake City

STEPHEN F. LOWRY, M.D.
Assistant Professor of Surgery, Cornell University Medical College, New York; Director, Hyperalimentation Unit, The New York Hospital-Cornell Medical Center

JOHN P. LYDEN, M.D.
Clinical Associate Professor of Surgery (Orthopedics), Cornell University Medical College, New York; Associate Attending Orthopedic Surgeon, The New York Hospital-Cornell Medical Center and Hospital for Special Surgery

JOHN R. LYNN, M.D.
Glaucoma Associates of Texas, Dallas; Clinical Professor of Ophthalmology, The University of Texas Southwestern Medical School, Dallas

RONALD V. MAIER, M.D.
Associate Professor of Surgery, University of Washington School of Medicine; Director, Surgical Intensive Care Unit, Harborview Medical Center, Seattle

JOHN C. McCABE, M.D.
Associate Professor of Clinical Surgery, Cornell University Medical College, New York

ROBERT N. McCLELLAND, M.D.
Alvin Baldwin, Jr., Professor of Surgery, The University of Texas Southwestern Medical School, Dallas

ANNE MOORE, M.D.
Associate Professor of Clinical Medicine, Cornell University Medical College, New York

ANDREAS P. NIARCHOS, M.D.
Associate Professor of Medicine, Cornell University Medical College, New York

MALCOLM O. PERRY, M.D.
Professor and Chief, Division of Vascular Surgery, Cornell University Medical College, New York

PAUL C. PETERS, M.D.
Professor and Chairman, Division of Urology, The University of Texas Southwestern Medical School, Dallas

WILLIAM A. RUBENSTEIN, M.D.
Assistant Professor of Radiology, Cornell University Medical College, New York

G. TOM SHIRES, M.D.
Lewis Atterbury Stimson Professor, Chairman, Department of Surgery, Cornell University Medical College, New York; Surgeon-in-Chief, The New York Hospital-Cornell Medical Center

CLIFFORD C. SNYDER, M.D.
Professor and Chairman, Department of Surgery, University of Utah School of Medicine; Chief of Surgery, Veterans Administration Hospital, Salt Lake City

ERWIN R. THAL, M.D.
Associate Professor of Surgery, The University of Texas Southwestern Medical School, Dallas

JAMES C. THOMPSON, M.D.
Professor and Chairman, Department of Surgery, The University of Texas Medical Branch at Galveston

COURTNEY M. TOWNSEND, Jr., M.D.
The Robertson-Poth Professor of Surgery, The University of Texas Medical Branch at Galveston

ROGER W. YURT, M.D.
Associate Professor of Surgery, Cornell University Medical College, New York; Director, Trauma Center, The New York Hospital-Cornell Medical Center

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Contents

Preface

The problem of trauma in the United States is not adequately recognized. Trauma is the leading cause of death during the first three decades of life in this country. It ranks overall as the fourth leading cause of death in the United States, and if arteriosclerosis is considered as a single entity, trauma is the third leading cause of death. Fifty million injuries occur annually in the United States; over 10 million of them are disabling. More than 100,000 deaths occur each year from accidents. Automobile accidents alone kill more Americans each year than were lost during the entire Vietnam War. Unlike some serious diseases in the United States, the incidence of and mortality from injuries increase each year.

The first edition of *Care of the Trauma Patient* was the work of the department of surgery at the University of Texas Health Science Center at Dallas and Parkland Memorial Hospital. The department received the impetus to write the book from the constant daily management of large numbers of injured patients. The second edition of *Care of the Trauma Patient* included a number of additional authors who were nationally known for their specific areas of expertise in the field of trauma. The present third, retitled edition, *Principles of Trauma Care,* includes the contributions of still more authors who bring the subject up to date. In addition to the revised chapters, a new chapter has been added on computed tomography, which has revolutionized noninvasive diagnostic body imaging. It has replaced previously standard radiographic procedures and reduced the need for invasive imaging with angiography.

Principles of Trauma Care is a practical guide to the principles, pathological physiology, and *in-hospital* clinical care of the injured patient. In recent years increasing emphasis has been placed on prehospital care. A number of definitive works have been written on immediate rescue and transportation of the injured patient; intercommunication between fire department, police department, and ambulance service in emergency rooms; and different staffing patterns in emergency rooms. The efficiency of emergency rooms is of infinite importance in the initial management of injured patients. Also of paramount importance is the quality of care patients receive immediately after injury, which is the time when many patients' subsequent course is determined.

Research in the area of trauma injuries is badly needed in the United States. It has been estimated that accidents are costing our society over 18 billion dollars per year, yet the annual investment of money spent on trauma research is only 0.04 percent of that amount. Many questions about the care of injuries remain unanswered. I hope that *Principles of Trauma Care* will stimulate continued research in the area of injuries and accidents.

I am indebted to the fine staff of New York Hospital residents, who are constantly being trained in the management and care of traumatized patients in a level I trauma center. To the resident physician goes much of the credit for the successful healing of many injured patients.

G. Tom Shires, M.D.

Part One

General Principles

Chapter 1

Principles and Management of Hemorrhagic Shock

G. TOM SHIRES, M.D.

CLASSIFICATION AND CLINICAL AND PHYSIOLOGICAL MANIFESTATIONS OF SHOCK

Definition and Working Classification

The scope of modern medicine is increasing steadily. As understanding of physiological and biochemical derangements is broadened, so is the horizon of possibilities for the relief of illness. As more seriously ill patients are presented, shock is a symptom complex more frequently encountered by the physician.

Although shock has been recognized for over 100 years, a clear definition and dissection of this complex and devastating state has emerged only slowly. Many attempts have been made over the years to define adequately the entity known as *shock*. In 1872 the elder Gross defined shock as a "manifestation of the rude unhinging of the machinery of life" [1]. Although the accuracy of this definition is unquestioned, it is obviously far from precise. In 1942 Wiggers, on the basis of an exhaustive examination of available evidence at that time, offered the definition: "Shock is a syndrome resulting from a depression of many functions, but in which reduction of the effective circulating blood volume is of basic importance, and in which impairment of the circulation steadily progresses until it eventuates in a state of irreversible circulatory failure" [2]. A definition which Blalock offered in 1940 was: "Shock is a peripheral circulatory failure, resulting from a discrepancy in the size of the vascular bed and the volume of the intravascular fluid" [3].

A more modern definition has been devised by Simeone [4]. He stated that shock may be defined as a "clinical condition characterized by signs and symptoms which arise when the cardiac output is insufficient to fill the arterial tree with blood under sufficient pressure to provide organs and tissues with adequate blood flow."

Shock of all forms appears to be invariably related to inadequate tissue perfusion. The low flow state in vital organs seems to be the final common denominator in all forms of shock.

For purposes of a working clinical classification, the etiologic classification offered by Blalock in 1934 is still a useful and functional one [5]. Blalock suggested four categories:

1. Hematogenic (oligemia)
2. Neurogenic (caused primarily by nervous influences)
3. Vasogenic (initially decreased vascular resistance and increased vascular capacity)
4. Cardiogenic
   a. Failure of the heart as a pump
   b. Unclassified category (including diminished cardiac output from various causes)

It is now clear that shock invariably results from loss of function of one or more of four separate but interrelated functions. These are

1. The pump (heart)
2. The fluid which is pumped (blood volume)
3. Arteriolar resistance vessels
4. The capacity of the venous bed (capacitance vessels)

In the context of Blalock's etiologic classification, these functions may be correlated:

I. Cardiogenic shock. This implies failure of the heart as a pump and may be brought about by
   a. Primary myocardial dysfunction from
      (1) Myocardial infarction

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(2) Serious cardiac arrhythmias
(3) Myocardial depression from a variety of causes

b. Miscellaneous causes would include mechanical restriction of cardiac function or venous obstruction such as occurs in the mediastinum with
(1) Tension pneumothorax
(2) Vena caval obstruction
(3) Cardiac tamponade

II. Reduction is the fluid which may be pumped, the blood volume. This loss of volume may be in the form of loss of whole blood, plasma or extracellular fluid in the extravascular space, or a combination of these three.

III. Changes in resistance vessels may be brought about by specific disorders, which would include

a. Decrease in resistance
(1) Spinal anesthesia
(2) Neurogenic reflexes, as in acute pain
(3) Possibly the end stages of hypovolemic shock

b. Septic shock
(1) Change in peripheral arterial resistance
(2) Change in venous capacitance
(3) Peripheral arteriovenous shunting

Therapy of shock will obviously revolve around the etiologic type or combination of types of shock present in a given patient who has undergone trauma.

The signs and symptoms of hypovolemic shock, when they are well established, are classic and usually easy to recognize. Most of the signs of clinical shock are characteristic of low peripheral blood flow and are contributed to by the effects of excess adrenal-sympathetic activity. The signs and symptoms of shock in humans, according to the severity of the shock, were well described by Beecher et al., as summarized in Table 1-1.

On first inspection the patient in shock presents an anxious, tired expression, which early is that of restlessness and anxiety and later becomes a picture of apathy or exhaustion. Typically, the skin feels cool and is pale and mottled, and there is evidence of decreased capillary flow exhibited by easy blanching of the skin, particularly the nail beds.

There are varying discrepancies in the classic picture of shock. In neurogenic shock, particularly that in response to spinal anesthesia, the pulse rate is normal or, more often, decreased; the pulse pressure is wide, and the pulse feels strong rather than weak. The rapid pulse characteristic of early hemorrhagic or wound shock may be absent, even if the patient has lost blood rapidly. This is also true if the patient's position is supine or prone, in which case a rapid pulse may not appear until the patient is moved or elevated to a sitting position [6].

In observing a large number of patients in hemorrhagic hypovolemic shock, one sees remarkably varied but typical responses of the sensorium to the shock episode. Most young, healthy patients who sustain hemorrhagic shock, when seen early, will appear to be restless and anxious and actually give the appearance of great fear. Shortly after being seen by a physician and started on treatment, this restlessness frequently gives way to great apathy, and the patient will appear sleepy. When aroused, the patient may complain of weakness or of a chilly sensation, although he or she does not actually have a chill. If blood loss is unchecked, the patient's apathy and sleepiness will rapidly progress into coma. In treating a large number of accident victims,

Table 1-1. GRADING OF SHOCK

Degree of shock	*Blood pressure (approx.)*	*Pulse quality*	*Skin temperature*	*Skin color*	*Circulation in skin (response to pressure blanching)*	*Thirst*	*Mental state*
None	Normal	Normal	Normal	Normal	Normal	Normal	Clear and distressed
Slight	To 20% increase	Normal	Cool	Pale	Definite slowing	Normal	Clear and distressed
Moderate	Decreased 20–40%	Definite decrease in volume	Cool	Pale	Definite slowing	Definite	Clear and some apathy unless stimulated
Severe	Decreased 40% to nonrecordable	Weak to imperceptible	Cold	Ashen to cyanotic (mottling)	Very sluggish	Severe	Apathetic to comatose, little distress except thirst

SOURCE: HK Beecher, FA Simeone, CH Burnett, SL Shapiro, ER Sullivan, TB Mallory: The internal state of the severely wounded man on entry to the most forward hospital. *Surgery* 22:672, 1947.

it has been our experience that patients who have bled into frank coma from which they cannot be aroused, resulting simply from blood loss alone (unassociated with other injuries such as brain damage), have usually sustained lethal blood loss. This sign usually indicates rapid massive hemorrhage for which the compensations to shock are inadequate to maintain sufficient cerebral blood flow to sustain consciousness.

Another characteristic of the wounded person, described by many investigators, is thirst. Thirst seems to be a characteristic of the injured person and is found in most emergency room patients brought in acutely ill from trauma with or without shock. The studies carried out to elucidate the nature of the thirst are many and varied. Most of these patients have intense adrenal medullary stimulation from trauma, not necessarily accompanied by shock. Consequently, caution must be used in allowing water, since dangerous water intoxication may be induced by this intense stimulus to imbibe liquids in the face of altered renal function.

Another characteristic of the patient in hemorrhagic shock is the low peripheral venous pressure, which is manifested by empty peripheral veins on inspection. Indeed, the starting of a simple intravenous infusion in a patient in hemorrhagic shock can be quite difficult. Obviously there are exceptions, such as shock due to cardiac tamponade, in which there is restriction to inflow of blood to the right side of the heart. In this instance the peripheral veins, including the neck veins, will be distended.

Nausea and vomiting from hypovolemic shock are common. It is true that other causes should be sought, but shock alone may be first manifest in this manner.

Another classic finding in hemorrhagic hypovolemia is a fall in body "core" temperature. Whether this is due to a lowered metabolic rate or to lower perfusion in areas where body temperature is measured is debatable.

Physiological Changes

BLOOD PRESSURE Arterial blood pressure is normally maintained by the cardiac output and the peripheral vascular resistance. Thus, when the cardiac output is reduced because of loss of intravascular volume, the blood pressure may remain normal so long as the total peripheral vascular resistance can be increased to compensate for the reduction in cardiac output. The vascular resistance varies for different organs and in different parts of the same organ, depending on the local conditions that determine the state of vasoconstriction or vasodilation at the time of the loss of intravascular volume. An example of the differential increase in peripheral resistance with reduction in cardiac output is seen in the change in distributional total blood flow to organs such as the heart and the brain as opposed to that to most other organs which are not essential for immediate survival. In hemorrhagic shock the heart may receive 25 percent of the total cardiac output as opposed to the normal 5 to 8 percent. The great increase in peripheral resistance in such organs as the skin and the kidneys causes significant reduction in flow in these organs while providing a lifesaving diversion of the cardiac output to the brain and the heart.

Consequently, the blood pressure may not fall until the reduction in cardiac output or loss of blood volume is so great that the adaptive homeostatic mechanisms can no longer compensate for the reduced volume. As the deficit continues, however, there is a progressive hypotension.

PULSE RATE Characteristically, reduction of the volume in the vascular tree is associated with tachycardia. A fall in pressure within the great vessels results in excitation of the sympathicoadrenal division of the autonomic nervous system and, simultaneously, inhibition of the vagal-medullary center. Consequently, with hemorrhage or loss of circulating blood volume, the resulting fall in arterial blood pressure should cause an increase in heart rate.

However, this compensatory mechanism is variable in its effectiveness. Obviously, the degree of loss of intravascular volume, the amount of reduction in venous return, and other variables such as ventricular function may markedly influence the ability of Marey's phenomenon to compensate for the reduction in blood volume. Work with slow hemorrhage in normal, healthy volunteers by Shenkin et al. [6] has shown that, as long as the supine position is maintained, as much as 1000 ml of blood may be lost without significant increase in pulse rate. Similarly, the pacemaker system of the heart within the sinoatrial node is obviously influenced by other stimuli such as fear and anxiety that may also accompany the trauma producing the loss of intravascular volume.

Consequently, during the course of observation and treatment of shock, changes in pulse rate are

of value only when followed over an extended period. Change in pulse rate may indicate response to therapy once other external sources that may have changed cardiac rate are diminished or removed.

VASOCONSTRICTION Increase in peripheral vascular resistance by production of peripheral vasoconstriction rapidly becomes maximal in an effort to compensate for the reduced cardiac output. Vascular resistance can be measured only indirectly in humans and in animals. There is good evidence that early disproportionate reduction in vascular resistance in the heart occurs while there is still little change in vascular resistance in many organs. Subsequently, maximal vascoconstriction occurs in the skin, kidneys, liver, and, finally, in the brain [4].

Concomitantly, there is generalized constriction of the veins in response to reduction in intravascular volume. Venoconstriction would be a necessary homeostatic mechanism since over half of the total blood volume may be contained within the venous tree [4].

These vascular responses to hemorrhage are immediate and striking. Within seconds following the onset of hemorrhage there are unequivocal signs of sympathetic and adrenal activation. Serum catecholamine levels show prompt elevation indicative of action of the adrenal medullary function [7]. The adrenal cortical and pituitary hormones also show prompt increase in serum levels following shock. Many of the clinical signs associated with shock are simply signs of response of the sympathetic and adrenal medullary system to the insult sustained by the organism.

HEMODILUTION All the responses to reduction of intravascular volume eventually result in decrease in volume flow to tissues and initiation of compensatory mechanisms directed at correction of the low flow state. One such compensation is movement of fluid into the circulation, resulting in hemodilution. This fluid is commonly known as *extravascular extracellular fluid;* it has the composition of plasma, but a lower protein content.

It is now clear, however, that the hematocrit or hemoglobin concentration in shock is simply an index of the balance between the relative loss of whole blood or plasma and gain into the blood system of extravascular fluid. For example, in hemorrhagic hypovolemia there is generally progressive hemodilution, which increases with the severity of the shock state. Obviously, in this circumstance there has been a greater movement of fluid from the extravascular to the intravascular space with the progression of the shock. This is in contradistinction to shock associated with loss of intravascular volume primarily due to plasma loss. High hematocrit shock may occur with massive losses of plasma and extravascular extracellular fluid, such as is associated with peritonitis, burns, large areas of soft-tissue infection, and the crush syndrome.

The mechanism of hemodilution following hemorrhage is probably on the basis of the Starling hypothesis; i.e., the reduction in hydrostatic pressure in the capillaries because of hypotension and arterial and arteriolar vasoconstriction results in a shift of the pressure gradient to favor the passage of fluid from the tissue extracellular space into the intravascular capillary bed.

It is worthy to note that the studies of Carey et al. do not demonstrate a significant reduction in serum protein content in patients following hemorrhagic shock and resuscitation [8,9].

Biochemical Changes

The biochemically measurable changes that occur as response to the stress invoked by shock fall into three fairly well defined categories. These are (1) the changes invoked by the pituitary-adrenal response to stress, (2) those changes brought about by a net reduction in organ perfusion imposed by a low rate of blood flow, and (3) those changes brought about by failing function within specific organs.

PITUITARY-ADRENAL RESPONSE The immediate effects seen from sympathicoadrenal activity are those associated with high circulating epinephrine levels. Characteristically, these include eosinopenia and lymphocytopenia along with thrombocytopenia. This doubtless represents the laboratory reflection of increased circulating epinephrine that, in itself, can be and has been measured to be elevated, as an early response to shock. These changes are nonspecific and are found early in a patient with shock or severe trauma. These phenomena usually disappear rapidly. Other evidences of the pituitary and hormonal response to shock are seen in the well-known stress reaction or metabolic responses so well described by Moore [10]. These

include a striking negative nitrogen balance and retention of sodium and water, as well as a notable increase in the excretion of potassium. Alterations in blood volume and arterial pressure may result in increases in both adrenocorticotropic hormone (ACTH) and cortisol secretion. Reexpansion of blood volume and the result of restitution of arterial pressure lead to feedback inhibition of the secretion of ACTH and cortisol. In addition, pain, hypoxia, and hypothermia in the traumatized patient also stimulate increased pituitary-adrenal activity [11].

LOW FLOW STATE Those changes incident to the low rate of blood flow during shock are now being better understood. More evidence is accumulating to support the observation that, as a result of a decreased blood flow or low rate of perfusion, there is a reduction in oxygen delivered to the vital organs and, consequently, a mandatory change in metabolism from aerobic to anaerobic. In the switch from aerobic to anaerobic metabolism, energy made available by the oxidation of glucose is greatly reduced during shock. The most striking example of a shift in metabolism is the production of the end product lactic acid instead of the normal aerobic end product of carbon dioxide. This is reflected in a metabolic acidosis with a reduction in the carbon dioxide combining power of the blood. The available buffer base is progressively decreased by combining with the increased lactic acid, and the respiratory compensation that occurs early in the course of hemorrhagic shock is frequently inadequate. Consequently the progressive decline in pH toward a striking acidosis is thereby hastened. Indeed, in several studies the ability of animals as well as humans to recover from shock has been found to correlate rather closely with the degree of lactic acid production and the decrease in the alkali reserve and pH of the blood.

In some cases determination of blood pH may not accurately reflect changes in pH at the cellular level. After the induction of hemorrhagic shock in experimental animals, skeletal muscle surface pH changes precede those in blood, and minimal changes may be masked by the efficient blood buffer systems [12]. Lactate and excess lactate levels correlate well with the clinical impression of the depth of shock, but the injuries producing the shock state have a much greater bearing on ultimate prognosis [13].

Drucker pointed out that there is a consistent elevation of the blood sugar level in relation to the degree of blood loss and the severity of shock [14]. This was earlier observed in battle casualties studied in World War II and has since been thoroughly confirmed by Simeone and others. It is Drucker's belief that this represents an increase in hepatic glycolysis by the change from aerobic to anaerobic metabolism, while Egdahl believes that there is decreased insulin secretion and decreased peripheral utilization of glucose [15].

Elwyn [16] concludes that changes in glucose metabolism are the result of increased production of glucagon, cortisol, and epinephrine together.

Other evidences of failure of different parameters of cell metabolism have been presented by Thal [17], Schumer [18], Mela [19], and Baue [20].

ORGAN FAILURE The biochemical changes that appear incident to organ failure seem to be dependent in large part on the duration and severity of the shock. The changes in renal function induced by hypovolemia may vary from simple oliguria with a concentrated and acid urine to high output renal failure with a urine of low specific gravity and high pH, or frank anuric renal failure. Similarly, the blood nonprotein nitrogen content will depend on the degree of impairment in renal function. This may vary from slight to no retention of nitrogenous products to a steep and progressive rise that may require therapy.

Changes in ion concentration, including a rise of serum potassium, are dependent on many things, among them adrenal cortical response, the change in metabolism from aerobic to anaerobic with resultant release of potassium, and also specific changes within tissues invoked by the shock. If renal function is maintained, the rise inevitably seen in serum potassium early after the onset of shock is short-lived, in that the renal excretion of potassium is high during recovery from hemorrhagic shock. If renal function is impaired, the concentration of potassium and magnesium as well as creatinine can rise to high levels in the serum. Other organ systems are more resistant than the kidney to the period of hypoperfusion. Hypovolemic shock alone is not the cause of posttraumatic pulmonary dysfunction occasionally seen in the injured patient. Numerous studies have failed to demonstrate a primary pulmonary injury due to hypotension alone [21,22]. The increase in the frequent syndrome of multiple organ failure following resuscitation will be discussed later in this chapter, under Septic Shock.

RESPONSE OF THE EXTRACELLULAR FLUID

Experimental Studies

EARLY RESULTS Hypovolemic shock is the most common form seen clinically and is also the form that has been studied most intensively both clinically and in the laboratory. Most of our own studies have been carried out using hypovolemic shock produced by external blood loss as the model. A method has been developed which allows the simultaneous measurement of total body red cell mass with the use of ^{51}Cr-tagged red blood cells, and total body plasma volume with the use of ^{131}I- and, later, ^{125}I-tagged human serum albumin. In addition, total body extracellular fluid can be measured simultaneously with the use of ^{35}S-tagged sodium sulfate [23]. These three isotopes are simultaneously injected intravenously, and by the use of appropriate energy-differentiating counting instruments, all three isotopes can be determined after equilibration. Volumes are then determined by the dilution principle using multiple sampling.

In an early study the three spaces were measured; splenectomized dogs were then bled a sublethal, subshock amount of 10 percent of the measured blood volume. After hemorrhage the three spaces were again measured. The measured loss of red cells and plasma, removed during the hemorrhage, could be detected by the method used. It was shown that the decrease in extracellular fluid volume was only what was lost as plasma removed during the hemorrhage [24].

By use of the same model, spaces were measured before and after hemorrhage of 25 percent of the measured blood volume. This hemorrhage was again sublethal, but it did produce hypotension. In this group of animals the loss of red cells and plasma could be measured by the method. In addition, however, the functional extracellular fluid volume as measured by the early ^{35}S-tagged sodium sulfate space decreased by 18 to 26 percent of the original volume. Since there was no measurable external loss of ^{35}S sulfate, this reduction was presumed to be an internal redistribution of extracellular fluid. Subsequent studies of external bleeding of 35 percent, 45 percent, and even above 50 percent hemorrhage always produced the same reduction in functional extracellular fluid, as long as the animal was in shock.

In subsequent studies splenectomized dogs were subjected to "irreversible" hemorrhagic shock according to a modified method of Wiggers, using a reservoir [25]. Return of shed blood in this severe preparation resulted in the return of blood pressure to near control levels followed by a fall in blood pressure within 1 to 16 h, with death in 80 percent of the dogs, a standard mortality rate.

In one group of animals the three volumes were measured; the dogs were then subjected to shock by the Wiggers method. The three spaces were remeasured by reinjection during the period of shock; then shed blood was returned. The decrease in blood volume was that which had been removed. Concurrently, the functional extracellular fluid exhibited a decided reduction. Immediately after the return of shed blood, the red cell mass returned to essentially normal levels, as did the plasma volume; however, there remained a deficit of functional extracellular fluid. In dogs treated with shed blood plus plasma (10 ml/kg), the losses during shock were again similar. After therapy with plasma, plus return of shed blood, there was a return of blood volume to normal. There remained, however, a decrease in functional extracellular fluid volume.

Dogs treated with an extracellular "mimic," such as a balanced salt solution plus shed blood, had comparable losses during shock. As in the previous groups, the blood volume returned essentially to normal after treatment. Dogs treated with salt solution plus shed blood exhibited return of functional extracellular fluid volume to control levels.

In this study only 20 percent of those treated with shed blood alone survived longer than 24 h. When plasma was used in addition to whole blood as therapy, 30 percent of dogs so treated survived. Of the animals treated with lactated Ringer's solution plus shed blood, 70 percent survived (Figure 1-1). The 80 percent mortality rate of a standard "irreversible" shock preparation was reduced to 30 percent by restoration of functional extracellular fluid volume in addition to return of shed blood.

All these early studies on the measurement of the functional extracellular fluid were based on volume distribution curves of sulfate measured up to approximately 1 h. At any point in the course of the shock volume distribution curve, there will be a reduction in extracellular fluid in the untreated state of shock.

Subsequent work has followed these volume distribution curves out for many hours [23]. In true untreated hemorrhagic shock there is a reduction in the total extracellular fluid, or final diluted volume

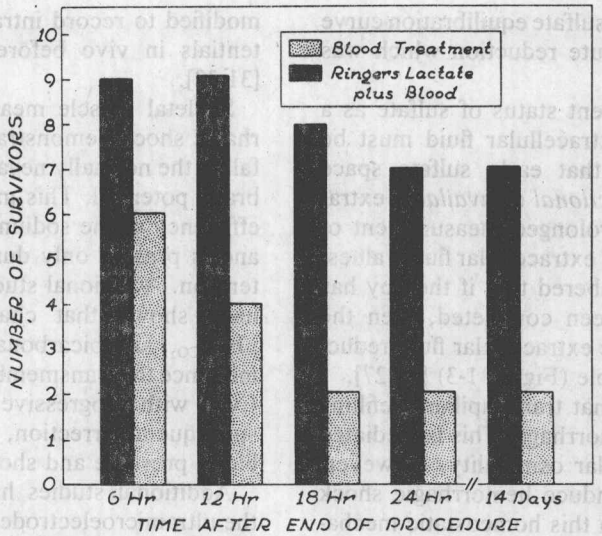
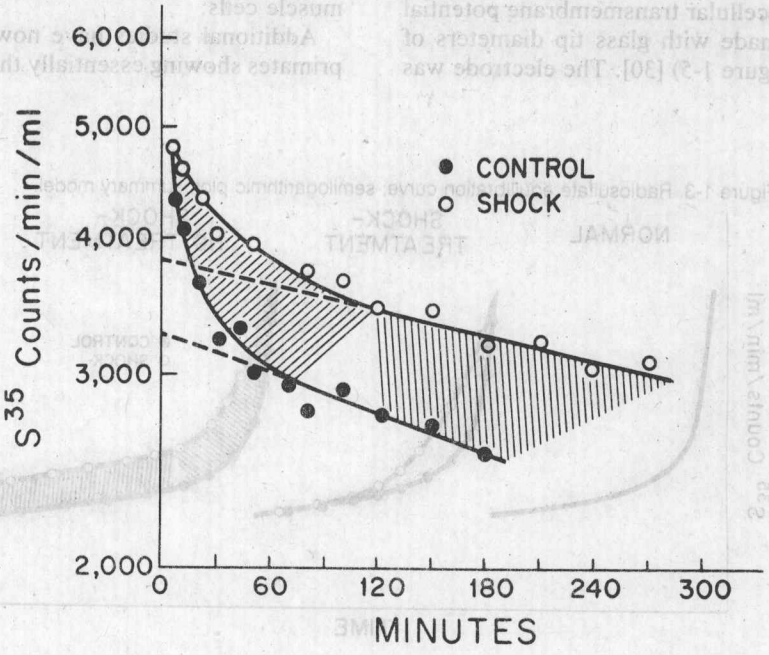


Figure 1-1. Acute hemorrhagic shock, survival study.

of radiosulfate, when compared with preshock volumes (Figure 1-2).

Even when a less severe shock preparation is used, there will still be a reduction in early equilibrating extracellular fluid, or early available extracellular fluid, whereas the total anatomic extracellular fluid may remain normal. Subsequent studies have shown that if shock is not of sufficient duration to produce reduction in both functional and total extracellular fluid, then the reduction may be only in functional extracellular fluid. Furthermore, if therapy is instituted quickly and blood pressure is

Figure 1-2. Shock, reinjection (splenectomized).



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