Chapter 4

HEMORRHAGE, SHOCK, AND FLUID RESUSCITATION

FREDERICK W. BURGESS, PH.D, M.D.[*]; MICHAEL J. SBOROV, M.D.[†]; AND DEAN R. CALCAGNI, M.D.[‡]

[*]*Formerly, Major, Medical Corps, U.S. Army Reserve; Director, Anesthesia Research, Madigan Army Medical Center, Tacoma, Washington 98431-5000; currently, Department of Anesthesia, Rhode Island Hospital, 593 Eddy Street, Providence, Rhode Island 02903*

[†]*Lieutenant Colonel, Medical Corps, U.S. Army Reserve; Chief, Anesthesia and Operative Services, Madigan Army Medical Center, Tacoma, Washington 98431-5000*

[‡]*Lieutenant Colonel, Medical Corps, U.S. Army; Staff Officer, Combat Casualty Care, U.S. Army Medical Research and Materiel Command, Fort Detrick, Frederick, Maryland 21702-5000*

INTRODUCTION

Victims of combat-related trauma may present with the classical manifestations of hemorrhagic shock following rapid exsanguination from a major vascular structure. Hemorrhagic shock is the most common cause of death among combat casualties. Early recognition, control of the hemorrhage source by effective first aid, and rapid volume restoration with intravenous fluids are the ideal preparation for definitive surgical repair of the injury. Unfortunately, most combat casualties manifesting evidence of hypovolemia may not be rapidly transported, may not have an obvious bleeding source, may suffer from sleep deprivation, may have occult injuries, and, depending on the climatic conditions, may suffer from hyperthermia, hypothermia, and dehydration. An understanding of the combat environment, the climate, infectious epidemics, and the time of injury are all important factors contributing to the delineation of an appropriate diagnosis and treatment plan. This chapter focuses on the recognition and *initial* treatment of hypovolemic shock in the combat casualty, with an emphasis on associated medical conditions that may accompany and confound the diagnosis and treatment of hypovolemia.

PHYSIOLOGICAL CONSEQUENCES OF HEMORRHAGE

The adverse effects of hemorrhage on young and healthy soldiers are directly related to two primary factors: (1) decreased intravascular volume and (2) inadequate oxygen-carrying capacity. Reductions in intravascular volume and the associated organ hypoperfusion initiate a variety of autonomic and neurohumoral homeostatic responses, which are designed to maintain or restore the circulating plasma volume and organ perfusion. The consequences of inadequate oxygen delivery are organ dysfunction and possible death.

Cardiovascular Response

The response to rapid hemorrhage is complex and time dependent. The sudden fall in venous return to the heart results in an immediate decrease in cardiac stroke volume. Because the aortic arch is less distended by blood, intraluminal pressure will fall. The immediate response to the fall in pressure is a diffuse activation of the sympathetic nervous system caused by reflexes that arise in the aortic and carotid baroreceptors. An additional component of this adrenergic response is caused by the release of catecholamines from the adrenal medulla. The initial physiological effects are (*a*) increased heart rate and myocardial contractility; (*b*) increased systemic vascular resistance, which is due primarily to vasoconstriction of arterioles in the splanchnic viscera and skeletal muscle; and (*c*) decreased vascular capacitance, which arises primarily from constriction of the smaller veins and venules. These responses tend to preserve perfusion of the critically important central organs such as the heart and brain at the expense of the more peripherally situated splanchnic viscera and skeletal muscles.

Augmentation of the immediate vasoconstrictor response to hemorrhage occurs within minutes, with the activation of the renin-angiotensin system (Table 4-1). The formation of angiotensin II, an extremely potent generalized vasoconstrictor, contributes to the restoration of the arterial blood pressure and stimulates sodium conservation via the release of aldosterone. Sodium conservation, together with water conservation mediated via antidiuretic hormone secretion, also tends to restore the missing blood volume. The major mechanism that reestablishes an effective circulating blood volume in the first hours following a major hemorrhage is transcapillary transfer of plasma water from the intracellular fluid space to the intravascular fluid space. Although this process begins within minutes, it requires many hours for completion.

If the hemorrhage continues or if its magnitude overwhelms these compensatory mechanisms, a vicious cycle may begin, arising from the profoundly ischemic splanchnic viscera and skeletal muscle (Figure 4-1). Ultimately, systemic hemostasis fails for the following reasons:

- the ability of ischemic cells to maintain a normal transmembrane ionic gradient is lost,
- precapillary sphincters in the ischemic vascular beds relax,
- intravascular fluid leaves the circulation and collects in the ischemic tissues,
- lactic and inorganic acids as well as possible myocardial depressants enter the circulation, and
- oxygen-derived free radicals are generated—especially at the beginning of resuscitation when the ischemic beds are

TABLE 4-1

COMPENSATORY RESPONSE TO HEMORRHAGIC SHOCK

Response Times	Systemic Response
Few seconds	*Sympathetic nervous system*
	Vasoconstriction
	Tachycardia
Few minutes	*Renin-angiotensin system*
	Vasoconstriction (Angiotensin II)
	Sodium conservation
	Aldosterone secretion
	Glucocorticoid secretion
	Water conservation
	Vasopressin secretion (ADH)
Several minutes	*Fluid compartment shifts*
	Transcapillary refill
	Thirst

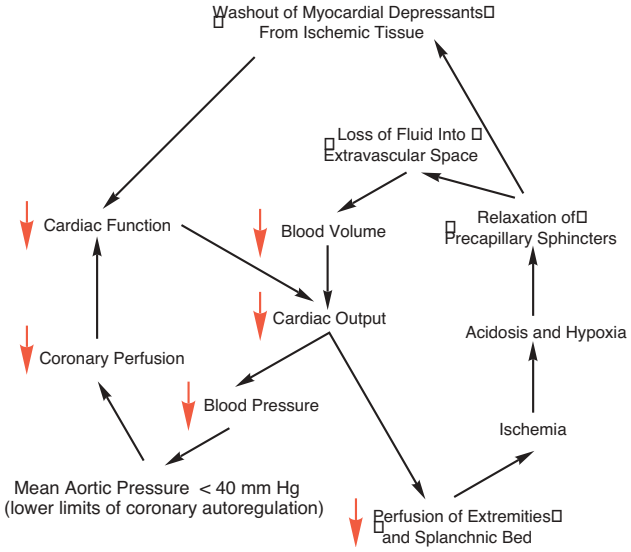

Fig. 4-1. *Positive* feedback loops (a departure from the normal steady state, which induces an ever-increasing departure) that may possibly develop and lead to death following massive hemorrhage can involve either the heart or the peripheral vascular beds of the soft tissues of the extremities and the splanchnic organs. The loops are not independent because the magnitude of cardiac output is a component of both. The central role of cardiac output means that a decline in the heart's pumping action (if of sufficient duration and magnitude) can, by reducing peripheral perfusion, adversely affect myocardial function. This causes a further decline in the heart's pumping action. The importance of myocardial depressants in hemorrhagic shock, in contrast to septic shock, remains controversial. Adapted with permission from Bellamy RF, Pedersen DC, DeGuzman LR. Organ blood flow and the cause of death following massive hemorrhage. *Circ Shock*. 1984;14:115.

reperfused—and add to the cellular damage already caused by ischemia.

As the peripheral component of systemic hemostasis fails, the centrally circulating blood volume falls, leading to cardiac and respiratory dysfunction and further reduction in cardiac output and blood pressure. Respiratory compensation for the increasing metabolic acid load fails as the capacity of respiratory muscles is impaired by hypoperfusion. Acidosis, in conjunction with declining coronary perfusion pressure, impairs cardiac function; this leads to a further fall in cardiac output and blood pressure. Even vigorous resuscitation instituted at this time may not prevent death.

Fluid Compartment Shifts

After an untreated casualty sustains a major hemorrhage, restoration of the intravascular fluid compartment may require many hours as interstitial fluid is drawn into the intravascular compartment through alterations in the normal balance between the transcapillary hydrostatic and oncotic pressures. This phenomenon, *transcapillary refill*, depends on both adequate interstitial fluid reserves and the integrity of the capillary bed. In the average adult male, total body water accounts for approximately 60% of total body weight.[1] Two thirds of body water is distributed within the intracellular compartment. The remaining one third is located in the extracellular fluid compartment; 25% to 30% of this compartment comprises the intravascular plasma volume (approximately 5% of the total body water), with the remainder making up the interstitial fluid space. Water freely distributes between each compartment by passive diffusion; however, ionic equilibrium is primarily determined by selective permeability, or ion-selective active transport, or both, within each compartment. Water, ions, and, to a variable extent, proteins, are freely exchanged between the intravascular and much of the interstitial fluid compartments. The transmural exchange of fluid between the intravascular compartment and the interstitium can be described by Starling's equation:

$$\text{fluid flux} = K[(P_c - P_i) - s\,(\pi_c - \pi_i)]$$

where K represents the capillary filtration coefficient; P_c and P_i represent the hydrostatic pressure of the intravascular (capillary) and interstitial compartments, respectively; s, which is known as the reflection coefficient, represents the impermeability of the membrane to a given substance; and π_c and π_i represent the oncotic pressure of the intravascular and interstitial fluid compartments, respectively.[2] (See Chapter 25, Acute Respiratory Failure and Ventilatory Management, for further discussion of Starling's equation.)

In hemorrhagic shock, constriction of the precapillary sphincter should decrease the net flow of fluid out of the intravascular space (decreased P_c) and promote the return of interstitial fluid into the intravascular compartment (transcapillary refill). Conversely, the loss of precapillary sphincter tone in late shock is an important factor leading to decompensation and death.

Extracellular Fluid Loss

Combat casualties presenting for care several hours after being injured may suffer from (*a*) hemorrhage-induced intravascular volume depletion and (*b*) preexisting depletion of the extracellular fluid compartment that is caused by concomitant dehydration secondary to environmental or nutritional factors. The importance of restoring the interstitial fluid compartment, in addition to restoring the circulating blood volume, was demonstrated in classic experiments performed during the 1950s.[3] Using a dog hemorrhagic shock model, the researchers found improved survival among the animals that were resuscitated with both lactated Ringer's solution and their shed blood, as opposed to resuscitation with shed blood alone or shed blood and additional plasma. Because the crystalloid fluid is distributed through the body water, it was soon afterwards determined that restoration of the intravascular volume required the infusion of volumes 3- to 4-fold greater than the putative missing volume; that is, after 1 L of lactated Ringer's solution is infused, only 250 mL will remain in the intravascular space.

Trauma and severe hemorrhage frequently lead to a reduction in the functional extracellular fluid compartment. The reduction in extracellular fluid is partly attributable to hemorrhage; transcapillary refill; and the sequestration of interstitial (isotonic) fluid, which represents the third-space loss. Recommendations for replacing the third-space fluid sequestration include administering (*a*) a balanced salt solution at 4, 6, and 8 mL/kg/h for minimal, moderate, and severe trauma, in addition to (*b*) the estimated hourly maintenance fluids.[4]

Oxygen Transport

Hemorrhage interferes with normal tissue oxygenation by two mechanisms: (1) the anemic (ie, inadequate oxygen-carrying capacity) and (2) the hemodynamic (ie, inadequate tissue perfusion). Anemia is rarely appreciated immediately after a hemorrhage. This may be due to inadequate time for equilibration or inadequate interstitial fluid reserves. However, even in the setting of severe hemorrhage, reduced hemoglobin content of the blood is rarely the cause of tissue hypoxia.[5] For example, a healthy, 20-year-old, male soldier (with body surface area of 1.73 m^2) arrives at a combat support hospital within 1 hour of sustaining an extremity wound. Medical personnel there estimate that he has experienced a 40% to 50% hemorrhage from the femoral artery. Fortunately, the bleeding had been controlled at an aid station and he had been resuscitated with 6% hetastarch and lactated Ringer's solution. On arrival at the combat support hospital, his vital signs are stable and the bleeding has stopped. His hemoglobin is determined to be 7 g/dL. Assuming a Pa_{O_2} (partial pressure of oxygen in the arteries) of 95 mm Hg, a hemoglobin saturation of 99%, and a normal cardiac output (C.O.) of 5 L/min, the arterial oxygen content (Ca_{O_2}) and oxygen delivery (D_{O_2}) of this hypothetical casualty can easily be calculated:

$$Ca_{O_2} = 1.36 \bullet (\text{hemoglobin concentration}) \bullet (\text{hemoglobin saturation}) + 0.003\,(Pa_{O_2})$$

$$Ca_{O_2} = 1.36 \bullet (7\ \text{g/dL}) \bullet (.99) + 0.003 \bullet (95) = 9.71\ \text{mL}\ O_2/\text{dL}$$

$$\text{C.O.} \bullet Ca_{O_2} = D_{O_2}$$

$$5\ \text{L/min} \bullet 9.71\ \text{mL/dL} = 486\ \text{mL}\ O_2/\text{min}$$

Assuming a normal C.O. of 5 L/min, with a Ca_{O_2} of 9.71 mL/dL, this hypothetical patient can deliver 486 mL of O_2/min or 281 mL/m^2. This value exceeds the predicted basal O_2 consumption rate of 250 mL/min. Under stress conditions, the basal oxygen requirement may increase 2- to 3-fold. However, the cardiac output of most young, healthy soldiers can increase 2- to 3-fold to keep up with increased metabolic demand, provided that an adequate cardiac preload has been maintained.

This example demonstrates that (*a*) blood replacement is frequently unnecessary and (*b*) vol-

ume restoration with asanguinous plasma expanders is, at least initially, the key element. If the circulating plasma volume is maintained, then the metabolic consequences of severe hemorrhage can be minimized. The critical point to be remembered in this scenario is that the soldier's hemorrhage was not ongoing but had been stopped by appropriate first aid. However, in the setting of unabated hemorrhage and shock (as indicated by hypotension, tachycardia, mental-status changes, and diminished or absent urinary output), the administration of blood is essential and should not be withheld.

When combat casualties who are in shock arrive at a medical treatment facility with surgical capabilities, medical officers should not underestimate the amount of fluid required to reestablish a circulating blood volume compatible with survival. For example, during the Vietnam War, soldiers who required a blood transfusion received, on average, 2.5 L of blood and 2 L of crystalloid fluid during their initial resuscitation and surgery.[6] Even combat casualties who are no longer bleeding—who have normal hemodynamic indices and have received what should be adequate restoration of red blood cell mass on the basis of estimated blood loss—frequently have a deficient red blood cell mass when this is measured several days after the resuscitative surgery.[7]

HYPOVOLEMIA

Vascular and visceral injuries secondary to missile wounds, fractures of the long bones, and crush injuries are frequently accompanied by visible and occult hemorrhage. The amount of blood lost will not always be evident, as blood loss tends to be overestimated with more-visible, superficial injuries and underestimated with abdominal and orthopedic injuries (see Chapter 20, Abdominal Injuries, and Chapter 21, Extremity Injuries). Intervention in the field and mobilization of extracellular fluid stores (transcapillary refill) may serve to confuse the initial estimation of blood loss and result in an underestimation of blood loss. The classic parameters for the estimating the severity of hemorrhage are often a useful starting point for determining the rapidity and types of fluid administered, and for determining the response to resuscitation (Table 4-2). Supine hypotension in all combat casualties should be recognized as hypovolemia that requires rapid and immediate fluid resuscitation.

Trauma victims presenting with a suspected mild hemorrhage (Class I: suspected blood loss in the absence of tachycardia or hypotension) require initial resuscitation with clear solutions, preferably intravenous crystalloids. Resuscitation for moderate hemorrhage (Class II: tachycardia without hypotension) should be initiated with crystalloid or colloid solutions or both; however, in the presence of continued bleeding, early transfusion therapy with packed red blood cells or whole blood should be initiated. The presence of hypotension (Class III or Class IV hemorrhage: tachycardia and hypotension) requires immediate blood-volume replacement with (*a*) packed red blood cells or whole blood and (*b*) crystalloid solutions. Other causes of

TABLE 4-2

ESTIMATION OF HEMORRHAGE

Degree of Hemorrhage	Blood Loss (L)	Blood Vol. Lost (%)	Heart Rate	Blood Pressure	Pulse Pressure	Respiratory Rate	Urinary Flow (mL/h)	Mental Status
Class I	< 1	< 15%	< 100	Normal	Normal or increased	14–20	≥ 30	Slight anxiety
Class II	0.75–1.5	15%–30%	> 100	Normal	Decreased	20–30	20–30	Mild anxiety
Class III	1.5–2.0	30%–40%	> 120	Decreased	Decreased	30–40	< 15	Anxious and confused
Class IV	> 2.0	≥ 40%	≥ 140	Decreased	Decreased	Rapid and shallow	Negligible	Confused and lethargic

Adapted with permission from Committee on Trauma, American College of Surgeons. *Advanced Trauma Life Support Program for Physicians*. Chicago, Ill: American College of Surgeons; 1989: 72.

hypotension (eg, tension pneumothorax, cardiac tamponade) should be sought after fluid resuscitation has begun.

In the combat setting, tachycardia is a useful, but often an insensitive, indicator of hypovolemia. Just how insensitive was one of the more interesting findings of the surgical research unit that worked during World War II. Data on the effects of shock on heart rate were published in the seminal volume, *The Physiologic Effects of Wounds,* published as part of The Surgeon General's official history of the U.S. Army Medical Department in World War II (Table 4-3).[8] The degree of shock in injured soldiers was stratified not only by blood pressure but also by measurement of blood volume. The table demonstrates that the differences between the heart rates in soldiers with varying degrees of shock are so small as to be useless for predictive purposes.

Subsequent studies on combat casualties have confirmed that there may be no simple relation between magnitude of hemorrhage and heart rate.[9] Although the generalized discharge of the sympathetic component of the autonomic nervous system following a precipitous fall in blood pressure leads to tachycardia, the decrease in venous return to the heart by unloading mechanoreceptors in the cardiac chambers, and especially the left ventricle, leads to reflex slowing of the heart through an efferent pathway that involves the parasympathetic nervous system. Thus, hemorrhage activates two different components of the autonomic nervous system, and although tachycardia due to the sympathetic component usually predominates, vagal slowing may also be seen.

In addition, other factors may confound any relation between the observed heart rate and the magnitude of blood loss. For example, atropine administered in the field for chemical exposure, fear, and pain makes the use of heart rate unreliable. Trained athletes and infantry soldiers may tolerate a substantial hemorrhage without manifesting tachycardia or hypotension until they are severely hypovolemic. When possible, performance of a tilt-test will help to distinguish hypovolemia from other causes of tachycardia.

It is important to recognize that heart rate and blood pressure may normalize with initial resuscitation efforts, but this may not be indicative of normal peripheral perfusion. Mixed venous blood oxygen saturation may be the best single index for assessing the adequacy of systemic perfusion.[10] However, this determination requires a blood gas analyzer, a device not usually available in forward surgical hospitals (this subject is discussed more fully in Chapter 5, Physiological Monitoring). Furthermore, a central venous line is required. Unfortunately, invasive arterial and central venous monitoring, although often desirable for fluid management, may not be easily managed in the combat theater.

Traditionally, restoration of urinary output has been used as a monitor of organ perfusion and as a guide to fluid therapy. An indwelling urinary bladder catheter should be considered an essential monitor in all trauma victims. There may be no change in hematocrit immediately following hemorrhage, because only when transcapillary fluid refill occurs (or when fluid resuscitation is initiated) does dilu-

TABLE 4-3

RELATIONSHIP OF DEGREE OF SHOCK TO HEART RATE (106 CASES)

	Degree of Shock			
	None (n=13)	Slight (n=24)	Moderate (n=34)	Severe (n=35)
Reduction of Blood Volume (%)	14.1 ± 4.9	20.7 ± 4.1	34.3 ± 3.1	45.9 ± 4.6
Blood Pressure (m ± SD)				
Systole	126 ± 11.9	109 ± 3.0	95 ± 4.9	49 ± 7.6
Diastole	75 ± 1.5	66 ± 2.7	58 ± 3.5	25 ± 5.8
Heart Rate (m ± SD)	103 ± 7.2	111 ± 3.4	113 ± 3.6	116 ± 3.3
(Range)	(70–140)	(88–150)	(80–160)	(60–144)

Data source: The Board for the Study of the Severely Wounded, North African–Mediterranean Theater of Operations. *The Physiologic Effects of Wounds*. In: Beecher, HK, ed. *Surgery in World War II*. Washington, DC: US Army Medical Department, Office of The Surgeon General; 1952: 34, 35, 56.

tion of the remaining red blood cell mass occur. Thus, acute changes in hematocrit and hemoglobin are not necessarily useful in evaluating ongoing blood loss.

Venous Access

The initial management of all combat trauma casualties must involve a team effort focusing on combined assessment and intervention as indicated by the immediacy of the situation. Casualties presenting with evidence of shock must quickly be inspected for evidence of external hemorrhage. If a site of major bleeding is found, appropriate first-aid maneuvers designed to stop the bleeding (eg, direct pressure) must be instituted immediately. Two or more large-bore (preferably 14-gauge) intravenous fluid cannulae must be established early in all casualties in shock, or those suspected of having injuries that might lead to shock. Venous access can be extremely difficult to obtain in the presence of hypovolemia and dehydration. Additionally, access sites may be limited by the location and nature of the traumatic injuries. Optimal sites for venous cannulation in trauma victims include the antecubital, subclavian, saphenous, and femoral veins.

Surgical access (cutdown) may be required in patients with severe hemorrhagic shock. The saphenous and antecubital vessels are convenient locations for cutdown cannulation. This approach allows the sterile intravenous extension tubing to be inserted directly into the vessel to maximize delivery. A major disadvantage of the surgical approach is the risk of infection.

Central venous access may be obtained via the internal and external jugular veins, the subclavian vein, and the femoral veins. Femoral access eliminates the risk of a pneumothorax (a recognized complication of gaining venous access via the neck and chest); however, the femoral site is remote from the anesthesiologist during most surgical procedures. Also, penetrating intraabdominal injuries may involve the inferior vena cava. In such cases, resuscitation via the femoral vessels may contribute to intraabdominal bleeding or be rendered ineffective by cross-clamping of the femoral vessel. The subclavian vein is a useful site for trauma access; however, this location carries a significant risk of a pneumothorax. Both the subclavian and internal jugular sites carry a significant risk of unintentional arterial injury or cannulation.[11] Major blood loss has occurred following arterial injuries at both sites, and the potential exists for an expanding hematoma in the neck to compromise the airway. Techniques for obtaining peripheral, central, and cutdown venous access are well taught in the American College of Surgeons' Advanced Trauma Life Support course, which should be taken by all medical officers.[12]

Intravenous Fluid-Delivery Systems

Some consideration must be given to ensuring the availability of large-bore, fluid-administration sets. The ideal set contains a large-surface-area combination drip chamber and blood filter (170 μ), dual vessel-access ports, and a hand-operated pumping chamber.

Large-caliber, high-efficiency, blood-warming units are essential for large-volume resuscitation. The Level 1 infusion system (manufactured by Level 1 Technologies, Inc., Rockland, Mass.) is a functional unit that provides rapid warming of intravenous infusions, through a counter-current warming system, at high rates of flow (500 mL/min) (Figure 4-2). This system allows quick and effortless priming, and has an in-line air vent to prevent accidental air emboli when used in conjunction with a pres-

Fig. 4-2. The Level 1 blood warming system is manufactured by Level 1 Technologies, Inc., Rockland, Mass.

sure infuser. These devices are being evaluated by the U.S. Army Medical Department for use in its field hospitals. The space required for the warming unit and the disposable components, and the expense of the disposable components are disadvantages of the Level 1 fluid warmer.

Alternative methods for rapid warming of crystalloid solutions include using microwave ovens or storing the solutions in warming ovens. The use of microwave ovens for blood components is presently not recommended. Under no circumstances should the fluid be infused at a temperature exceeding 40°C.

Pressure-assist, fluid-administration devices (pressure bags) are helpful in speeding fluid delivery. Most of these are manually operated bladder devices. Pneumatic infusers are currently marketed; however, under field conditions, the availability of compressed gas may limit their use. The Biomed Spring Activated Infusion Pressor (manufactured by Migada, Inc., Burbank, Calif.) offers a useful mechanical alternative. This spring-operated system provides a constant pressure and thus a more stable rate of delivery. Complications secondary to pressure-assisted fluid administration can occur (eg, venous air emboli, which may occur with compression of the residual air in an exhausted intravenous fluid bag). Air traps in blood-warming sets and air vents (eg, Level 1 system) can help to minimize this risk. Also, inverting the container and venting the air prior to administration will also reduce this risk. Some concern has been raised over the potential for damage to cellular elements with pressurized delivery systems, but the significance of this concern remains to be clarified.

Blood-Salvage Techniques

Salvaging blood for intraoperative autotransfusion is becoming an increasingly important part of routine anesthetic and surgical practice. Much of the recent emphasis on blood salvage has arisen over concerns about the safety of community autologous blood supplies, predominantly relating to the transmission of the human immunodeficiency virus. Although blood-salvage techniques can reduce the need for homologous transfusion, blood salvage usually does not completely eliminate the need for transfusion of homologous blood products in major trauma. Currently, there are two principal methods for salvaging blood from the operative field. The simplest method involves collecting the blood into sterile vacuum containers containing an anticoagulant (citrate-phosphate-dextrose or heparin) and reinfusing the unwashed filtered blood directly into the patient. The advantages of this technique include low cost, simplicity, and minimal requirements for storage or trained personnel. The disadvantages are that procoagulants, bacteria, and free hemoglobin will also be infused into the patient.[13] This approach appears to be ideally suited for use in field hospitals; unfortunately, the risks and benefits involved with transfusing unwashed blood have not yet been well delineated.

The alternative approach to blood salvage involves the collection of shed blood and processing it through a cell-washing device prior to reinfusion. These cell-washing devices, such as the Cell Saver 4 (manufactured by Haemonetics, Boston, Mass.) have become increasingly efficient and automated (Figure 4-3). The drawbacks include the need for personnel training, equipment expense, the need for expensive disposable plastic ware, and a time delay during processing. Furthermore, the benefits of cell washing, as opposed to the reinfusion of unwashed blood, have not been clearly demonstrated to improve patient outcome. With both techniques, coagulation factors and platelets are lost, and there is a risk of trauma to the cellular elements. Although blood salvage is clearly beneficial in the manage-

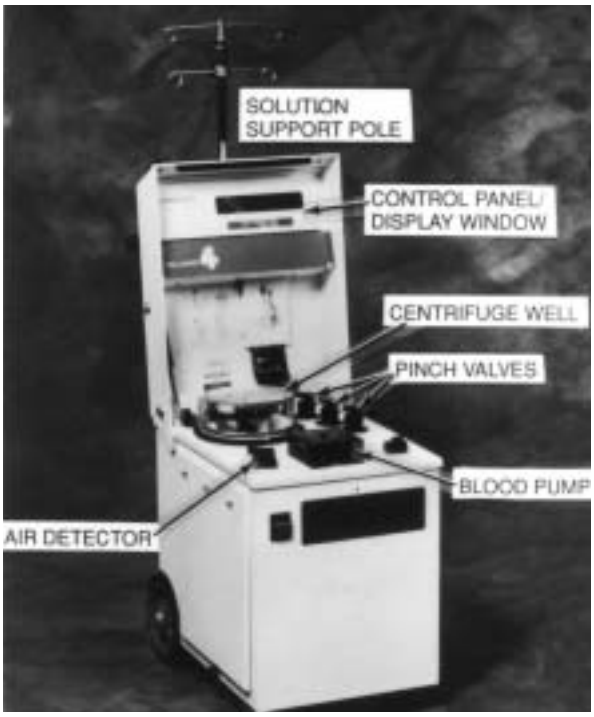

Fig. 4-3. The Cell Saver 4 is manufactured by Haemonetics, Boston, Mass.

ment of elective surgery, the advantages of introducing this methodology into the care of the combat casualty have yet to be demonstrated.

Intraosseous Fluid Infusion

Intraosseous fluid infusion has been advocated in situations in which venous access is difficult or impossible to obtain.[14] This route of delivery is particularly amenable in the pediatric population, and because pediatric casualties always occur in urban warfare, military anesthesiologists need to be familiar with the technique. Intraosseous access in adults is somewhat more difficult to obtain. This is partly related to the replacement of the marrow space by fat and partly related to the density of the overlying bone. Recent experimental animal studies have demonstrated the benefits of intraosseous infusion of a small-volume mixture of hypertonic saline and dextran via the sternum. A sternal infusion device has been developed that could be placed in the field with minimal training of medics, and then utilized for fluid access.[15] This approach is still in the early phase of development and testing.

At present, the intraosseous approach in adults is impractical and inadequately tested. Special needles are required for this technique (bone marrow sets) and the clinical utility in adults is unknown. Typically, the marrow space is entered by inserting a 15- or 18-gauge iliac bone marrow needle into the medial aspect of the proximal tibia. The distal femur, in the midline above the condyles, is an alternative site. The medial malleolus has been used in patients in cardiac arrest, in whom venous access is difficult. This approach may prove useful for the administration of selected drugs. Unfortunately, the distal tibia provides inadequate flow rates for it to prove useful in the management of hypovolemia. Complications with intraosseous infusions are uncommon.[16] The most common complication is that of local extravasation. Local cellulitis and osteomyelitis are fortunately rare (< 1%). Deaths have occurred with the sternal approach secondary to unrecognized penetration of the thoracic cavity.[17]

RESUSCITATION FLUID SELECTION

Hypovolemia and hemorrhagic shock fall along a continuum from mild to profound organ hypoperfusion. As cardiac output falls, oxygen transport to less crucial end-organ cellular elements (eg, skin, muscle) may become progressively inadequate, contributing to anaerobic metabolism and the accumulation of organic acids (eg, lactate). Homeostatic efforts (vasoconstriction) to preserve perfusion to the heart and brain inevitably sacrifice perfusion to the kidneys, muscle, intestine, and skin, leading to the classic manifestations of shock. The eventual lethality of the shock state probably results from the maintenance of this restricted perfusion pattern and progressive tissue hypoxia and acidosis.[18] Some evidence suggests that blockade of the sympathetic nervous system may reverse the vasoconstriction-induced acidosis and improve survival after bleeding has been stopped and blood volume restored.[19–21] However, a more effective and clinically relevant treatment plan is to restore the circulating blood volume and organ perfusion.

A variety of treatment regimens, including colloids, crystalloid solutions, whole blood, and blood components, have been proposed (Table 4-4). Perhaps the best initial approach to the casualty with evidence of a mild-to-moderate hemorrhage (10%–30% of the estimated blood volume) is to rapidly administer an intravenous fluid challenge of a warmed, balanced, salt solution in 10- to 20-mL/kg increments, depending on the severity of symptoms and ongoing blood loss. Repeated fluid challenges are administered until the desired hemodynamic response is obtained. Blood products should be administered to keep pace with concurrent blood loss and to maintain an acceptable hemoglobin level (> 7 g/dL). When uncontrolled bleeding occurs, isovolumic transfusion with fresh whole blood or packed red blood cells, in conjunction with a crystalloid solution, is necessary.

Crystalloid Solutions

Although a variety of crystalloid solutions is available, 0.9% sodium chloride or lactated Ringer's solution are reasonable first choices. Isotonic sodium chloride (0.9%) may be readily mixed with blood products and is an excellent choice for volume resuscitation. However, it has the minor disadvantage of contributing to a hyperchloremic acidosis when large volumes of resuscitation fluid must be used. Lactated Ringer's solution has the advantage of having a somewhat more physiological composition, providing elemental replacement of calcium and potassium, and more physiological concentrations of sodium and chlorine ions. A disadvantage of lactated Ringer's solution is its relative incompatibility with blood products. The

TABLE 4-4

RESUSCITATION FLUIDS

Solution	Advantages	Disadvantages
0.9% NaCl	Inexpensive Compatible with blood Replenishes ECF space Increases GRF	Nonphysiological Na^+ and Cl^- concentrations Contributes to interstitial edema Large volumes required for replacement of blood loss ($\geq$ 3-fold estimated blood loss)
Lactated Ringer's	Inexpensive Physiological electrolyte concentrations Increases GFR Replenishes ECF space	Same as those for NaCl
5% Albumin (MW $6 \bullet 10^4$)	Replaces plasma loss volume for volume Provides rapid expansion of the intravascular space with less volume	Expensive Redistributes into the interstitial fluid space relatively quickly (compared to other colloids) May aggravate edema in leaky capillary states
6% Hetastarch (Hespan, Dupont) (MW 10^4–10^7)	Same as those for 5% albumin Provides a more persistent expansion of the intravascular space, relative to albumin Plasma volume expansion may persist > 24 h	Expensive May interfere with coagulation cascade if > 20 mL/kg is administered
5% Plasma Protein Fraction (83% albumin)	Resembles albumin	Similar to albumin, somewhat less osmotically active Contaminating plasma proteins may produce hypotension with rapid administration
Dextran 40 (average MW $4 \bullet 10^4$)	Provides rapid expansion of the intravascular space with a small volume Reduces incidence of thromboembolism	Risk of anaphylaxis 0.5%–5% May contribute to renal failure Interferes with platelet function and clotting Risk of bleeding with > 1.5 g/kg/d Interferes with blood cross-matching May produce osmotic diuresis
Dextran 70 (average MW $7 \bullet 10^4$)	Same as those for dextran 40 Larger MW confers a greater duration of plasma volume expansion	Same as those for dextran 40
HDS	Very rapid restoration of intravascular volume with a small volume Small volume makes field use easier	Same as those for dextran 40 Brief hypotension with rapid infusion Hypernatremia, especially if used in dehydrated casualties

ECF: extracellular fluid; GRF: glomerular filtration rate; HDS: hypertonic saline (7.5%) and dextran 70 (6%); MW: molecular weight

calcium content of lactated Ringer's solution may activate the coagulation cascade in blood products; however, slight dilution of packed red cells with lactated Ringer's solution has not been shown to cause a problem.[22] The lactate content of lactated Ringer's solution may aggravate the correction of the ongoing metabolic acidosis in individuals suffering from profound acidosis or ketoacidosis. Under most circumstances, however, when organ perfusion has been restored, the liver is quite capable of metabolizing the lactate load; the problem is, therefore, rarely of concern.[23]

Intravenous fluids containing dextrose (glucose) are rarely indicated and are potentially harmful in the management of trauma casualties. The stress response induced by major trauma or surgery frequently contributes to a normal or an elevated blood glucose level. Rapid administration of large volumes of glucose solutions during resuscitation may lead to an osmotic diuresis, confounding ef-

forts to restore the intravascular deficit. Hyperglycemia has also been linked to a poorer neurological outcome among victims of trauma, cardiac arrest, and stroke.[23–25] Glucose may be included in maintenance fluids during the postresuscitation phase.

Colloid Solutions

The use of colloid-containing intravenous solutions in the management of trauma and surgical patients remains a source of controversy and is thoroughly discussed in other textbooks[26,27] and in Chapter 24, The Syndromes of Septic Inflammatory Response and Multiple Organ Dysfunction. Historically, the only colloids that were available in the military for field use were albumin and plasma. More recently, the dextrans and hydroxyethyl starch (Hespan, manufactured by Du Pont Pharmaceuticals, Wilmington, Del.) have become widely available and are used by some civilian paramedic rescue squads for field resuscitation of casualties in hemorrhagic shock. The most significant drawback to these agents is their cost, which is excessive relative to crystalloid solutions. The attractive features of the colloids include

- rapid repletion of the intravascular compartment with a smaller fluid volume,
- a more-prolonged expansion of the plasma volume, and
- less peripheral edema.

Some have suggested a reduced risk of increased intracranial pressure as another attractive feature of colloids. This may be true in comparison to lactated Ringer's solution; however, the effects of isotonic solutions (eg, normal saline) on intracranial pressure appear comparable to those of albumin.[28]

Colloid solutions produce an increase in the intravascular colloid oncotic pressure and may draw interstitial water into the intravascular compartment.[29,30] Thus, the plasma volume expands in excess of the administered fluid volume. Conversely, in "leaky capillary" states (eg, burns, sepsis, the adult respiratory distress syndrome), the colloids can traverse the endothelial barrier and contribute to the formation of pulmonary and peripheral edema. Although abnormal capillary membranes are not likely to be present during the initial treatment of most combat casualties, other disadvantages of colloid solutions that argue against their use include the following:

- the potential for impaired coagulation,
- interference with cross-matching of blood,
- increased viscosity,
- a transient fall in ionized calcium levels (which albumin may produce), and
- the potential for a relative reduction in glomerular filtration.

The reduction in glomerular filtration rate probably reflects (*a*) the depletion of the interstitial fluid compartment through third-space losses and (*b*) the inability of colloids to replace the loss.[30] A relatively hyperoncotic state may result. Crystalloids are better suited to improving glomerular filtration. Patients with compromised glomerular filtration may be placed at increased risk of renal failure if resuscitated with a dextran solution.[31] In hypovolemic patients, renal tubular obstruction may develop due to precipitation of the dextran in the tubules.

In circumstances where blood loss is substantial and continuing, dextran and hetastarch should be used cautiously. Dextran interferes with platelet adhesion and may aggravate blood loss.[32] Hetastarch, when used in large cumulative volumes, has been shown to produce a coagulopathy, probably by interfering with Factor VIII activity.[33] Clinical case reports have linked bleeding catastrophes to the use of hetastarch in quantities greater than 20 mL/kg.[34] Life-threatening anaphylaxis can occur with the dextrans and hetastarch. The overall risk of anaphylaxis appears to be on the order of 0.5% to 5% with the dextrans and 0.08% with hetastarch.[33]

Hypertonic Solutions

Hypertonic saline solutions, alone or in combination with concentrated colloid solutions, have become the focus of intense interest over their possible use as small-volume resuscitation fluids in the field. The primary advantage of hypertonic solutions compared to conventional fluids for field resuscitation is logistical: much less hypertonic fluid is required for an equivalent degree of resuscitation—thus, the load of the combat medic is reduced. The U.S. Army Medical Research and Materiel Command at Fort Detrick, Frederick, Maryland, has had an ongoing research program into the development and clinical application of a hypertonic saline dextran solution (7.5% sodium chloride and 6% high-molecular-weight dextran 70). This solution, when used in animal models for hemorrhagic shock, has effected dramatic improvements in hemodynamic parameters and survival following a hemorrhage

that would otherwise have been lethal.[35] Limited field trials using human volunteers have documented the beneficial hemodynamic effects when hypertonic saline dextran is administered by paramedics; however, improved patient outcome has not yet been demonstrated convincingly.[36,37] One reason for this could be the rapidity with which most patients were transported to hospitals and received definitive care.

Hypertonic saline produces a rapid expansion of the intravascular space by drawing extracellular and intracellular water into the vascular compartment. Hypertonic saline alone produces short-lived improvements in hemodynamics, as the sodium tends to redistribute rapidly. The addition of a concentrated colloid solution (eg, dextran, hetastarch) increases the duration of the plasma volume expansion and improves survival in animal models.[35] However, two recent clinical investigation studies involving traumatized humans show no added benefit from dextran compared with 7.5% saline alone.[38,39] Subsequent volume replacement with isotonic, balanced salt solutions is essential to maintain organ perfusion as the effects resolve, and to reverse the relative hypernatremic/hyperoncotic state.[40] Hypertonic saline resuscitation may offer additional advantages in the management of head-injured, hypovolemic casualties by minimizing elevations in intracranial pressure and by improving the cerebral-perfusion pressure.[41] While the benefits are clearly evident in the short term (hours), the long-term benefits or disadvantages of hypertonic solutions on cerebral perfusion and neurological outcome need to be demonstrated.

One important hazard associated with the use of hypertonic-resuscitation measures in the field is the risk of promoting further exsanguination and dehydration. Improvements in blood pressure and cardiac output in casualties with uncontrolled bleeding may increase the rate of blood loss, leading to further loss of oxygen-carrying capacity, progressive acidosis, and increased morbidity. Data from studies[42] with experimental animals appear to support these conclusions. Thus, the administration of hypertonic solutions in the presence of uncontrolled hemorrhage, and in the absence of guaranteed, short-term delivery of definitive supportive and surgical treatment, may be contraindicated. Of course, the same stricture applies to resuscitation with any fluid that raises blood pressure, including lactated Ringer's solution. Further study is needed on this issue.

At present, and under most conditions, crystalloid solutions appear to be the resuscitation fluid of choice. Perhaps some combination of colloid and crystalloid solutions will prove to be the optimal approach. Colloid solutions should probably be avoided during the initial resuscitation (ie, the first 24 h) of victims with extensive burn injuries, casualties suffering from the adult respiratory distress syndrome, and in casualties suffering from preexisting dehydration. Hypertonic solutions may have a role in the immediate resuscitation of hypovolemic trauma casualties, provided continued volume support with isotonic solutions is available. Colloid solutions or hypertonic saline may prove particularly useful prior to the induction of anesthesia in conscious, hypovolemic casualties, or in hypovolemic casualties with head injuries who require immediate surgery. Of course, the development of an oxygen-carrying solution—such as a stroma-free hemoglobin solution—which, in contrast to blood, could be taken into the field by the medic, would be an important step forward in our ability to treat the combat casualty.

SUPPLEMENTAL THERAPEUTIC MEASURES

Although control of bleeding and rapid infusion of crystalloid fluids and blood are the essential steps that must be taken if the combat casualty in shock is to be salvaged, a variety of supplemental therapeutic measures have been suggested in the past; in some instances, they may play an important role. The use of other measures, such as steroid theroid therapy in the management of head trauma, continues to be controversial and unresolved. However, in the absence of Addison's disease, adrenocorticoids are unlikely to be beneficial in the management of hypovolemic shock.

Patient Position

Conventional management of hypotensive trauma victims frequently involves placing the patient in the head-down (Trendelenburg's) position to improve venous return to the heart (Figure 4-4). In otherwise normovolemic subjects experiencing hypotension (ie, those in neurogenic shock), lowering the head or elevating the legs may facilitate venous return. However, studies[43] of subjects with hemorrhagic shock have failed to demonstrate consistent hemodynamic improvement from

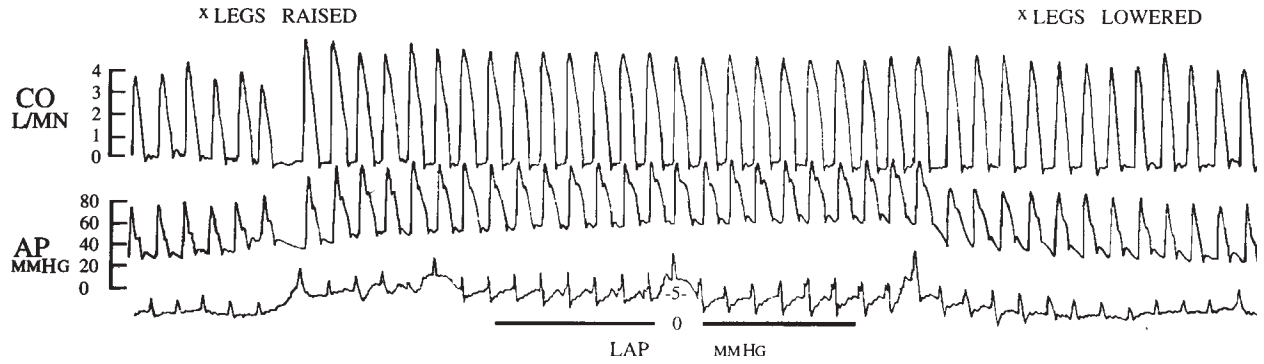

Fig. 4-4. The data shown in this phasic pressure and flow diagram were obtained in an anesthetized swine instrumented with an aortic flow probe for measuring cardiac output (CO) and pressure transducers in the proximal aorta (AP) and left atrium (LAP). When the animal's hind legs were raised abruptly, cardiac output, aortic pressure, and left atrial pressure were transiently elevated. Previously, 50% of the animal's circulating blood volume had been removed. Cardiac output remained elevated when the legs were lowered, suggesting that the blood that transfused into the central circulation when the extremities were raised has not yet been redistributed back to the legs. The fall in aortic pressure caused by lowering the legs may mean that the hydrostatic pressure head above the site of the pressure catheter is itself a factor in determining the magnitude of the measured pressure. If applicable to humans in shock, data such as these suggest that Trendelenburg's position may be hemodynamically beneficial. Source: Medical Audio Visual Aids Division, Letterman Army Institute of Research, Presidio of San Francisco, Calif. File Number 229-82-1, 1983.

Trendelenburg's position, and this position may have an adverse impact on cardiac output and blood pressure.[44] Furthermore, Trendelenburg's position may produce adverse consequences in the hypovolemic, head-injured victim by increasing intracranial pressure and reducing the cerebral perfusion pressure. Most of these problems can be avoided simply by elevating the legs to facilitate venous return, without placing the head in a dependent position.

Pneumatic Antishock Garment

The use of the pneumatic antishock garment (a compression device first known as military antishock trousers [MAST]) was introduced by the U.S. Army during the Vietnam War. Until recently, use of this garment was considered to be a standard of practice but its use in the acute management of hemorrhagic shock has now waned. The pneumatic antishock garment appears to support the blood pressure by a combination of increased systemic vascular resistance and increased venous return.[45]

There seems little doubt that proper use of the pneumatic antishock garment results in an autotransfusion of blood from the compressed tissue in the lower half of the body into the central circulation, but the actual volume is small (2–3 mL/kg) and the augmented cardiac output secondary to the increased venous return is transient (Figure 4-5). Maintenance of blood pressure with this device probably is the result of a mechanical increase in resistance in the compressed tissue and therefore is purchased at the cost of decreased perfusion of already ischemic tissue. Clinical studies[46] have demonstrated significantly reduced survival in patients treated with the pneumatic antishock garment when the site of hemorrhage is in the chest and, therefore, is not compressed by the device. This is not surprising given that any increase in blood pressure will simply increase the rate of hemorrhage. If the pneumatic antishock garment has any utility in combat casualty care, it is in stabilizing fractures of the pelvis and the long bones of the legs. In this setting, the device may help to reduce ongoing blood loss and therefore permit hemodynamic stabilization prior to definitive intervention.

Supplemental Oxygen

The routine administration of supplemental oxygen to trauma casualties may be beneficial in some victims, in that multiple or massive injuries (eg, splinting, flail chest, fat emboli) are often associated with impaired oxygenation. However, expecta-

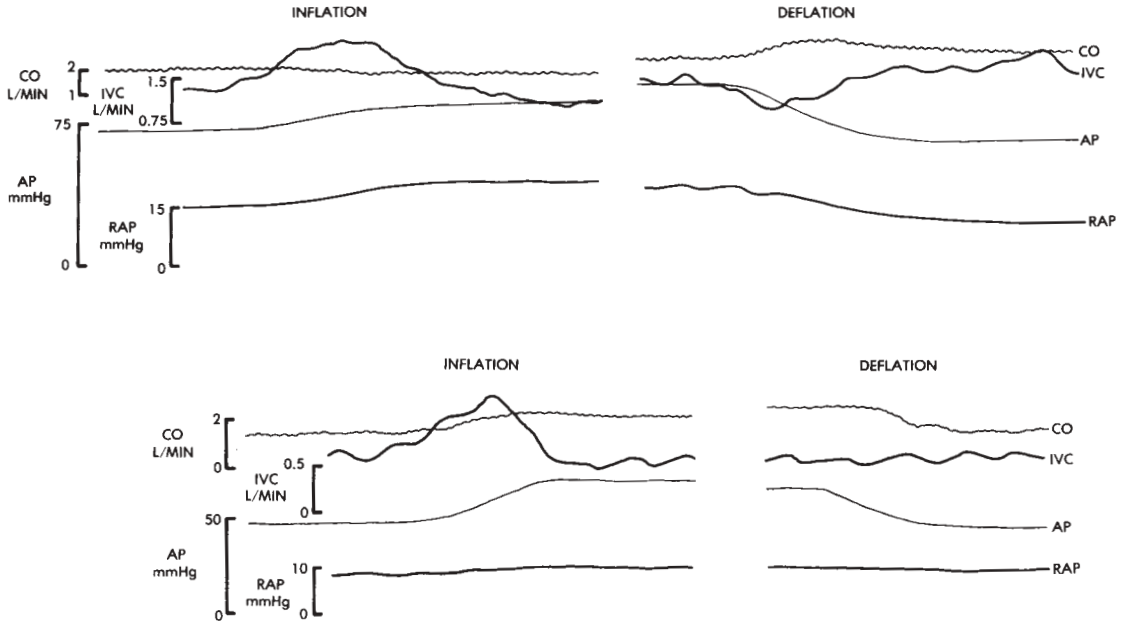

Fig. 4-5. The phasic pressure and flow data shown in this figure were obtained in an anesthetized swine instrumented with an aortic flow probe for measuring cardiac output (C.O.), a flowprobe inserted into the inferior vena cava (IVC), and pressure transducers in the proximal aorta (AP) and right atrium (RAP). A pneumatic antishock garment especially designed for application to a large swine was applied around the animal's lower trunk and hind legs. The upper panel shows pressure and flow transients associated with abrupt inflation and deflation of the pneumatic antishock garment. A bolus of blood is translocated through the inferior vena cava into the heart (equivalent to about 2 mL/kg, or 100 mL total), but cardiac output actually falls. Possibly this is due to an increase in arterial impedance caused by the external compression. The lower panel shows the effects of inflating the pneumatic antishock garment after 50% of the animal's circulating blood volume is removed: both cardiac output and aortic pressure rise. Reprinted with permission from Bellamy RF, DeGuzman LR, Pedersen DC. Immediate hemodynamic consequences of MAST inflation in normo- and hypovolemic anesthetized swine. *J Trauma*. 1984;24:892.

tions of improved tissue oxygenation in the presence of hypovolemic shock are unrealistic: hemoglobin oxygen saturation in the typical young combat casualty—who is usually hyperventilating because of the injury—is already maximal. Furthemore, the added amount of dissolved oxygen in the plasma caused by breathing supplemental oxygen is too small to be of importance. Until the circulating blood volume is restored, supplemental oxygen is unlikely to reverse the anaerobic metabolism in poorly perfused tissues.

Therapeutic Hypothermia

Reductions in body temperature are associated with a reduced metabolic rate: approximately 13% for every 1°C decrease in temperature.[47] In addition, oxygen solubility in plasma increases at reduced temperatures. Thus, hypothermia (< 37°C) may be beneficial in reducing oxygen consumption in individuals with reduced oxygen-carrying capacity if blood transfusion cannot be provided, but this approach to managing the seriously injured combat casualty can only be considered speculative.

Intraarterial Infusion

Intraarterial infusion was advocated during the Korean War because it appeared to provide the potential for more-rapid volume replacement, compared with the intravenous routes then being used. However, clinical evidence failed to show any significant advantage of this route over more conventional intravenous approaches, and its complexity argues against its use today.

Vasopressors

Efforts to support the arterial blood pressure through the administration of vasoconstrictors are mentioned here only to be condemned. Intense vasoconstriction is the typical homeostatic response to hemorrhagic shock and may be primarily responsible for the adverse consequences of hypovolemia (eg, acidosis, tissue hypoxia). Pharmacological support of the blood pressure with vasoconstrictors will only aggravate the reduction in tissue perfusion by decreasing cardiac output, and will contribute to the progressive acidosis. The benefits of transiently supporting the blood pressure with vasoconstrictors, until volume replacement or control of bleeding is possible, have not been carefully evaluated.

Vasodilators

At face value, the administration of vasodilators to a hypovolemic patient would seem to be grossly inappropriate. However, numerous animal studies[19–21]—using fixed-pressure shock models of the Wigger's type, involving the use of epidural anesthesia or the administration of adrenergic antagonists in the presence of hemorrhagic shock—have demonstrated both reductions in the severity of the systemic acidosis and improved survival. The beneficial effects of sympathetic antagonism are related to a reversal of the reflex vasoconstriction associated with hypovolemia. Tissue oxygenation and perfusion, albeit reduced, are maintained at levels adequate to reduce the progression of the acidosis. However, the beneficial application of central neuraxis anesthesia has not rigorously been evaluated in the clinical setting. Case reports have appeared in the literature; however, it is impossible to draw valid conclusions based on a limited number of uncontrolled reports. Conventional wisdom has dictated the avoidance of spinal and epidural anesthesia in hypovolemic and bleeding patients. Further study clearly is needed.

CONDITIONS PECULIAR TO RESUSCITATING COMBAT CASUALTIES

The conditions of the battlefield, which are so unlike those encountered in civilian practice, can create unique problems for military anesthesiologists. Not only are combat injuries quite different from those encountered in civilian practice, but the propensity for environmental hazards to become important sources of comorbidity must also be kept in mind.

Hyperthermia and Dehydration

Military conflicts frequently occur under adverse climatic conditions. Elevated ambient temperatures, physical exertion, and inadequate supplies on the battlefield can combine to contribute to the development of heat injury and dehydration. Coincident trauma and hemorrhage in the setting of preexisting dehydration and volume contraction will adversely influence survival. Elevated body temperature and hypotension will accelerate the progression to renal failure and contribute to the development of rhabdomyolysis. Rapid replacement of the intravascular and extracellular fluid space with isotonic, balanced, salt solutions is key to reversing this life-threatening process. Hyperoncotic colloid or hypertonic saline solutions are relatively contraindicated in casualties suffering from dehydration. The hyperosmotic state will adversely impact on glomerular filtration and further deplete the extracellular and intracellular fluid spaces.

Hypothermia

Hypothermia is a common problem afflicting trauma victims. The pathophysiology of hypothermia affects virtually all organ systems, and is discussed in detail in Chapter 28, Systemic Hypothermia. Profound hypothermia (core temperature < 30°C) is associated with substantial reductions in renal blood flow (75%) and glomerular filtration.[48,49] Despite this, profoundly hypothermic victims become progressively dehydrated due to a cold-induced diuresis secondary to defective tubular reabsorption. The urine produced is usually dilute, contributing to the development of a relatively hyperosmotic serum. Temperatures below 30°C are associated with ventricular fibrillation. Hyperkalemia and hyponatremia frequently occur with hypothermia; however, specific treatment is seldom necessary. Restoration of fluid deficits with warm hypotonic solutions (lactated Ringer's) is generally all that is required. Metabolic acidosis frequently develops as the patient is rewarmed. Fluid resuscitation to restore and maintain perfusion will correct the acidotic state; however, some may choose to treat with intravenous sodium

bonate. Colloids and hypertonic solutions are relatively contraindicated in this hyperosmotic state.

Thermal Burns

Fortunately, extensive burns are uncommon combat injuries in land warfare. They are much more common as nonbattle injuries:

> [A]lthough the incidence of combat-related burns has historically been about 3%, in recent wars the incidence is higher because mechanized modern warfare—tanks and other armored vehicles—actually places soldiers at higher risk [than previously] of being burned…. During the Yom Kippur War, burns comprised 10.5% of all injuries; during the Falkland Island Conflict, 18% of British casualties were burned. Furthermore, medical officers should know that in the United States military, burns and inhalation injuries have always been far more important sources of morbidity and mortality in both the navy and the air force than in the army.[50(p350)]

The fluid management of burn victims has been well described elsewhere[51] (also see Chapter 22, Burn Injuries) and will only briefly be discussed here. Ordinarily, burns involving more than 15% of the total body surface area will require intravenous fluid therapy. A number of treatment protocols have been developed to predict fluid requirements in the immediate and delayed phases of injury. However, as with any regimen, any given patient may require more or less fluid. Parameters of adequate organ perfusion (eg, urinary output) provide the most accurate indicators with which to guide fluid therapy.

Most authorities divide the fluid resuscitation of burned patients into two phases: the initial 24 hours and after the initial 24 hours. During the initial 24 hours, burn victims experience a generalized increase in capillary permeability that involves, in addition to the burned areas, normal capillary beds. This increased permeability has led some authorities[52,53] to suggest that colloid solutions be avoided in most patients during the initial 8 to 24 hours; the concern is that protein loss into the extracellular space will accelerate edema formation.[51] This effect is particularly evident during the initial 24-hour period. Isotonic crystalloids will lead to interstitial edema, but they also improve glomerular filtration and allow for replacement of evaporative losses. Hypertonic solutions have been touted to maintain the intravascular compartment with minimal edema.[40] However, careful monitoring of serum sodium is necessary to avoid hypernatremia. In the absence of data demonstrating a clear improvement in survival with any particular solution, it is reasonable to emphasize the use of isotonic crystalloids during the initial 24 hours after the burn. The revised Brooke formula[54] recommends administering 2 mL of lactated Ringer's solution per kilogram of body weight per percentage of total body surface area burned. Of the amount estimated to be required during the first phase, one half should be administered during the initial 8 hours, with the remaining volume dispersed over the next 16 hours. The Rule of Nines[55] is a useful indicator for estimating the percentage of body surface area burned:

- head and neck, 9%;
- anterior and posterior trunk, 18% each;
- upper extremities, 9% each;
- lower extremities, 18% each; and
- perineum, 1%.

After the initial 24 hours, colloid solutions are clearly beneficial for restoring the plasma oncotic pressure. Plasma proteins continue to be lost, not only into the burned areas but also owing to dilution and the patient's general catabolic state. Albumin solutions should be administered to maintain the plasma albumin concentration at 2.0 to 2.5 g/dL to support the plasma oncotic pressure.[51] Evaporative losses may be replaced with hypotonic dextrose solutions during this period. During this second phase, the requirement for sodium replacement generally declines and the risk of hypernatremia arises secondary to free-water loss via evaporation and urine.

Hemorrhagic Shock and Head Injury

Severe head injury coexisting with hypovolemic shock is an ominous combination (this subject is discussed in greater detail in Chapter 16, Neurological Injuries). Cerebral edema or compression by an expanding intracranial hematoma, or both, produce an elevation in intracranial pressure. Hypotension secondary to hemorrhage contributes to further decline in the cerebral perfusion pressure and thereby potentiates ischemic injury. Improvement of the mean arterial pressure with fluid resuscitation may lead to further increases in intracranial pressure, secondary to increased cerebral blood volume and progressive cerebral edema. Resuscitation with hypotonic fluids, especially dextrose solutions, is contraindicated. Unfortunately, lactated Ringer's solution is relatively hypotonic and may contribute to increased cerebral edema. Col-

bicarloid solutions have been advocated; however, with diffuse injuries, the blood–brain barrier may be damaged, contributing to interstitial leakage of colloid and worsening edema in the area of injury. Because of their adverse interactions with the coagulation system, the dextrans and hetastarch solutions must be used with caution in the setting of intracranial hemorrhage. Isotonic crystalloid solution (0.9% NaCl) does not accelerate brain edema and may be the optimal resuscitation fluid.[28]

The Chemically Contaminated Casualty

Although the anesthetic management of casualties of chemical warfare agents is discussed in detail in Chapter 30, Anesthesia for Casualties of Chemical Warfare Agents, and elsewhere, several brief points on the fluid management of the chemically injured casualty will be outlined here. The risk of exposure to chemical weapons continues to persist throughout the world, particularly in third-world nations, as witnessed by the use of chemical weapons on civilian and military targets during the Iran–Iraq War. Combat casualties in high-risk chemical environments may present a confusing diagnostic picture if chemical protective treatments (atropine, pyridostigmine) have been used. Atropine-induced tachycardia and the associated autonomic effects may present a picture easily confused with hypovolemia. Certain incapacitating agents (eg, quinuclidinyl benzylate) may produce similar effects. Pyridostigmine, an acetylcholinesterase inhibitor, was provided to many troops as a chemical protectant during the Persian Gulf War. Pyridostigmine tends to produce bradycardia and may adversely interact with the compensatory response to hemorrhage.

Fluid replacement is an important component in the management of blister agents (ie, mustard). Casualties exposed to blister agents may present with considerable cutaneous denuding and subsequent fluid loss. Treatment requires aggressive fluid management, similar to the treatment of thermal burns. Choking agents (eg, phosgene, chlorine), which may cause alveolar capillary injury, represent the opposite end of the spectrum, in that strict fluid management is needed to avoid contributing to the development of noncardiac pulmonary edema. Because careful fluid management may not be easily accomplished in patients with multiple trauma who are also exposed to choking agents, supportive therapy, including mechanical ventilation, may be needed in this population.

SUMMARY

Combat injuries associated with hemodynamic instability should be regarded as hypovolemic until proven otherwise. The rapid attainment of large-bore venous access and the initiation of fluid resuscitation should occur simultaneously with evaluation of the casualty's injuries. The administration of fluids—colloid or crystalloid—should be guided by the normalization of hemodynamic indices and especially by evidence of good peripheral perfusion (eg, high urinary output). Coexisting medical concerns peculiar to the battlefield may create confusion in the interpretation of the physiological response to intervention. Military healthcare providers must be aware of the medical implications associated with environmental and chemical concerns of the combat theater.

REFERENCES

1. Gamble JL. *Chemical Anatomy, Physiology, and Pathology of Extracellular Fluid*. 6th ed. Boston, Mass: Harvard University Press; 1954.

2. Starling EH. On the absorption of fluids from the connective tissue spaces. *J Physiol*. 1896;9:312.

3. Shires T, Coln D, Carrico J, et al. Fluid therapy in hemorrhagic shock. *Arch Surg*. 1964;88:688–693.

4. Giesecke AH, Egbert LD. Perioperative fluid therapy. In: Miller R, ed. *Anesthesia*. New York, NY: Churchill Livingstone; 1985: 1313–1328.

5. Lichtenstein A, Eckhart WF, Swanson KJ, et al. Unplanned intraoperative and postoperative hemodilution: Oxygen transport and consumption during severe anemia. *Anesthesiology*. 1988;69:119–122.

6. Mendelson JA. The use of whole blood as blood volume expanders in US military medical facilities in Vietnam 1966–1971. *J Trauma*. 1975;15:1–13.

7. Crosby WH. Acute anemia in the severely wounded battle casualty. *Milit Med*. 1988;153:25–27.

8. Beecher HK, ed. The Board for the Study of the Severely Wounded, North African–Mediterranean Theater of Operations. *The Physiologic Effects of Wounds*. In: *Surgery in World War II*. Washington, DC: US Army Medical Department, Office of The Surgeon General; 1952: 34.

9. Vayer JS, Henderson JV, Bellamy RF, Calper AR. Absence of a tachycardia response to shock in penetrating intraperitoneal injury. *Ann Emerg Med*. 1988;17:227–231.

10. Scolea TM, Holman M, Fuortes M, et al. Central venous blood oxygen saturation: An early, accurate measurement of volume during hemorrhage. *J Trauma*. 1988;28:725–732.

11. Scott WL. Complications associated with central venous catheters. *Chest*. 1988;94:1221–1224.

12. Committee on Trauma, American College of Surgeons. *Advanced Trauma Life Support Program for Physicians: Instructor Manual*. 5th ed. Chicago, Ill: American College of Surgeons; 1993.

13. Glover JL, Broadie TA. Intraoperative autotransfusion. *World J Surg*. 1987;11:60–64.

14. Fiser DH. Intraosseous infusion. *N Engl J Med*. 1990;322:1579–1581.

15. Halvorsen L, Bay BK, Perron PR, Holcroft JW, Blaisdell FW, Kramer, GC. Evaluation of an intraosseous infusion device for the resuscitation of hypovolemic shock. *J Trauma*. 1990;30:652–658.

16. Rosetti VA, Thompson DM, Miller J, et al. Intraosseous infusion: An alternative route of pediatric intravascular access. *Ann Emerg Med*. 1985;14:885–888.

17. Papper EM. Bone marrow route for injecting fluids and drugs into general circulation. *Anesthesiology*. 1942;3:307.

18. Lellehei RC, Longerbeam JK, Bloch JH, et al. The nature of irreversible shock: Experimental and clinical observations. *Ann Surg*. 1964;160:682–710.

19. Shibata K, Yamamoto Y, Murakami S. Effects of epidural anesthesia on cardiovascular response and survival in experimental hemorrhagic shock in dogs. *Anesthesiology*. 1990;71:953–959.

20. Gould L, Ettinger S, Carmichael A, et al. The use of phentolamine in experimental hemorrhagic shock. *Angiology*. 1970;21:330–335.

21. Da Luz PL, Shubin H, Weil MH. Effectiveness of phentolamine for reversal of circulatory failure. *Crit Care Med*. 1973;1:135–140.

22. King WH, Patten ED, Bee DE. An in vitro evaluation of ionized calcium levels and clotting in red blood cells diluted with lactated Ringer's solution. *Anesthesiology*. 1988;68:115–121.

23. Lanier WL, Stangland KJ, Scheithauer BW, et al. The effects of dextrose infusion and head position on neurologic outcome after complete cerebral ischemia in primates: Examination of a model. *Anesthesiology*. 1987;66:39–48.

24. Pulsinelli WA, Levy DE, Sigsbee B, et al. Increased damage after ischemic stroke in patients with hyperglycemia with or without diabetes mellitus. *Am J Med*. 1983;74:540–544.

25. Prough DS, Coker LH, Lee S, et al. Hyperglycemia and neurologic outcome in patients with closed head injury. *Anesthesiology*. 1988;69:A584.

26. Rainey, TG, English JF. Pharmacology of colloids and crystalloids. In: Chernaw B, ed. *The Pharmacologic Approach to the Critically Ill Patient*. 2nd ed. Baltimore, Md: Williams & Wilkins; 1988: Chap 12: 219–240.

27. Haupt MT. The use of crystalloidal and colloidal solutions for volume replacement in hypovolemic shock. *Crit Rev Clin Lab Sci*. 1989;27:1–26.

28. Kaieda R, Todd MM, Warner DS. Prolonged reduction in colloid oncotic pressure does not increase brain edema following cryogenic injury in rabbits. *Anesthesiology*. 1989;71:554–560.

29. Quon CY. Clinical pharmacokinetics and pharmacodynamics of colloidal plasma volume expanders. *J Cardiothoracic Anesth*. 1988;2:13–23.

30. Lamke LO, Liljedahl SO. Plasma volume expansion after infusion of 5%, 20%, and 25% albumin solutions in patients. *Resuscitation*. 1976;5:85–92.

31. Mailoux L, Swartz CD, Capizzi R, et al. Acute renal failure after administration of LMW dextran. *N Engl J Med*. 1967;277:1113–1118.

32. Weiss HJ. The effect of clinical dextran on platelet aggregation, adhesion, and ADP release in man: In vivo and in vitro studies. *J Lab Clin Med*. 1967;69:37–46.

33. Mishler JM. Synthetic plasma volume expanders—Their pharmacology, safety and clinical efficacy. *Clinics in Haematology*. 1984;13:75–92.

34. Cully MD, Larson CP, Silverberg GD. Hetastarch coagulopathy in a neurosurgical patient. *Anesthesiology*. 1987;66:706.

35. Wade CE, Hannon JP, Bossone CA, et al. *Resuscitation of Conscious Pigs Following Hemorrhage: Comparative Efficacy of Small-Volume Resuscitation with Normal Saline, 7.5% NaCl, 6% Dextran 70, and 7.5% NaCl in 6% Dextran 70*. San Francisco, Calif: Letterman Army Institute of Research, US Department of the Army; 1988. Institute Report 305.

36. Holcroft JW, Vassar MJ, Turner GC, et al. 3% NaCl and 7.5% NaCl/dextran 70 in the resuscitation of severely injured patients. *Ann Surg*. 1987;206:279–287.

37. Maningas PA, Mattox KL, Pepe PE, et al. Hypertonic saline–dextran solutions for the prehospital management of traumatic hypotension. *Am J Surg*. 1989;157:528–533.

38. Vassar MJ, Perry CA, Holcroft JW. Prehospital resuscitation of hypotensive trauma patients with 7.5% NaCl versus 7.5% NaCl with added dextran: A controlled trial. *J Trauma*. 1993;34(5):622–633.

39. Vassar MJ, Fischer RP, O'Brien PE, et al. A multicenter trial for resuscitation of injuried patients with 7.5% sodium chloride. *Arch Surg*. 1993;128:1003–1013.

40. Monafo WW, Halverson JD, Schectman K. The role of concentrated sodium solutions in the resuscitation of patients with severe burns. *Surgery*. 1984;95:129–134.

41. Prough DS, Johnson JC, Stump DA, et al. Effects of hypertonic saline versus lactated Ringer's solution on cerebral oxygen transport during resuscitation from hemorrhagic shock. *J Neurosurg*. 1986;64:627–632.

42. Gross D, Landau EH, Klin B, et al. Quantitative measurement of bleeding following hypertonic saline therapy in "uncontrolled" hemorrhagic shock. *J Trauma*. 1989;29:79–83.

43. Taylor J, Weil MH. Failure of the Trendelenburg position to improve circulation during clinical shock. *Surg Gynecol Obstet*. 1967;124:1005–1010.

44. Bivins HG, Knoop R, dos Santos PA. Blood volume distribution in the Trendelenburg position. *Ann Emerg Med*. 1985;14:641–643.

45. Zippe C, Burchard K, Gann D. Trendelenburg versus PASG application in moderate hemorrhagic hypoperfusion. *J Trauma*. 1985;25:923–931.

46. Mattox KL, Bickell W, Pepe PE, et al. Prospective MAST study in 911 patients. *J Trauma*. 1989;29:1104.

47. Michenfelder JD, Uihlein A, Daw EF, et al. Moderate hypothermia in man: Haemodynamic and metabolic effects. *Br J Anaesth*. 1965;37:738–745.

48. Vandam, LD, Burnap, TK. Hypothermia. *N Engl J Med*. 1959;261:546.

49. Hervey GR. Hypothermia. *Proc R Soc Med*. 1973;66:1055.

50. Cioffi WG, Rue LW, Buescher TM, Pruitt BA. The management of burn injury. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Part 1, Vol 5. In: Zajtchuk R, Bellamy RF, eds. *Textbook of Military Medicine*. Washington, DC: US Department of the Army, Office of The Surgeon General, and Borden Institute; 1991: 350.

51. Demling RH. Fluid replacement in burned patients. *Surg Clin North Am*. 1987;67:15–30.

52. Baxter CR. Fluid volume and electrolyte changes in the early post-burn period. *Clin Plast Surg*. 1974;1:693–709.

53. Demling RH, Kramer GC, Gunther R, et al. Effect of non-protein colloid on post-burn edema formation in soft tissues and lung. *Surgery*. 1984;95:593–602.

54. Reiss E, Stirmon JA, Artz CP, Davis JH, Amspacher WH. Fluid and electrolyte balance in burns. *JAMA*. 1953;152:1309.

55. Bowen TE, Bellamy RF, eds. *Emergency War Surgery NATO Handbook*. 2nd rev US ed. Washington, DC: Department of Defense, Government Printing Office; 1988: 37.