

Fluid Resuscitation Therapy for Hemorrhagic Shock

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■ ABSTRACT

Hemorrhagic shock is a severe life-threatening emergency affecting all organ systems of the body by depriving tissue of sufficient oxygen and nutrients by decreasing cardiac output. This article is a short review of the different types of shock, followed by information specifically referring to hemorrhagic shock. The American College of Surgeons categorized shock into 4 classes: (1) distributive; (2) obstructive; (3) cardiogenic; and (4) hemorrhagic. Similarly, the classes of hemorrhagic shock are grouped by signs and symptoms, amount of blood loss, and the type of fluid replacement. This updated review is helpful to trauma nurses in understanding the various clinical aspects of shock and the current recommendations for fluid resuscitation therapy following hemorrhagic shock.

■ KEY WORDS

Advanced Trauma Life Support, Hemorrhagic shock, Resuscitative fluids

The leading cause of death with regard to civilian and military traumas is hemorrhagic shock.¹ Since hemorrhagic shock has a high mortality rate, research is crucial in finding the most effective treatment. The article pro-

vides a review of the 4 types of shock, the 4 classes of hemorrhagic shock, and the latest research on resuscitative fluid. The 4 types of shock are categorized into distributive, obstructive, cardiogenic, and hemorrhagic shock. Hemorrhagic shock has been categorized into 4 classes, and based on these classes, appropriate treatment can be planned. Crystalloids, colloids, dopamine, and blood products are all considered resuscitative fluid treatment options. Each individual case requires various resuscitative actions with different fluids. Healthcare professionals who are knowledgeable of the information in this review would be better prepared for patients who are admitted with hemorrhagic shock, thus providing optimal care.

■ DISTRIBUTIVE SHOCK

Distributive shock is composed of 3 separate categories based on their clinical outcome. Distributive shock can be categorized into (1) septic; (2) anaphylactic; and (3) neurogenic shock.

Septic shock

In accordance with the American College of Chest Physicians and the Society of Critical Care Medicine in August of 1991,² sepsis was described as an infection-induced syndrome characterized by an inflammatory cascade that is activated within a patient. This cascade leads to a reaction in the body with 4 main areas being affected. When 2 of the 4 areas of the body become excessively inflamed, the systemic inflammatory response syndrome is triggered. These 4 parameters and their respective

- Body temperature higher than 38°C or lower than 36°C;
- Heart rate more than 90 beats per minute (bpm);
- Respiratory rate more than 20 breaths per minute; and
- White blood cell count more than 12,000/mm or lower than 4,000/mm or with more than 10% bands.^{3,4}

Treatment of septic shock includes multiple modalities. A protocol was published in the *New England Journal of Medicine* in 2001 and termed the early goal-directed

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therapy. The advantages of recommended therapy was 3-fold: (1) early recognition of patients with potential sepsis; (2) early broad-spectrum antibiotics; and (3) a rapid crystalloid fluid bolus.^{5,6}

The early goal-directed therapy includes an algorithm that depends on the patient response. For instance, if tissue hypoperfusion results when persistent hypotension is not controlled despite fluid resuscitation, there are possible treatment modalities.⁷ Vasopressive drugs including dobutamine, dopamine, and, possibly, packed red blood cells (PRBCs) may be administered to the patient in septic shock.⁸

Anaphylactic shock

This form of shock is due to the body's response to an allergen, antigen, drug, or a foreign protein that causes the release of histamine. Histamine systemically causes rapid and widespread vasodilation of blood vessels, leading to hypotension and increased capillary permeability.⁹ Rapid constriction of the airway (angioedema), urticaria, and bronchospasm also occurs with anaphylactic shock.¹⁰ According to the Advanced Trauma Life Support (ATLS) guidelines, the priority in treating anaphylactic shock is to maintain a rapid airway patency, which might include endotracheal intubation.¹¹ Rapid infusion of a crystalloid solution and intravenous epinephrine may also be necessary. Inhaled β -agonists and antihistamines may be useful in opening the compromised airway and other symptoms. To treat the bronchospasm, corticosteroids may be administered intravenously.¹²

Neurogenic shock

Neurogenic shock is the least occurring type of shock observed and is typically caused by trauma to the spinal cord or brain.¹³ This type of shock leads to the immediate loss of autonomic and motor reflexes below the location of the injury. The inability of the sympathetic nervous system to stimulate the vessel walls causes them to relax. Relaxation of these walls leads to rapid and widespread vasodilation and hypotension. The classic presentation of neurogenic shock is hypotension without tachycardia or cutaneous vasoconstriction.¹¹ One suggested treatment is placing the patient in the Trendelenburg position, with the head lower than the pelvis, to increase blood flow to the brain. However, this treatment has not been shown to be successful in multiple studies.¹⁴⁻¹⁷ Using vasopressors such as intravenous dopamine or dobutamine may be more appropriate and efficacious in forcing blood flow and volume to increase, thus reversing shock.¹⁸

OBSTRUCTIVE SHOCK

In obstructive shock, blood flow is reduced and thus prevented from entering the heart. This lack of blood flow leads to circulatory arrest, causing the heart to stop

pumping blood throughout the body.¹⁹ One common circumstance that may cause cardiac arrest is cardiac tamponade in which the pressure of the blood within the pericardium greatly decreases its venous return to the heart. Other causes may be a tension pulmonary embolism pneumothorax or a tumor.^{20,21} The obstruction must be removed to reverse the obstructive shock. The treatments differ according to the initial cause of the obstruction.

In a case where a pulmonary embolism exists, resulting in interference of ventricular emptying, a thrombectomy may be necessary, which has been shown to improve hemodynamics as well as lowering the critically high pulmonary pressures.²² The most common method of treatment of cardiac tamponade is removal of the fluid (usually around 50 mL) by syringe.²³ Pneumothoraces call for a variety of procedures, which usually involve catheter placement within the chest cavity to remove the lung-collapsing agent.²⁴

CARDIOGENIC SHOCK

Cardiogenic shock occurs when the ventricles are unable to function effectively, resulting in inadequate circulation due to decreased cardiac output. Much like other types of shock, there are classic symptoms of cardiogenic shock. These symptoms include decreased urine output, altered mentation, and hypotension. Symptoms specific to cardiogenic shock consist of jugular venous distention, cardiac gallop, and pulmonary edema. A study by Muhlberg and Ruth-Sahd in 2004 defined the existence of cardiogenic shock as systolic blood pressure of less than 90 mm Hg for longer than 30 minutes and evidence of tissue hypoperfusion with left ventricular filling pressure remaining adequate.²⁵ This tissue hypoperfusion is manifested in patients with symptoms such as cold peripheral tissues, oliguria (<30 mL/h), or the two combined (Table 1).²⁶⁻²⁹

When treating cardiogenic shock, clinicians focus on targeting the underlying cause, early diagnosis, and prevention of further ischemia. Intravenous fluids, vasopressors, vasodilators, analgesics, phosphodiesterase enzyme inhibitors, diuretics, and natriuretic peptides may all be used for treatment depending on the clinical signs and the cause of such shock.^{28,30,31}

HEMORRHAGIC SHOCK

Hemorrhagic shock is generally precipitated by a traumatic event that results in an acute loss of blood from the intravascular space. Patients who experience hemorrhagic shock severely impair their ability to provide adequate tissue perfusion and oxygenation after blood loss.³² Loss of circulating volume leads to a decrease of venous return to the heart and reduces end-diastolic volume (preload). This reduction in preload decreases the myocardial

TABLE 1

Causes of Cardiogenic Shock

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|--|--|
| ■ Acute myocardial infarction | ■ Myocardial contusion |
| ■ Aortic stenosis | ■ Myocarditis |
| ■ β -Blocker or calcium channel blocker overdose | ■ Respiratory acidosis |
| ■ Coarctation of the aorta | ■ Restrictive cardiomyopathies |
| ■ Endocarditis | ■ Rupture of the free wall |
| ■ Hypertrophic cardiomyopathy | ■ Rupture of the inter ventricular septum |
| ■ Hypocalcaemia, hypophosphatemia | ■ Septic shock with severe myocardial depression |
| ■ Malignant hypertension | ■ Valvular heart disease |
| ■ Mitral or aortic regurgitation | ■ Ventricular hypertrophy |
| ■ Mitral stenosis | ■ Ventricular infarctions |

muscle fiber length, reducing contractility of the heart and thus decreases cardiac output. A decrease in cardiac output then causes inadequate cellular oxygen supply and impaired tissue perfusion.³³⁻³⁶

An acute loss of blood results in a cascade of compensatory events that affect all organ systems. The initial response to hypovolemia is to decrease circulation to less vital organs such as the kidneys, gut, and skin in order to preserve circulation to priority organs such as the heart, brain, lungs, and skeletal muscle.^{11,37} This shunting to vital organs is triggered from a decrease in cardiac output and subsequently pulse pressure, which is sensed by baroreceptors within the aortic arch and atrium. Neural reflexes then cause a sympathetic outflow to the heart and other organs, which respond by increasing heart rate and vasoconstriction.^{33,37-39} A hormonal response occurs, and activation of the renin system leads to vasoconstriction and the retention of sodium and water. The anterior pituitary and adrenal medulla are stimulated to release adrenocorticotrophic hormone, epinephrine, and norepinephrine, which enhance compensatory mechanisms. At the cellular level, a decrease in perfusion causes the cells to switch from aerobic to anaerobic metabolism. Lactic acid is formed that causes metabolic acidosis.^{11,33,37,40} If the blood loss continues, the compensatory mechanisms begin to fail and damage occurs throughout the body. Myocardial hypoperfusion and lactic acidosis lead to cardiac dysfunction, which, in turn, perpetuates the entire process. Cerebral hypoperfusion leads to cardiac and respiratory depression and failure of the sympathetic nerv-

ous system. Vasodilation then occurs because of the failure of the systemic nervous system, leading to venous pooling and increased capillary permeability. Disseminated intravascular coagulation develops because of hematologic dysfunction including hypotension, hypoxemia, acidosis, and cessation of capillary blood flow. Respiratory distress syndrome may result from increased pulmonary capillary membrane permeability, microemboli formation, and pulmonary vasoconstriction. Renal vasoconstriction and hypoperfusion lead to acute tubular necrosis, and eventually to renal failure. Gastrointestinal organs also fail because of hypoperfusion and vasoconstriction.^{33,41}

At the cellular level, irreversible damage commences because the cell membrane loses its integrity, much of which is due to free radicals, particularly reactive oxygen species (ROS) and reactive nitrogen species. Both of these types of free radicals have an unpaired electron that leads to the unwanted oxidation of DNA molecules, fatty acids, and amino acids, thus advancing cell degradation. The electrical gradient is lost, which causes the cell to swell. The endoplasmic reticulum and mitochondria are damaged and utilization of oxygen becomes dysfunctional. Enzymes that are released by ruptured lysosomes digest other cellular structures. Cell death eventually occurs, which further enhances the impact of the initial hemorrhage.^{11,33,42}

Much of the damage of hemorrhagic shock at the cellular level comes from the formation of ROS in neutrophils, the most abundant type of white blood cells.⁴³

Neutrophils are the chief combatant of infections, using ROS as a double-edged sword to fight pathogens. ROS function directly by breaking down microbial pathogens, and indirectly through activation of diverse signaling pathways between neutrophils as mediators in fighting an infection.⁴⁴ However, ROS also act as signaling molecules in apoptotic (programmed cell death) pathways. Therefore, an increase of ROS in a patient would result in increased levels of apoptosis. In an effort to slow down apoptosis, the mitochondria in the cells may undergo respiratory burst. During respiratory bursts, the mitochondria work to produce higher levels of the reducing agent nicotinamide adenine dinucleotide phosphate oxidase (NADPH), which is a free radical scavenger. Although ROS act beneficially by breaking down pathogens, neutrophils overproduce and accumulate ROS, thus causing apoptosis.⁴⁴ In hemorrhagic shock in a clinical setting, increased apoptosis as a result of free radicals would be noticeable in the expression of sepsis.⁴⁵

Recent studies have investigated the cellular pathways involved in the immune response following hemorrhagic shock, particularly T cells.⁴⁶⁻⁴⁹ By studying T-cell proliferation in response to stimulators in the adaptive and innate immune responses, researchers have found decreased levels of helper T-cell cytokine production and

reduced antibody secretion.⁴⁶ The abatement in these specific levels corresponds to the suppressed adaptive immune function observed in patients with hemorrhagic shock. Thus, researchers are working to find which receptors are involved in the reduction of the adaptive immune function and how to trigger the specific receptors to keep the adaptive immune system function at a level that would prevent infection after hemorrhagic shock or serious injury.⁴⁷⁻⁴⁹ From a physiological perspective, studies have shown that the detrimental effects of hemorrhagic shock can be lowered by the interaction of different cytokines with the vagus nerve.

Classes of hemorrhagic shock

The clinical manifestations of hemorrhagic shock vary depending on the amount of blood lost and the body's ability to compensate for that loss. There are 4 classes of hemorrhage that are based on the amount of blood lost, and each class has associated clinical manifestations.⁵⁰ However, in individual patients, a distinction between classes may not be appropriate and treatment should be guided by the individual's response to the initial therapy. The American College of Surgeons ATLS defines the classes of hemorrhagic shock according to the percentage of blood loss (Table 2).^{11,51}

TABLE 2

Classes of Hemorrhagic Shock Based on Percentage of Blood Loss

Blood Loss ^a			
0%–15% or up to 750 mL	15%–30% or 750–1,550 mL	30%–40% or 1,500–2,000 mL	>40% or >2,000 mL
<ul style="list-style-type: none"> Minimal tachycardia Normal or increased pulse pressure 	<ul style="list-style-type: none"> Tachycardia Tachypnea Decrease in pulse pressure Cool clammy skin Delayed cap refill Slight anxiety 	<ul style="list-style-type: none"> Tachycardia Tachypnea Decreased systolic blood pressure Oliguria Changes in mental status such as confusion or agitation 	<ul style="list-style-type: none"> Marked tachycardia Decreased systolic blood pressure Narrowed pulse pressure Markedly decreased (or no) urinary output Loss of consciousness Cold, pale skin

^aThese guidelines are for a 70-kg adult male.

Class I (initial stage)

The initial stage of hemorrhagic shock is characterized by a blood loss of up to 750 mL or 15% of blood volume. Compensatory mechanisms maintain cardiac output and in healthy patients no changes occur in blood pressure, pulse pressure, or respiratory rate. Blood volume is generally restored within 24 hours and replacement of fluid loss is generally unnecessary.¹¹

Class II (compensatory stage)

The second stage is characterized by a blood loss of 750 to 1,500 mL or 15% to 30% of blood volume. Cardiac output decreases, which initiates several compensatory mechanisms. Tachycardia (heart rate >100 bpm) results from an increase in stimulation of the sympathetic nervous system. Pulse pressure narrows because of a rise in diastolic blood pressure. This rise is caused by an increase in circulating catecholamines, which produce an increase in peripheral vascular resistance. Systolic pressure remains relatively stable in this stage.¹¹ Respiratory rate increases in an effort to improve oxygenation. Arterial blood gas results often have lowered partial pressure of both arterial carbon dioxide (PaCO_2) and arterial oxygen (PaO_2) levels, indicating respiratory alkalosis and hypoxemia. Urinary output declines mildly because of decreased renal perfusion. Peripheral vasoconstriction in these patients result in decreased capillary refill and pale, cool skin.^{33,52} Neurologically, the patient may become anxious, irritable, or mildly confused because of decreased cerebral perfusion. At this stage, replacement of fluid is necessary but blood transfusion is generally not required.¹¹

Class III (progressive stage)

The third stage is characterized by a blood loss of 1,500 to 2,000 mL or 30% to 40% of blood volume. Compensatory mechanisms begin to fail and impaired tissue perfusion occurs. Heart rate continues to increase (>120 bpm), which can lead to myocardial ischemia and dysrhythmias. At this stage, systolic blood pressure falls and respiratory distress occurs, which is evident by an increased respiratory rate. Arterial blood gas values usually reveal high PaCO_2 , low bicarbonate (HCO_3^-), and low PaO_2 levels, indicating respiratory and metabolic acidosis and hypoxemia. Urinary output significantly decreases because of reduced renal perfusion. Blood urea nitrogen and serum creatinine levels rise because the kidneys begin to fail. Mental status begins to deteriorate significantly as cerebral perfusion decreases even further. At this stage stopping the hemorrhage is priority and generally blood transfusions are necessary.¹¹

Class IV (refractory stage)

The fourth stage is characterized by a blood loss of more than 2,000 mL or 40% of blood volume. This amount of

loss is life-threatening. Compensatory mechanisms deteriorate and organ failure ensues. Clinical manifestations include significant tachycardia (>140 bpm), severe hypotension, narrow or unobtainable pulse pressure, absent peripheral pulses, and negligible urinary output. The patient becomes cold, cyanotic, with decline in mental status, leading to unconsciousness. An immediate and rapid fluid transfusion and surgical intervention is required.¹¹

Promptly alleviating the cause of hemorrhage is essential before administering resuscitative fluids. For example, first applying a tourniquet in an amputated limb is essential to decrease the excessive bleeding. In addition, vasopressive drugs are highly recommended for maintaining coronary and cerebral blood flow during the acute event.⁵³ Administration of a crystalloid or colloid fluid such as normal saline (NS) or lactated Ringer's solution (LR), blood products, or hemoglobin-based fluids are also essential. Pinto⁵⁴ identified that appropriate treatment is essential to avoid what they called a "classic trimodal distribution of deaths."⁵⁴ The trimodal distribution includes one of the following events: (1) death due to immediate exsanguination within minutes of the event; (2) death within the first 24 hours due to progressive decompensation; or (3) death within days to weeks of the initial event due to sepsis and eventual organ failure.⁵⁵ With appropriate action, patient deaths due to hemorrhagic shock can be prevented.

■ CURRENT RECOMMENDATIONS FOR FLUID RESUSCITATION

The ATLS guidelines published by the American College of Surgeons address many factors when managing patients requiring life support, including treatment of hemorrhagic shock. Initial management includes rapid intravenous infusion of supportive fluids. There are debates among healthcare providers on the type of fluid that is most effective in relieving shock. The ATLS guidelines suggest that diagnosis and treatment of shock need to occur simultaneously. Life-threatening injuries must be assessed and managed immediately to prevent fatal complications. The initial fluid therapy should be started as soon as possible and subsequent therapy determined on the basis of the patient's response to the initial fluids. The response should be continually assessed to determine whether perfusion is adequate or not. The type and amount of fluid given must be adjusted on the basis of the patient's response. Many factors including age, previous health status, other injuries, and medications can all affect a patient's response to fluid resuscitation.^{1,56,57}

The ATLS guidelines recommend that a crystalloid solution be administered intravenously immediately. The estimated amount of fluid needed for replacement is 3 mL for every 1 mL of blood loss because some of the fluid

administered is lost into the interstitial and intracellular spaces. Other types of fluids used for replacement are discussed in detail in the next section.¹¹

Normal saline/Ringer's lactate solution

The American College of Surgeons ATLS guidelines recommend the Ringer's lactate solution as the preferred choice for the initial treatment of uncontrolled hemorrhagic shock. NS, a different crystalloid, may also be used. However, there are concerns regarding hyperchloremic acidosis with the administration of NS. Acidosis is a physiologic problem that often complicates early management of patients with hemorrhagic shock.⁵⁸ Warmed LR solution provides intravascular expansion and promotes stabilization of the vascular volume by replacing fluid losses in the interstitial and intracellular spaces.^{11,54} The initial dose is 1 to 2 L for an adult or 20 mL/kg in a pediatric patient. However, it is important to keep in mind that this volume concentration is a rough guideline and close monitoring of the patient is critical. The responses that nurses should monitor after the infusion of LR include urinary output, level of consciousness, respiratory rate, peripheral perfusion, blood pressure, pulse pressure, and pulse rate. Lastly, recognizing the reversal of the initial signs and symptoms of hemorrhagic shock is one of the main goals when administering treatment.

One area of concern with LR administration is stimulation of apoptosis. *Apoptosis* is defined as programmed cell death in a multicellular organism.^{59,60} A study by Rhee et al⁶¹ demonstrated an increased apoptosis in intestinal mucosa, smooth muscle, liver, and lung cells with LR infusion following hemorrhagic shock. However, fluid resuscitation with sham, plasma, fresh blood, and hypertonic saline all demonstrated no significant apoptosis. The infusion of LR revealed increased adhesion molecule expression, leading to increased neutrophil activation and release of ROS, thus furthering apoptosis. Apoptosis then culminated in pulmonary edema and inflammation. However, neutrophil activation did not occur with whole blood transfusions. The effects of hypertonic saline on adhesion were between those of LR and whole blood.⁶¹

Colloids

Colloids are high-molecular weight substances such as albumin, dextran, and hetastarch that increase plasma oncotic pressure.³⁵ These substances can be used to rapidly increase circulatory volume. However, there is considerable controversy as to whether colloids are a better choice than crystalloids for fluid resuscitation in hemorrhagic shock. Colloids maintain intravascular volume without causing tissue edema in comparison with crystalloids. However, colloids are associated with increased risk of coagulopathy, renal dysfunction, and hyper-

chloremic acidosis.⁶² Currently, the guidelines of the American College of Surgeons recommend the replacement of 3 mL of colloids for every 1 mL of blood lost.³⁵

Schierhout and Roberts⁶³ found a 4% increase in the absolute risk of mortality with the use of colloids during fluid resuscitation in patients with hemorrhagic shock. These authors recommended that because of this increased risk and the increased cost, colloids should not be used for volume replacement.⁶³ However, Ferreira et al.⁶⁴ demonstrated that early colloid replacement with hydroxyethyl starch was more effective than LR in restoring cardiac output and tissue perfusion.⁶⁴ They also found that mean arteriolar pressure, central venous pressure, and plasma colloid osmotic pressure were significantly higher after the infusion of hydroxyethyl starch than with the infusion of LR. Hydroxyethyl starch maintained microcirculation, hemodynamics, and colloidal osmotic pressure better than LR following hemorrhage.⁶⁵ Rhee et al⁶¹ found that hypotonic(isotonic solutions, including LR and artificial colloid solutions, caused a severe immunologic response, coagulopathy, and renal failure after administration in hemorrhagic shock. This effect, which plays a significant role in organ injury, was not observed with the use of plasma, natural colloids (albumin), and whole blood during fluid resuscitation.⁶¹

Although there are multiple studies on the effectiveness and safety of crystalloids versus colloids in fluid resuscitation, the results are varied. Additional research is needed to determine optimal fluid resuscitation for hemorrhagic shock.⁶⁶⁻⁶⁸

Therapeutic drug: Dopamine

A side effect of using resuscitating fluids such as LR and NS in a patient with hemorrhagic shock is hypoperfusion. Dopamine, an inotropic drug, may be used to increase cardiac output and blood pressure in such patients. Dopamine is used not only to increase cardiac output but also to improve renal output as a naturally occurring catecholamine.⁶⁹ Dopamine is a sympathomimetic agent that stimulates adrenergic receptors. The actions of this drug on different receptors are dose dependent. Lower dosages (<5 µg/kg per minute) stimulate dopaminergic receptors, which cause renal, mesenteric, cerebral, and coronary bed vasodilation, thus increasing blood flow to these tissues.^{70,71} Moderate dosages (5–10 µg/kg per minute) stimulate β-1 receptors, which increase myocardial contractility and heart rate that improve cardiac output.⁷² Higher dosages (>10 µg/kg per minute) stimulate β-receptors and result in an increase in systemic vascular resistance, which may counteract the actions of the dopaminergic and β-1 receptors.^{33,69,73,74}

The ATLS guidelines suggest that although the use of exogenous vasoconstrictors in hemorrhagic shock increases peripheral vascular resistance, it does not

necessarily indicate an increase in cardiac output and may further reduce end-organ perfusion and oxygenation.¹¹ There are previous and ongoing studies that suggest that dopamine benefits several major organs during hemorrhagic shock.⁷⁵⁻⁸⁰

An early study by Carvalho et al⁷⁶ evaluated the effect of dopamine on blood flow to the myocardium, intestine, kidney, and cardiac output of dogs after inducing hemorrhagic shock.⁷⁶ Dopamine was then given at a rate of 1 to 8 µg/kg per minute with NS over a period of 30 to 200 minutes. The results of this study demonstrated that dopamine caused an increase in cardiac output and systemic pressure. Coronary, renal, and mesenteric flow increased as the resistance to these areas decreased. Trachte et al.⁷⁵ found that dopamine injected at a rate of 4 µg/kg per minute had hemodynamically beneficial effects on hemorrhagic shock, raising mean arterial pressure and liver blood flow, but did not improve the metabolic and cellular responses.^{75,77} Sakahira et al⁷⁸ demonstrated that dopamine injected at a rate of 5 µg/kg per minute increased renal blood flow. However, at this concentration, it did not significantly improve cardiac output.⁷⁸ A study by Nordin et al⁸⁰ demonstrated that a dopamine infusion (5 µg/kg per minute) during crystalloid volume replacement led to enhanced tissue oxygen perfusion in the liver and subcutaneous and transcutaneous tissues. These effects were optimized when the dopamine was administered at the beginning of resuscitation, before hemodynamic function had been restored.⁸⁰

Blood products

Blood products are used to restore circulating volume, replace coagulation factors, and improve oxygen-carrying capacity. The ATLS guidelines state that a transfusion should be given on the basis of the patient's response to the initial fluid resuscitation. A replacement of PRBCs is recommended to maintain a hematocrit of more than 30%, which generally results in a volume that is less than the 3:1 rule recommended for crystalloids. Type-specific blood is preferred, but if unavailable, type O negative may be used. Platelets and/or fresh frozen plasma should be administered to a patient with a platelet count of less than 10 000 µL, transfusion of more than 6 units of PRBCs, or having abnormal coagulation studies.¹¹

There are many studies that are currently examining alternatives for hemorrhagic shock fluid resuscitation. One area of interest is the use of blood substitutes that have been modified from human hemoglobin extracted from red blood cells. These substitutes were developed as an alternative to the infusion of PRBCs and plasma.⁸¹ A study conducted by the Department of Surgery at Denver Health Medical Center in 2002 examined the use of Polyheme in hemorrhagic shock resuscitation. The study focused on measuring the inflammatory response in

patients using Polyheme versus PRBCs. The results demonstrated higher elevation in 3 interleukins in patients resuscitated with PRBCs than those resuscitated with Polyheme.⁸² Another study found that Polyheme may be a useful alternative to PRBCs. In this study, Polyheme maintained total hemoglobin despite a marked fall in red blood cell count and a reduced use of allogenic blood.⁸³ These products are currently in stage III clinical trials and have not yet received the Food and Drug Administration approval.

CONCLUSION

There are different types of shock, each can be life-threatening and each having an array of suggested treatment protocols. Hemorrhagic shock research suggests conflicts with regard to treatment methods. However, new methods of fluid resuscitation involving the use of crystalloids, colloids, dopamine, and blood products are being advised. Although the 2004 ATLS guidelines continue to be the "criterion standard" for treatment of hemorrhagic shock, these protocols do not always reflect the newest research, specifically pertaining to the mechanisms of hemorrhagic shock. Understanding how new fluid resuscitation therapies might mitigate the harmful effects of treating hemorrhagic shock will assist nurses in providing better care to patients with hemorrhagic shock in a trauma setting.

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