

Shock and Infection in Critical Care Primer

Introduction

Introduction/Purpose Statement

Failure of the normal regulatory mechanisms in the body can lead to rapid and profound shock. The purpose of this home study is to review the pathophysiology of cardiogenic, hypovolemic, anaphylactic, and neurogenic shock. A brief review of sepsis and septic shock is also covered.

Target Audience

This home study was designed for the novice critical care or telemetry nurse; however, other health care professionals are invited to complete this packet.

Content Objectives

1. List the classifications of shock.
2. List the functions of the cell and the microcirculation.
3. Describe the stages of shock.
4. Describe three major mechanisms put into action to compensate for shock.
5. Define terms related to shock.

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Contact Hour Information

For completing this Home Study , you are eligible to receive:	2.0 contact hours (calculated using the Minnesota Board of Nursing 50 minute contact hour). OR 1.66 contact hours (calculated using the WNA-CEAP criteria for certified nurses needing ANCC-approved contact hours). Criteria for successful completion: You must read the home study packet, complete the post-test and submit it to TCHP for processing.
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Please see the last page of the packet before the post-test for information on submitting your post-test for contact hours.

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An Overview of Shock

Definition

Shock is a state of inadequate perfusion relative to tissue demands.

Classification

The integrity of the circulatory system is dependent on: (a) efficient cardiac pump, (b) an adequate blood volume, and (c) a healthy vascular bed. The loss of any one of these three essential components leads to one of the three major classes of shock:

- **Cardiogenic:** loss of an efficient cardiac pump
- **Hypovolemic:** inadequate blood volume
- **Distributive** (neurogenic, anaphylactic, and septic): an unhealthy vascular bed

The cascading events of shock begin with inadequate oxygen transport and cellular dysfunction, which proceed to tissue and vascular disturbances, and end with organ dysfunction or failure.

Oxygen Transport

Oxygen transport has two components: oxygen delivery (DO_2) and oxygen utilization /consumption (VO_2). Oxygen delivery (DO_2) is the product of cardiac output and arterial oxygen content. Calculation of the arterial oxygen content depends on (1) the hemoglobin content of blood, (2) the oxygen saturation of hemoglobin, and (3) the amount of oxygen bound to hemoglobin. Changes in any of these three factors and/or changes in cardiac output alters oxygen delivery to tissues.

Normally, systemic oxygen delivery is five times greater than oxygen consumption. In other words, 20 percent of DO_2 is absorbed (VO_2), while 80 percent of DO_2 remains in returning venous blood. The body adjusts to maintain this ratio; usually by increasing or decreasing cardiac output.

Tissue oxygen utilization cannot be directly measured; however, the calculation of VO_2 infers utilization and serves as a guide to the adequacy of tissue perfusion and cellular metabolism. Factors that determine VO_2 are: (1) DO_2 , (2) state of microcirculation, and (3) cellular milieu.

Life at the Cellular Level

The cell is the unit, or building block, of all living things. The cell has several structures that are vital for functioning:

1. **Cell membrane:** a barrier with selective permeability between plasma and interstitial fluid that allows interchanges to occur between the cell and its environment. When damaged, it becomes permeable to almost anything.
2. **Nucleus:** controls the biochemical reactions; site of cellular reproduction.
3. **Cytoplasm:** the protoplasm within the cell but outside of the nucleus; site of most cellular activity.
4. **Organelles:** specialized metabolic machinery of the cell that produce and store protein, detoxify contents, aid in phagocytosis, and provide cellular energy.

Cellular metabolism refers to all chemical and energy transformations that occur in the body, including anabolic and catabolic reactions. Carbohydrates, proteins, and fats are oxidized, producing CO_2 , H_2O , heat, and chemical energy. This oxidation (catabolism) is a complex, slow process which liberates energy (ATP) in small, usable amounts.

The Microcirculation

The term microcirculation is used to describe a group of blood vessels within the tissues that acts as an independent organ unit in regulating blood supply to the tissues. The functions of the microcirculation are to:

- Deliver nutrients to, and remove wastes from, cells
- Adjust blood flow in response to tissue metabolic needs
- Maintain intravascular/interstitial osmotic equilibrium

The portion of the vascular bed lying between the arterioles and the venules is considered the microcirculation. There are no distinct boundaries between the divisions, and the arrangement and distribution differ from tissue to tissue depending on architecture and function.

The artery has a strong, smooth muscle wall, and directs blood to capillary beds and controls pressure

of the blood delivered to those beds. Arterioles are referred to as “resistance vessels.” Adjustments to the blood flow, and therefore, tissue perfusion pressure, is made by the sympathetic innervation and vasomotor influences.

The arteries branch into the metarterioles, and from there into the pre-capillary sphincters. The capillaries at the end of the arterial system form a junction with the venous system.

It is in the capillary system that nutrients, oxygen and waste products are exchanged from the arterial side to the venous side. Once that process is complete, the blood exists into the venules and finally the veins.

The microcirculation is controlled by the metabolites from surrounding tissues. These metabolites have an intrinsic capacity to regulate blood flow to compensate for changes in the perfusion pressure and metabolic needs. There is a delicate balance between blood flow and tissue demand that is maintained by the (1) autonomic nervous system (modulates vascular tone), (2) humoral, (3) chemical, and (4) metabolic influences.

Moment to moment redistribution of blood flow through the microcirculation is known as **autoregulation**. Actively metabolizing cells release **local mediators** such as K^+ , H^+ ion, CO_2 , and lactic acid, causing local vasodilatation in order to deliver greater blood flow to vascular beds with higher metabolic activity.

Pathophysiology of Shock: Initial Stage

This is the stage in which there are (theoretically) cellular changes in response to shock. There are also no clinical signs or symptoms except elevated lactate levels.

In the initial stage of shock, the cell switches from aerobic metabolism to anaerobic metabolism, which causes decreased energy production and increased lactic acid levels. Diminished blood flow to the microcirculation reduces oxygen delivery and sequesters metabolic by-products, thereby reducing oxygen delivery and utilization. The cell metabolism suffers, and the cell begins to deteriorate.

Compensatory Stage of Shock

The homeostatic compensatory mechanisms of the body are activated by decreased cardiac output. Compensation is mediated through neural, hormonal, and chemical changes.

Neural Compensation

Baroreceptors located in the aorta and carotid bodies sense a decrease in the blood pressure. Messages are sent to the medullary vasomotor center that stimulates the sympathetic nervous system. The SNS uses the endogenous catecholamines (epinephrine and norepinephrine), which are released from the adrenal medulla, to:

1. Constrict the blood vessels in the skin, GI tract and kidneys
2. Dilate the blood vessels in the skeletal muscles and coronary arteries
3. Sweat
4. Increase the heart rate and contractility
5. Increase the rate and depth of breathing
6. Dilate the pupils

Hormonal Compensation

Mediated through the sympathetic nervous system, humoral compensation begins. The **anterior pituitary** releases ACTH, which causes a release of mineralocorticoids and glucocorticoids. The mineralocorticoids balance the sodium and water levels. The glucocorticoids regulate the metabolic function of the body through the stress response. Cortisol sensitizes the muscle of the arteriole to the effects of catecholamines.

The **posterior pituitary** releases ADH, causing vasoconstriction and renal retention of water.

The **kidneys**, which are flow dependent, also sense the decreased blood pressure. The kidneys release renin in response, which then stimulates the angiotensin and aldosterone systems. These hormones cause:

- Retention of sodium and water
- Increased blood volume in the major blood vessels because of water retention and vasoconstriction of the smaller blood vessels

- Decreased urine volume and sodium excretion
- Increased potassium excretion and increased urine osmolarity

Chemical Compensation

Hypoxemia and cellular hypoxia cause an increase in respiratory depth and rate. The acid-base balance is disturbed with the “blowing off” of CO₂, which leads to respiratory alkalosis. The combination of hypoxemia and alkalosis adversely affects the level of consciousness.

Progressive Stage of Shock

In this stage of shock, previously helpful compensatory responses are no longer effective. Severe hypoperfusion to all organ systems causes multi-organ dysfunction syndrome (multi-system organ failure). The microcirculation loses the ability to autoregulate blood flow, leading to decreased blood volume returning to the central blood vessels. This causes further organ hypoperfusion.

Refractory Stage of Shock

This final and irreversible stage reflects the very last part of a patient’s life. The cellular and organ destruction has been so severe that death is inevitable.

Essential versus Non-essential Organs

The body long ago developed a priority list for scant amounts of blood. On the top of the list:

- Brain
- Heart
- Lungs

These organs will receive the most blood possible during shock through stimulation of the beta receptors, which causes vasodilation.

The other organs of the body, such as the skin and gut, have primarily alpha-receptors, which when stimulated cause vasoconstriction. They are considered to be “non-essential organs.”

Organ-Specific Effects of Shock

Brain - Essential Organ

Beta adrenergic stimulation dilates cerebral vessels to attempt to maintain enough flow for a MAP of 50. Late in shock, the vasomotor center fails to recognize and respond to sympathetic stimulation. Early symptoms of hypoperfusion are irritability and agitation, replaced by unresponsiveness in late stages.

Heart - Essential Organ

In all forms of shock except cardiogenic shock, the myocardium experiences a protective flow. Autoregulation maintains coronary flow as long as arterial pressure does not fall below 70 mm/Hg. The deterioration of heart function makes shock irreversible.

All other organs are considered biologically expendable.

Skeletal Muscle, Fat, Skin

Vasoconstriction from alpha receptor stimulation results in muscle weakness, cramping, and fatigue. The skin becomes cool; its color ashen to cyanotic. The potential for skin breakdown is enormous.

Kidneys

The low blood pressure is seen as a decreased glomerular filtration rate (GFR) by the kidney. In order to increase flow, the kidneys activate the renin-angiotensin-aldosterone compensatory mechanism. Metabolic acidosis created by cellular increases of lactic acid is perpetuated by the kidneys’ inability to break down and excrete lactic acid.

Lungs

Hyperpnea occurs as a compensatory response to sympathetic stimulation, hypoxia, and metabolic acidosis. The increased respiratory rate increases pulmonary muscle oxygen consumption. Coupled with primary damage from centrally mediated chemicals to pulmonary capillary endothelial cells, increased capillary permeability results in interstitial and intra-alveolar edema and decreased pulmonary compliance. Resultant decreased ventilation and impeded gas exchange further decrease oxygen delivery to cells.

Mesentery

In early stages of shock, there is a marked decrease in blood flow to the gut manifested by nausea, vomiting, and hypoactive bowel sounds. Later, intestinal damage and necrosis by digestive enzymes cause damage to the protective mucosal barrier. Bacteria and toxins are released into the bloodstream. Hypoperfusion to the intestines also enhances the formation and absorption of endotoxins released from native gram negative bacteria. When released, these endotoxins cause extensive vascular dilatation, greatly increasing cellular metabolism despite inadequate oxygen and nutrients to cells.

Liver

The liver filters and detoxifies drugs, metabolites, and coagulation products. The liver also stores glucose as glycogen. The metabolic rate of the liver is very high with consumption of large quantities of oxygen and nutrients. In shock, the catecholamines stimulate liver activity. Glucose is made available to the cells, which are unable to use it, resulting in hyperglycemia. Hepatic ischemia results in a decrease in its metabolic and detoxification functions. Loss of clotting factors induce coagulopathies such as DIC.

Pancreas

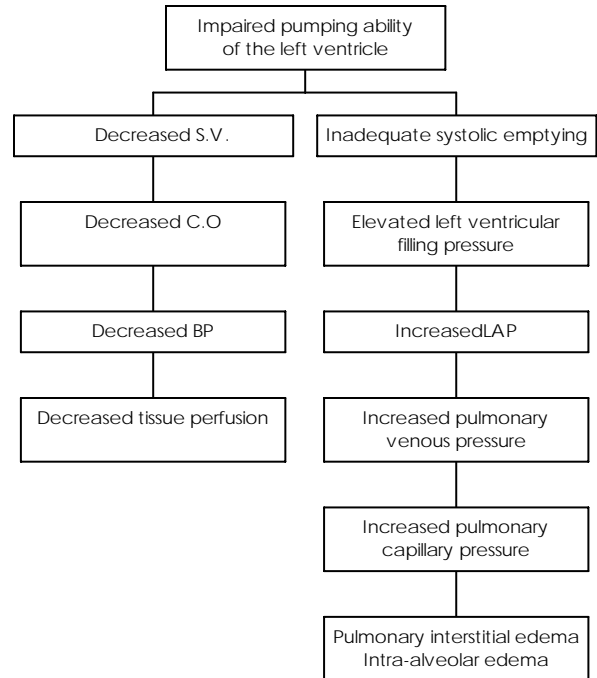
Shock induces the release of amylase and lipase into the circulation. A chemical called myocardial depressant factor (MDF), released from the pancreas, decreases myocardial contractility.

Cardiogenic Shock

Cardiogenic shock is caused by inadequate myocardial contractility from acute myocardial infarction, coronary artery disease, or mechanical factors (valvular regurgitation, low output syndrome, arrhythmias).

Pathophysiology of Cardiogenic Shock

In cardiogenic shock, the left ventricle has been injured in some way, leading to impaired pumping.



Because the pumping is ineffective, less blood is pushed out with each heartbeat, leading to a decreased stroke volume*. The heart rate increases to compensate for a low cardiac output and blood pressure, but will eventually be insufficient to compensate for the decreased stroke volume. The tissues begin to be inadequately perfused.

The impaired pumping also leads to less blood being pushed from the ventricle during systole. The left ventricle gradually fills with more and more blood, causing an elevated pressure within the LV and left atrium. This pressure “backs up” into the pulmonary vasculature, causing an increased pulmonary capillary pressure.

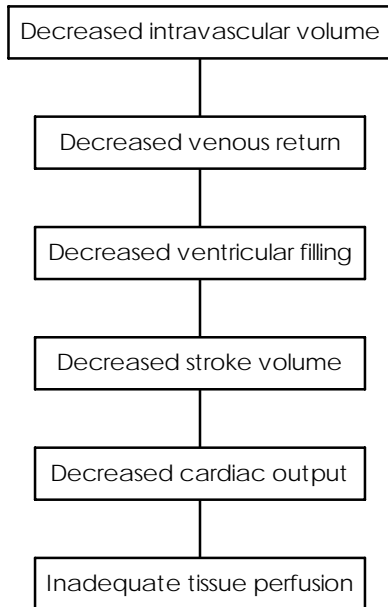
* Stroke volume = the amount of blood pumped out of the left ventricle with each contraction.

Hypovolemic Shock

In hypovolemic shock, there is a critical depletion of intravascular volume from hemorrhage (most common), plasma loss due to burns, dehydration, traumatic shock due to blood loss and major tissue damage.

Pathophysiology of Hypovolemic Shock

The pathophysiologic process of hypovolemic shock is straight-forward. Blood and/or fluids have left the body, causing a decreased amount of volume in the blood vessels.



Venous return is decreased because of the lack of fluid in the vascular space, causing decreased ventricular filling. The ventricles do not have as much blood as normal to pump out, so the stroke volume is decreased.

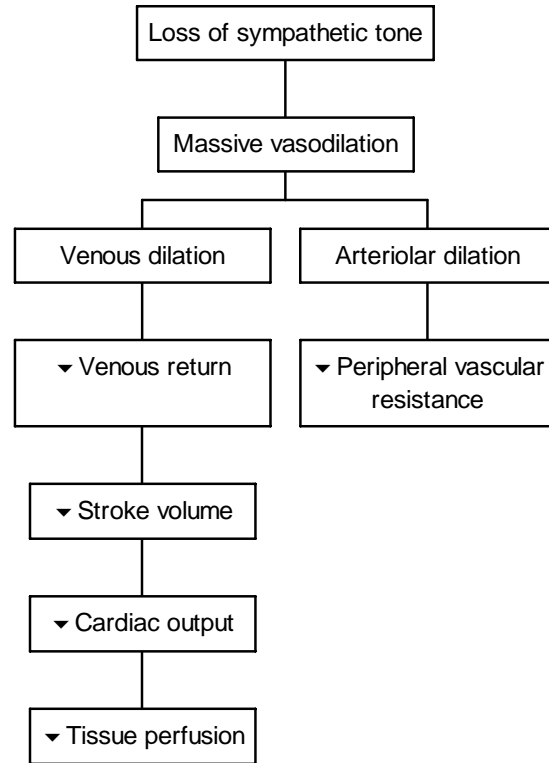
The heart rate will increase to compensate for the diminished stroke volume and resulting poor cardiac output and blood pressure. Eventually, if the fluid or blood loss continues, the heart rate will not be able to compensate for the decreased stroke volume.

The end result of hypovolemic shock is inadequate tissue perfusion.

Neurogenic Shock

Neurogenic shock is caused by the loss of sympathetic control (tone) of resistance vessels, resulting in the massive dilatation of arterioles and venules. Neurogenic shock can be caused by general or spinal anesthesia, spinal cord injury, pain, and anxiety.

Pathophysiology of Neurogenic Shock



In neurogenic shock, there has been an insult to the nervous system so that impulses from the sympathetic nervous system (the fight or flight response) cannot maintain normal vascular tone or stimulate vasoconstriction.

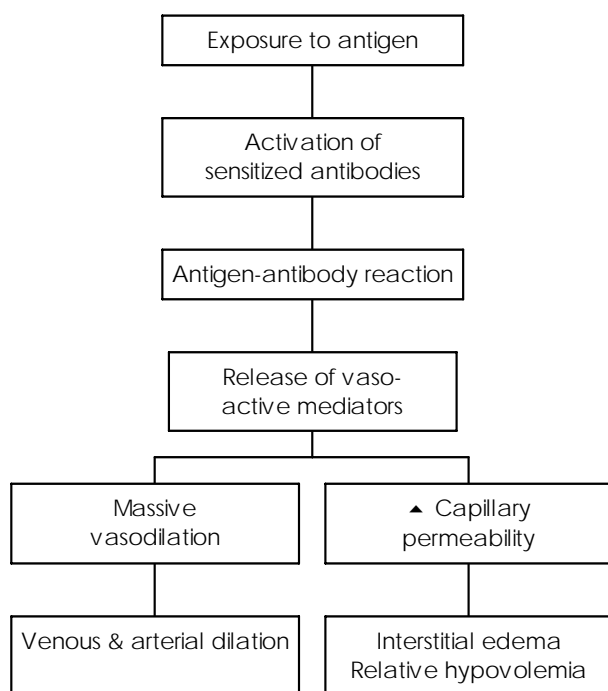
The lack of SNS stimulation causes a massive venous and arterial vasodilation. On the venous side, blood pools in the distensible veins and does not return to the larger veins. Because of this pooling, there is a diminished amount of blood that returns to the heart. Stroke volume, cardiac output, and blood pressure all fall.

On the arterial side, there is decreased peripheral vascular resistance, which actually helps the heart to pump with less energy. The drawback is that with decreased peripheral pressure, there is not the driving force to get blood, oxygen, and nutrients to the cells. This also causes a small degree of arterial blood pooling, which decreases the amount of blood returning to the heart.

Anaphylactic Shock

Shock due to the severe allergic antigen-antibody reaction to substances such as drugs, contrast media, blood products, or insect or animal venom is called anaphylactic shock. Food products such as seafood, nuts, peanuts, peanut butter, and MSG can also cause anaphylactic shock.

Pathophysiology of Anaphylactic Shock



The immune system goes “haywire” in anaphylactic shock in an extreme allergic reaction. At some point, the individual is exposed to the substance and develops antibodies against it. On subsequent exposure to the substance (the antigen), these antibodies will aggressively bind to the antigen, forming an antigen-antibody complex. This complex causes the release of chemicals that cause vasodilation (in particular, histamine).

Both veins and arteries vasodilate, leading to decreased blood returning to the heart. The capillaries become permeable to nearly everything, allowing fluids, proteins, and sometimes blood to pass through into the interstitial space. This causes massive interstitial edema. The vasodilation and fluid sequestration in the interstitium causes a relative hypovolemia.

Septic Shock

Sepsis is a condition that occurs in many critically ill patients. Sepsis is the systemic response to infection. Many types of organisms can cause sepsis, including gram-negative bacteria, gram-positive bacteria, and fungi. The infections can occur anywhere in the body; urinary tract infections are probably the most common cause of sepsis. Septic shock is said to occur when the sepsis has progressed to the point where it is affecting many organ systems.

Pathophysiology of Septic Shock

The immune and inflammatory response begins to try to combat the organism that is causing an infection. The body releases multiple chemicals into the blood stream, including cytokines, vasodilators, complement factors, and free radicals. In septic shock, this response is not adequate to eliminate the infection and actually causes increased damage. The organism itself also releases substances called endotoxins or exotoxins, which further harm the organs and tissues.

The combination of these chemicals and toxins cause: (1) peripheral vasodilation – interstitial edema and decreased blood return to the heart, and (2) decreased ability of the cells and tissues to take up oxygen and nutrients.

The heart tries harder and harder to get oxygen and nutrients to the cells by increasing the heart rate and contractility initially, sometimes driving the cardiac output twice to three times its normal amount.

Eventually, however, the immune response and compensatory mechanisms may not be enough to combat the infection and resulting cellular destruction. The patient may develop multi-organ dysfunction syndrome (MODS); AKA multi-system organ failure (MSOF).

Conclusion

Patients with a wide variety of problems can develop shock. Knowing the underlying pathophysiology may help guide you in assessing and managing the care of the patient with cardiogenic, hypovolemia, and distributive types of shock.

Resources

1. Barone, J., and Snyder, A. (1991). Treatment strategies in shock: Use of oxygen transport measurements. Heart and Lung, 20(1), 81-86.
2. Rice, V. (1992). Shock, a clinical syndrome: An update. Part 1: An overview of shock. Critical Care Nurse, 11(4), 20-27.
3. Rice, V. (1992). Shock, a clinical syndrome: An update. Part 2: The stages of shock. Critical Care Nurse, 11(5), 74-82.
4. Rice, V. (1992). Shock, a clinical syndrome: An update. Part 3: Therapeutic Management. Critical Care Nurse, 11(6), 34-39.
5. Rice, V. (1992). Shock, a clinical syndrome: An update. Part 4: Nursing care of the shock patient. Critical Care Nurse, 11(7), 28-38.

Recommended Reading

1. Brozene S, Russell SS. (1999). *Core Curriculum for Medical-Surgical Nursing*, 2nd ed. Academy of Medical-Surgical Nurses, Janetti NJ.
2. Phipps WJ, Sands JK, Marek JF, eds. (1999). *Medical-Surgical Nursing: Concepts & Clinical Practice*, 6th ed. St. Louis: Mosby, Inc.
3. Seidel HM, Ball JW, Dains JE et al, eds.(2002) *Mosby's Guide to Physical Examination*, 5th ed. St. Louis: Mosby, Inc.
4. Stillwell, S. (2002). *Mosby's Critical Care Nursing Reference*. 3rd ed. St. Louis, Mo: Mosby/Elsevier.
5. Smeltzer SC, Bare BG, eds. (2002) *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 10th ed. Philadelphia: Lippincott William and Wilkins.
6. Wiegand, D.J.L. & Carlson, K.K. (eds.) (2005). *AACN Procedure Manual fro Critical Care*. 5th ed. Philadelphia: Elsevier.

Directions for Submitting Your Post Test for Contact Hours

To obtain a certificate of completion for this home study program, please complete the post-test on the next page.

HCMC, MVAMC & Regions Hospital Employees

If you are an employee of HCMC, MVAMC, or Regions Hospital, simply send the post-test to TCHP for processing.

Paid Participants

If you are not an employee of one of the TCHP hospitals, please send the post-test to TCHP with a check for \$10.00. Please make check payable to **Regions Hospital** and mail to:

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Shock & Infection in Critical Care Primer Post-Test

Name _____

Date Completed _____

HCMC, MVAMC, or Regions Hospital employees:

Hospital _____ Unit _____

Paid Participants: _____

Street address

City

State

Zipcode

- 1) The sympathetic nervous system will do all of the following actions in response to a low cardiac output **except**:
 - a) increase cardiac rate and contractility
 - b) constrict the pupils
 - c) dilate blood vessels in the skeletal muscles and coronary arteries
 - d) sweat
- 2) Which organ is considered essential in relation to blood supply in the shock states?
 - a) gastrointestinal tract
 - b) kidneys
 - c) heart
 - d) lungs
- 3) The two pathophysiologic processes that occur in cardiogenic shock are:
 - a) anoxia and decreased tissue perfusion
 - b) decreased stroke volume and inadequate systolic emptying
 - c) low cardiac output and high urine output
 - d) pulmonary edema and decreased stroke volume
- 4) What is the most common cause of hypovolemic shock?
 - a) dehydration
 - b) burns
 - c) hemorrhage
 - d) vomiting
- 5) The massive vasodilation that occurs in neurogenic shock results in:
 - a) venous dilation
 - b) arteriolar dilation
 - c) decreased cardiac output
 - d) all of the above
- 6) What of the following will NOT cause anaphylactic shock?
 - a) a **first** bee sting
 - b) blood products
 - c) peanut butter
 - d) contrast media
- 7) The most common cause agent of septic shock is:
 - a) upper respiratory infection
 - b) urinary tract infection
 - c) central line infection
 - d) none of the above



Evaluation: Shock and Infection Critical Care Primer

We'd appreciate it if you could take a moment to complete the evaluation for this program. Thank you!

1. Who do you work for? _____

2. How did you hear about this program?

- brochure
- co-worker
- education department/clinical educator
- TCHP website
- Other _____

3. Were the objectives met?

Objective	Was the objective met?	
List the classifications of shock.	Yes	No
List the functions of the cell and the microcirculation.	Yes	No
Describe the stages of shock.	Yes	No
Describe three major mechanisms put into action to compensate for shock.	Yes	No
Define terms related to shock.	Yes	No

4. Would you recommend this program?

- yes no

5. Was this educational activity...

- too long
- too short
- just right

6. I can use the information from this activity in my job.

- strongly agree
- agree
- disagree
- strongly disagree

7. The information was....

- easy to understand
- difficult to understand