TCHP Education Consortium

You must print out your own course materials! None will be available at the class. Click on the link below to access:

www.tchpeducation.com/coursebooks/coursebooks_main.htm
If the link does not work, copy and paste the link (web page address) into your internet browser. Available **1** week prior to class.

Neurological Critical Care

January 16th, 2019
7:30 a.m. to 4:00 p.m.
Hennepin County Medical Center—
Room BL.320

Description/Learning Outcomes

Adults with neurologic disorders can be among the most difficult to assess and manage. The learning outcome is for the learner's ability to assess and care for patients with neurologic disorders such as increased intracranial pressure, spinal cord injury, cranial surgery, cerebrovascular disease, and seizures will be improved.

Target Audience

This class was designed for the novice critical care or telemetry nurse; however, other health care professionals are welcome to attend.

Before You Come to Class

It is highly recommended that you complete the **Shock in Critical Care Primer** prior to attending, if you have not already done so. It will be assumed that you have this knowledge. You can find the primer on the TCHP website; it will be attached to your pre-class materials: http://tchpeducation.com/coursebooks/preclass_docs.html

Schedule

7:30 - 7:45 a.m.	Registration	
7:45 - 9:15 a.m.	Assessment of the Patient with Neurological System Abnormalities	Beth Heather
9:15 - 9:30 a.m.	Break	
9:30 - 10:15 a.m.	Types of Head Injuries and Hematomas, Management of the Head Injured Patient	Beth Heather
10:15 - 10:45 a.m.	Cranial Surgery, Increased Intracranial Pressure, Routine Invasive Intracranial Pressure Monitoring	Beth Heather
10:45 - 11:45 a.m.	Herniation Syndromes, Brain Death	Beth Heather
11:45 - 12:30 p.m.	Lunch	
12:30 - 1:00 p.m.	SIADH/DI	Donna Lindsay
1:00 - 1:30 p.m.	Seizures	Donna Lindsay
1:30 – 2:30 p.m.	Spinal Cord Injury	Donna Lindsay
2:30 - 2:45 p.m.	Break	
2:45 - 4:00 p.m.	Cerebrovascular Disease	Donna Lindsay

Continuing Education Credit

For attending this class, you are eligible to receive:	*7.00 contact hours* **Criteria for successful completion:** All participants must attend the program and complete an online evaluation form to receive contact hours. Note that you must attend the ENTIRE activity to receive contact hours. The Twin Cities Health Professionals Education Consortium is an approved provider of continuing nursing education by the Wisconsin Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.
If you complete the primers for this class, you are eligible to receive additional contact hours.	Criteria for successful completion of primers: You must read the primer and complete the online post-test and evaluation.

Please Read!

- Check the attached map for directions to the class and assistance with parking.
- Certificates of attendance will be emailed to class participants once the online evaluation is completed.
- You should dress in layers to accommodate fluctuations in room temperature.
- Food, beverages, and parking costs are your responsibility.
- If you are unable to attend after registering, please notify the Education Department at your hospital or TCHP at 612-873-2225.
- In the case of bad weather, call the TCHP office at 612-873-2225 and check the answering message to see if a class has been cancelled. If a class has been cancelled, the message will be posted by 5:30 a.m. on the day of the program.
- More complete class information is available on the TCHP website at www.tchpeducation.com.

Finding HCMC Blue Building Lower Level Conference Room (BL.320)

900 South 8th Street, Minneapolis, MN 55404 (Blue Building)
Corner of South 9th Street and Chicago Ave. for Parking—can enter ramp from 8th or 9th

Finding the classroom <u>from Outside</u> the Building:
Enter the main entrance of HCMC "B" (Blue) building from South 8th Street (directly across the street from the Parkside Professional Building). Once inside the door, take a right and head towards the information desk. Turn left and go past the gift shop and coffee stand to the open stairway on your right. Take the stairs to the lower level. Turn to your right at the bottom of the stairs; go past the vending machines until you see a blue and white sign for the classroom (classroom is on your left).

***Finding the classroom <u>from the Hospital Parking 2 Ramp</u>:**
Take the ramp elevators to the lower level. Follow the signs to the hospital. Follow the hallway past the stairway and vending machines. You will see a blue sign for the classroom ahead of you (classroom is on your left).

Driving Directions to HCMC:

From the Northeast:
Take 35W south to Exit 17C (Washington Avenue). Turn right onto Washington. Follow Washington Avenue to Chicago Avenue and turn left. Take a left onto 9th street. Turn left again to enter the Hospital Parking 2 Ramp. Take the ramp elevator to the lower level and follow the instructions above.*

From the Northwest: Take I-94 east to exit 230 (4th Street). Follow 4th Street through downtown to Chicago Avenue and turn right onto Chicago Avenue. Follow Chicago to 9th Street and turn left. Turn left again to enter the Hospital Parking 2 Ramp. Take the ramp elevators to the lower level and follow the instructions above.*

From the East: Take I-94 W to exit 234B (7th Street). Follow 7th Street and turn left on Chicago Avenue. Follow Chicago to 9th Street and turn left. Turn left again to enter the Hospital Parking 2 Ramp. Take the ramp elevators to the lower level and follow the instructions above.*

From the South: All downtown exits are closed from northbound 35W. Consult www.mndot.gov/35w94 for information about closures and alternate routes. Park in the Hospital Parking 2 Ramp; entrances off of 8th or 9th Street. Take the ramp elevators to the lower level and follow the directions above.*

From the West: Take 394 east to exit 9B (6th Street). Follow 6th Street to Chicago Avenue; turn right onto Chicago. Take Chicago Avenue to 9th Street and turn left. Turn left again to enter the Hospital Parking 2 Ramp. Take the ramp elevators to the lower level and follow the directions on the previous page.*

Public transportation is another option for getting downtown. For bus schedules and information, go to www.metrotransit.org. **Light Rail Transit to HCMC:** Exit at the US Bank Stadium Station. Walk south west down Kirby Pucket Place until it becomes Chicago Ave. Turn left on 8th St. and enter Blue Building on you Left. See Lower Level Map attached for location of BL.320.

Parking:
There are various options for parking around HCMC, but we suggest you park in the Hospital Parking 2 Ramp (see HCMC Campus Map). Directions and maps guide you to and from this ramp. Meters are available around the hospital and vary in price. Check www.mplsparking.com for rates.

Parking rates are subject to change without notice. The program coordinator will have a limited number of discount coupons for the Hospital Parking 2 Ramp available for $7.00. You must pay with cash or check in the exact amount for the discount coupon—change is not available.

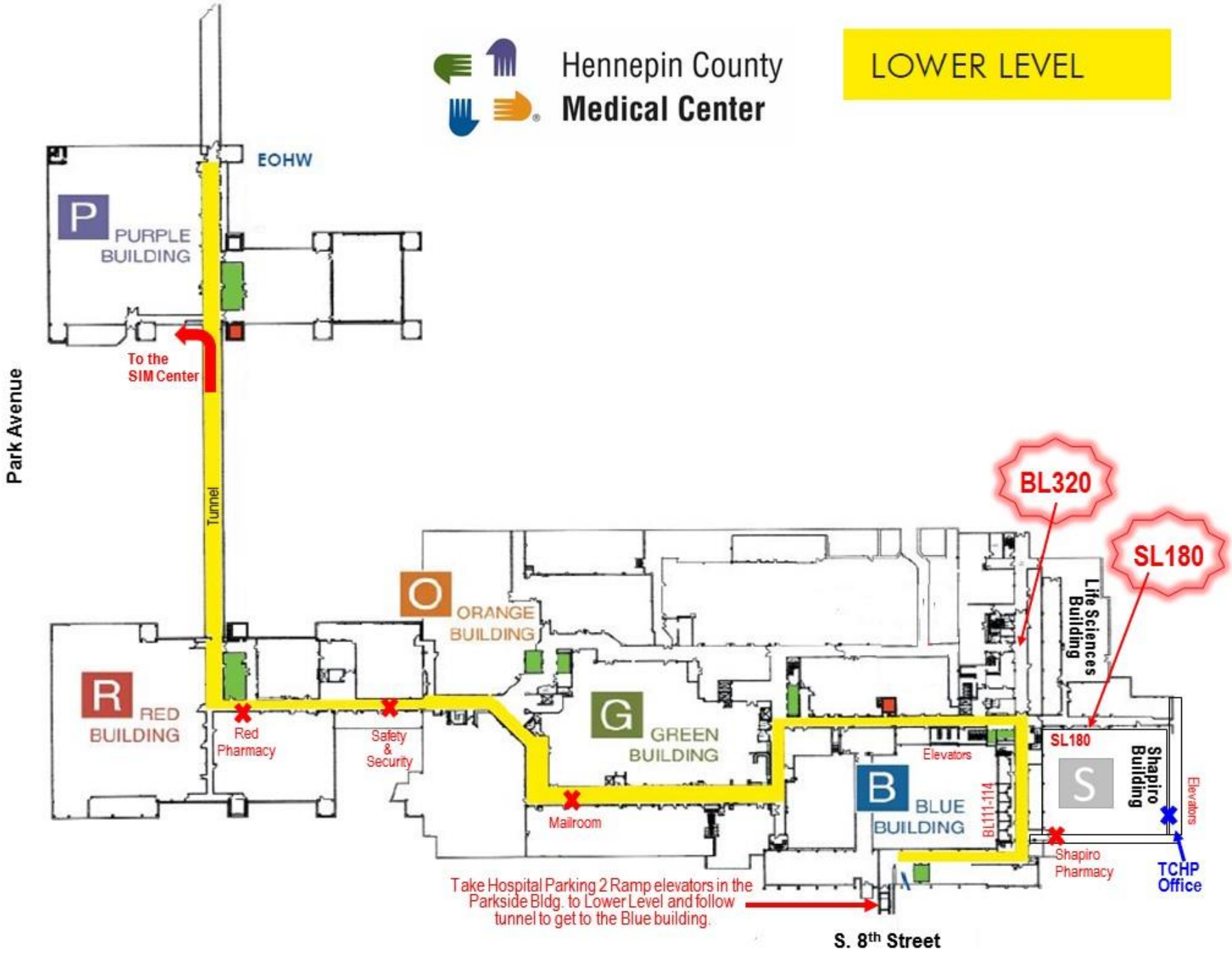
HennepinHealthcare | HCMC CAMPUS

LEGEND

- Entrance
- Parking Entrance
- Visitor Parking
- Valet Parking
- Cafeteria
- Metro Transit

Hennepin County
Medical Center

LOWER LEVEL



TCHP Education Consortium

This home study is pre-reading for a class. Please complete before class time. If contact hours are desired, follow instructions at the end of the packet.

Shock in Critical Care Primer

Introduction

Introduction/Learning Outcomes

Failure of the normal regulatory mechanisms in the body can lead to rapid and profound shock. The learning outcome of this home study is for learners to self-report an improvement in their knowledge base related to the pathophysiology of cardiogenic, hypovolemic, anaphylactic, neurogenic, and septic shock.

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.

Disclosures

Requirements for successful completion of this educational activity:
In order to successfully complete this activity you must read the home study and complete the online post-test and evaluation.

Conflicts of Interest
It is the policy of the Twin Cities Health Professionals Education Consortium to provide balance, independence, and objectivity in all educational activities sponsored by TCHP. Anyone participating in the planning, writing, reviewing, or editing of this program are expected to disclose to TCHP any real or apparent relationships of a personal, professional, or financial nature. *There are no conflicts of interest that have been disclosed to the TCHP Education Consortium.*

Expiration Date for this Activity:
As required by ANCC, this continuing education activity must carry an expiration date. The last day that post tests will be accepted for this edition is **December 31, 2020**—your envelope must be postmarked on or before that day.

Planning Committee/Editors*

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*Denotes expert for current edition

Contact Hour Information

For completing this **Home Study and evaluation**, you are eligible to receive:	**1.50 contact hours** (see information that follows) ***Criteria for successful completion:*** You must read the home study and complete the online post-test and evaluation. The Twin Cities Health Professionals Education Consortium is an approved provider of continuing nursing education by the Wisconsin Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.

Please see the last page of the packet for information on submitting your post-test and evaluation for contact hours.

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: (1) an efficient cardiac pump to circulate blood (2) an adequate blood volume, and (3) a healthy vascular bed where gas, nutrients and waste are exchanged. 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 circulating blood volume
- **Distributive** (neurogenic, anaphylactic, and septic): a dysfunctional vascular bed

The cascading events of shock begin with an insult, leading to inadequate oxygen transport and cellular dysfunction, proceeding to tissue and vascular disturbances, and ending 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 (which can also be obtained as the SVO2 reading from a Swan-Ganz catheter) helps infer the amount of oxygen being used at the cellular level 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.

Other components that affect carried and released oxygen are pH, temperature and CO2. This relationship can be mapped with the oxyhemoglobin dissociation curve.

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 that surrounds the organelles; site of most cellular activity.

4. **Organelles:** "small cellular organs" or 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) which is then utilized by the cells 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.

Arteries have strong, smooth muscle walls that direct blood to capillary beds and control the 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, are made by the sympathetic innervation of these vessels.

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 for return to the heart and lungs.

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) and (2) hormonal, (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^+ ions, 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 no clinical signs or symptoms except elevated lactate levels.

In the initial stage of shock, the cell switches from aerobic metabolism to anaerobic metabolisn, which causes decreased energy production and increased lactic acid levels. Diminished blood flow to the microcirculation and sequestration of metabolic by-products reduces 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

Baroceptors located in the aorta and carotid bodies sense a decrease in the blood pressure. Messages are sent to the medullary vasomotor center that stimulate the sympathetic nervous system (SNS). 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 cardiac contractility
5. Increase the rate and depth of breathing
6. Dilate the pupils

Hormonal Compensation

Mediated through the sympathetic nervous system, hormonal compensation begins. The **anterior pituitary** releases ACTH, which causes a release of mineralocortocoids 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 arterioles to the effects of catecholamines.

The **posterior pituitary** releases anti-diuretic hormone (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 excessive "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, causing pooling in the periphery and 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 which organs are perfused first when blood volume or transport is insufficient. 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 cause 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 may eventually make 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 becomes ashen, then cyanotic. The potential for skin breakdown is enormous.

Kidneys

The kidneys view a low blood pressure as a decreased glomerular filtration rate (GFR). 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

Respiratory rate increases occur 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. The decreased ventilation and impeded gas exchange that result 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 the liver's metabolic and detoxification functions. Loss of clotting factors induce coagulopathies such as DIC.

Pancreas

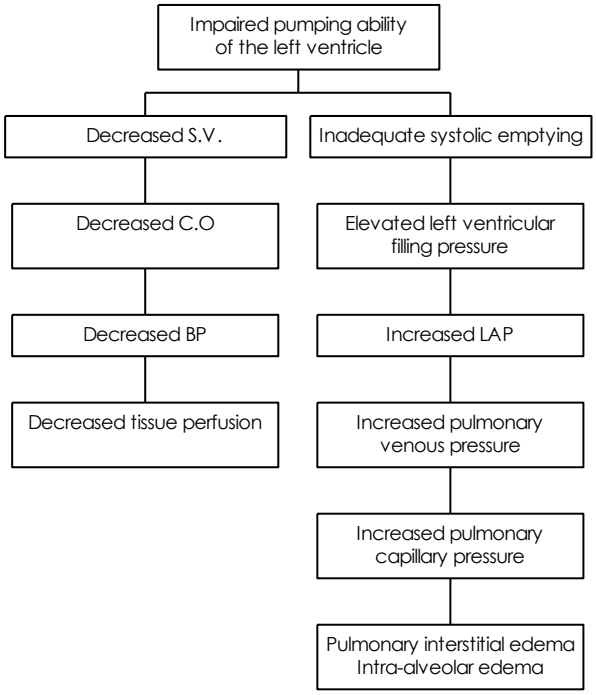
Shock induces the release of amylase and lipase into the circulation. A chemical called myocardial depressant factor (MDF), released from the pancreas, decreases myocardial contractility.

Cardiogenic Shock

Cardiogenic shock is caused by inadequate myocardial contractility from acute myocardial infarction, coronary artery disease, or mechanical factors (such as 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 and pulmonary edema.

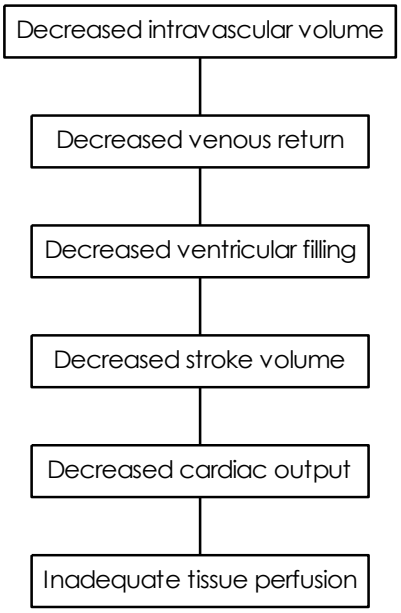
** Stroke volume = the amount of blood pumped out of the left ventricle with each contraction.*

Hypovolemic Shock

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

Pathophysiology of Hypovolemic Shock

The pathophysiologic process of hypovolemic shock is straight-forward. Blood and/or fluids have left the body (or vascular space), 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.

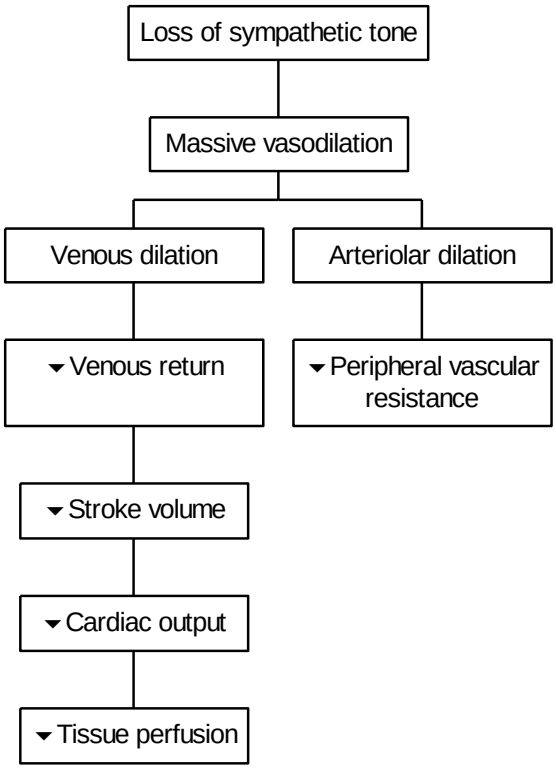
The heart rate will increase to compensate for the diminished stroke volume and resulting poor cardiac output and blood pressure. Eventually, if the fluid or blood loss continues, the heart rate will not be able to compensate for the decreased stroke volume.

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.

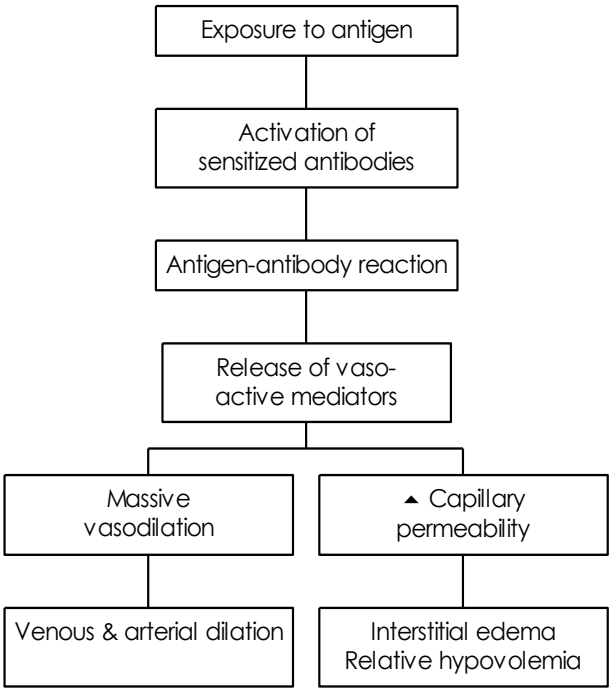
On the arterial side, there is decreased peripheral vascular resistance, which actually helps the heart to

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 leading to 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 enough to combat the infection and resulting cellular destruction. The patient may develop multi-organ dysfunction syndrome (MODS); acute kidney injury (AKI) and/or 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. Dellinger, R. (2013). Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2012. Critical Care Medicine www.ccmjournal.org, 41(2), 580-637.
2. Hochman, J., & Reventovicj, A. (2015). Clinical manifestations and diagnoses of cardiogenic shock in myocardial infarctions. In G.S. Saperia (Ed.), *UpToDate*. Retrieved from http://www.uptodate.com/home/index.html
3. Kemp, S. (2017). Pathophysiology of anaphylaxis. In A. M. Feldweg (Ed.), *UpToDate*. Retrieved from http://www.uptodate.com/home/index.html
4. Sonneville, R. (2013). Understanding brain dysfunction in sepsis. Annals of Intensive Care, 3(15), 1-11.

Recommended Reading

1. Alspach, J.G. (2006) *AACN Core Curriculum for Critical Care Nursing*. 6th ed. Philadelphia: Elsevier.
2. Seidel HM, Ball JW, Dains JE et al, eds. (2010) *Mosby's Guide to Physical Examination*, 7th ed. St. Louis: Mosby, Inc.
3. Stillwell, S. (2006). *Mosby's Critical Care Nursing Reference*. 4th ed. St. Louis, Mo: Mosby/Elsevier.
4. Cheever K.H. & Hinkle J.L. (2013) *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 13th ed. Philadelphia: Lippincott William and Wilkins.
5. Wiegand, D.J.L. & Carlson, K.K. (eds.) (2011). *AACN Procedure Manual for Critical Care*. 6th ed. Philadelphia: Elsevier.

Directions for Submitting Your Post Test for Contact Hours

1. Go to the TCHP website Home Study page to get the **electronic** post-test (no password is required): http://tchpeducation.com/homestudies/homestudies.html

2. The **electronic** post-test will take you to a quick and easy Survey Monkey post-test and evaluation. Fill in your answers and click "done." Your certificate of completion will be sent to you in a week or 2 ***(Note: This process is not automatic so do not expect an immediate return of a certificate of completion).***

Please Note: Survey Monkey does not save your work so plan to do the post-test all the way through.

If you are having difficulty with Survey Monkey, please contact tchp@hcmed.org for help.

Be sure to complete all the information requested on the post-test and evaluation. If required items are skipped, your post-test will automatically be classified as *Incomplete* in the survey system. Plan to complete the entire post-test in one visit as your responses are not saved in the system. The date recorded on your certificate of completion will be the date that your home study is received by TCHP. **Any materials received with a time stamp after the expiration will be discarded.**

TCHP is not responsible for lost or misdirected mail/email. We suggest that you print out your post-test before submitting to keep a copy for your records as the post-test will not be returned with the certificate of completion.

TCHP Consortium Hospital Employees

If you are an employee of a TCHP Consortium hospital (consult www.tchpeducation.com if you are unsure), your certificate of completion will be sent to you via work email. It cannot be sent to your home.

Paid Participants

If you are not an employee of one of the TCHP hospitals, you will need to submit a payment of $15.00 to TCHP in order to have your home study processed. If submitting a check, please make it payable to **TCHP Education Consortium** and indicate which home study you are paying for. You can also pay online using PayPal (see the website at www.tchpeducation.com under home studies for information). If you received this packet as pre-reading for a class you are taking, the processing fee is included in the course tuition.