NWC EMS System

Pathophysiology & Management of shock

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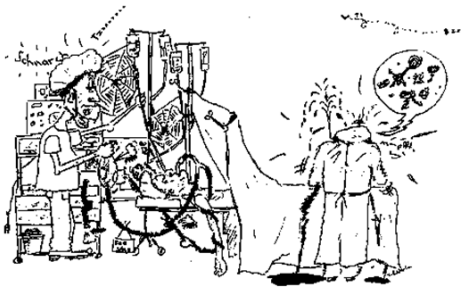
EMS Administrative Director



Reading assignment: **Text Vol.1;** pp.205-212; 232-236
Assumed knowledge:
Genetics & Familial dx: pp 226-228; disease risks: pp. 228-232

SOP: Shock

UNIT TERMINAL OBJECTIVE

Upon the completion of this unit, the participant will be able to integrate pathophysiological principles and assessment findings to formulate a field impression and implement a treatment plan for the patient with shock.

OBJECTIVES

1. At the completion, the paramedic will independently do the following with at least an 80% degree of accuracy:
2. Discuss hypoperfusion.
3. Differentiate the etiologies of cardiogenic, hypovolemic, neurogenic, anaphylactic and septic shock.
4. Describe the epidemiology, including the morbidity/ mortality and prevention strategies, for shock.
5. Discuss the anatomy and physiology of the cardiovascular system as it relates to perfusion and shock.
6. Discuss the pathophysiology of shock.
7. Discuss the general assessment findings associated with shock.
8. Define shock based on aerobic and anaerobic metabolism.
9. Describe the incidence, morbidity, and mortality of shock.
10. Describe the body's physiologic response to changes in perfusion.
11. Describe the effects of decreased perfusion at the capillary level.
12. Discuss the cellular ischemic phase related to shock.
13. Discuss the capillary stagnation phase related to shock.
14. Discuss the capillary washout phase related to hemorrhagic shock.
15. Relate pulse pressure changes to perfusion status.
16. Relate orthostatic vital sign changes to perfusion status.
17. Differentiate between compensated and decompensated shock.
18. Differentiate between the normotensive, hypotensive, or profoundly hypotensive patient.
19. Synthesize assessment findings and patient history to form a field impression for the patient with shock.

Shock pathophysiology – all forms

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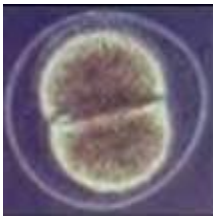
Shock syndrome defined

Nothing brings the body's machinery to a grinding halt quite like the process that occurs when essential nutrients and metabolic fuel like oxygen fail to be delivered to cells to meet their demands at the moment. While the word, shock, may conjure up mental images of patients who are cool, sweaty, hypotensive, and tachycardic, clinical signs can vary remarkably based on the cause or etiology of the problem. To understand the essence of shock, one needs to consider what is happening at the cellular level.

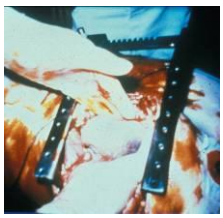
All body cells require a constant supply of fuel in the form of oxygen and other nutrients like glucose. They cannot storehouse O_2 for even a minute when breathing room air. This just in time supply is provided by the constant passage of oxygenated blood through the body's tissues in a process called **perfusion**.



The simplest definition of shock can begin with two words, **cellular hypoxia**. This hypoxia usually stems from a sustained perfusion deficit where blood flow is restricted despite compensatory adjustments. If unchecked, the perfusion failure will end in eventual organ failure. In a more complete definition, shock is a metabolic condition resulting from a sustained perfusion deficit leading to **oxygen debt (cellular hypoxia), anaerobic metabolism, cellular membrane dysfunction, fluid influx, and cellular death**. The common denominator in shock, regardless of cause, is a failure of the circulatory system to deliver the chemical substances necessary for cells to survive and to remove the waste products of cellular metabolism.

Factors necessary to maintain perfusion

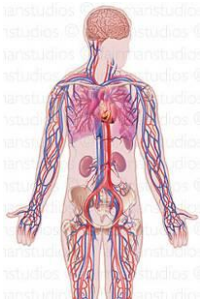
Given that perfusion is absolutely necessary to maintain cell function, understanding the components that contribute to adequate perfusion will provide insight into possible etiologies of shock.



Adequate pump: The heart must generate the power necessary to keep the vascular container filled and to move blood forward to meet body demands. It does this by generating a cardiac output to maintain circulation.

Circulating fluid: There must be sufficient blood volume to fill the vascular container plus the ability to carry O2 to the tissues, release it to the cells, and to remove waste products.

Intact vascular container: (pipes)



Resistance vessels (arterioles) and capacitance vessels (veins) conduct blood to and from the capillary beds for gas exchange. The vascular system must be intact and capable of regionalizing blood flow by responding to autonomic nervous system stimulation to change size and caliber to maintain a minimum mean arterial pressure (MAP). The vascular container cannot be too large for the volume of blood. Dilation of the vessels without volume compensation can result in shock.

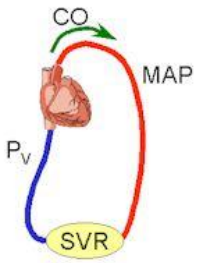
Causes of relative volume losses (maldistribution from vasodilation)

Anaphylactic shock
Neurogenic shock
Septic shock

Factors affecting pump performance

Mean arterial pressure (MAP) depends on:

- Cardiac output
- Systemic vascular resistance
- Central venous pressure

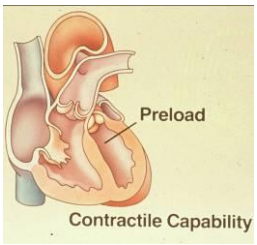


The **cardiac output (CO)** is the amount of blood the heart pumps in a given period of time and is a product of the stroke volume times the heart rate.

Stroke volume is the quantity of blood ejected with each contraction (ave. 70 mL). A normal adult heart rate ranges from 60 to 100 beats per minute (ave. 72-75 BPM). Thus, an adult cardiac output ranges from 4 to 7 liters per minute but can increase or decrease significantly with changes in contractile strength and/or heart rate.

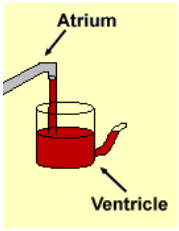
To understand the factors that affect stroke volume, think of the heart like any other pump. All pumps have inflow and outflow determinants that influence their performance. In the heart, these factors include preload, afterload, and myocardial contractility.

Preload - A pump must fill in order to squeeze anything out.



Preload is the end diastolic filling pressure or wall tension in the ventricle at the end of venous filling (diastole). Preload depends on the rate and duration of ventricular filling, ventricular compliance, venous tone, the total blood volume, and the amount of venous return. Normal preload pressures are 4-12 mmHg.

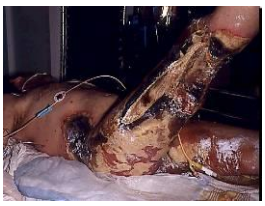
These pressures are clinically significant because they determine the amount of blood the ventricle will have to circulate during systole. **NO BLOOD IN MEANS NO BLOOD OUT!**



Ventricular volume will also influence myocardial fiber length or stretch.

Dr. Frank Starling postulated the correlation between myocardial stretch and contractility as **Starling's Law**:

Optimal stretch (preload) = optimal contractility (stroke volume) up to a certain point. To illustrate this concept, liken the heart to a rubber band. If it is barely stretched, there is very little contraction. If optimally stretched, there is a forceful snap back. If overstretched over time, contractility weakens.

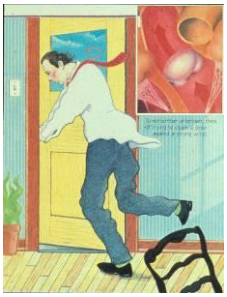


Preload can be adversely impacted by volume losses due to hemorrhage, excessive diaphoresis, vomiting/diarrhea, or third space losses such as those that occur with burns, ascites, or bowel obstructions. Venous dilation, and therefore preload reduction, occurs with hyperthermia, use of drugs like nitroglycerin, and with vasodilatory shocks (septic, neurogenic, and anaphylactic).

Obstructive shock: Preload is also influenced by intrathoracic and intrapericardial pressures. Mechanical obstruction of venous return to the right heart occurs with pericardial tamponade and tension pneumothorax. Preload to the left heart is markedly reduced in the presence of extensive pulmonary embolism.

Volume changes that increase preload occur following administration of IV fluids and are also associated with conditions that cause fluid retention such as heart failure (HF) and renal failure.

Afterload: Force the ventricle must pump against in order to eject blood.

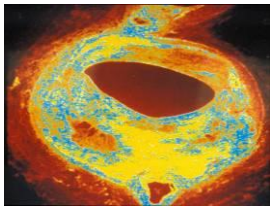


Ventricles cannot eject blood until they are able to generate more tension in their chambers than is present in the vessels into which they empty. These afterload pressures are determined by systemic and pulmonary vascular resistance and the degree of vasoconstriction.

Constricted or diseased arteries have smaller internal diameters and provide high resistance (afterload pressures). Dilated arteries provide little resistance (afterload) and allow for increased stroke volumes.

Right ventricle afterload: pressure in the pulmonary artery.
Left ventricle afterload: pressure in the aorta and systemic arterioles.

The elasticity of the aorta greatly affects afterload pressures. Resistance is high in patient with arteriosclerosis or atherosclerosis.

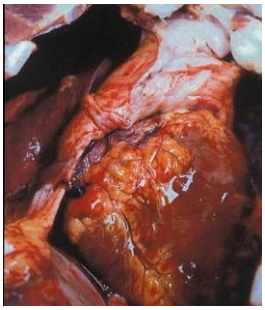


Afterload pressures are increased in hypovolemic or cardiogenic shock due to vasoconstriction and following administration of alpha stimulants such as epinephrine, norepinephrine or dopamine in high doses (greater than 10 mcg/kg/min).

Afterload is decreased in the presence of severe hypoxemia and low resistance or distributive forms of shock, e.g., neurogenic, anaphylactic, and septic. Vasodilating drugs like nitroglycerin in high doses, alpha or calcium blockers, ACE inhibitors and angiotensin II blockers reduce afterload pressures.

Myocardial contractility

The last determinant of stroke volume is inherent myocardial contractility, not influenced by preload or afterload pressures. This contractile strength is related to the isovolumetric contraction capacity of the heart muscle. **Reduced contractility is the primary cause of cardiogenic shock** and contributes to the late phase of any form of shock.



Cardiac contractility is determined by sympathetic nervous system activity, circulating catecholamines (epinephrine and norepinephrine) that enhance fiber shortening by acting on beta-1 receptors, the rate and rhythm of contractions, certain drugs (positive inotropes - beta-1 stimulants); the ionic environment (calcium, potassium levels), myocardial oxygenation, and the amount of functional myocardium.

Inotropes (make the heart contract more forcefully) commonly used by EMS personnel include epinephrine and dopamine. Additional drugs that increase myocardial contractility include calcium chloride 10%, digoxin, Isuprel (isoproterenol hydrochloride), milrinone, and norepinephrine bitartrate (Levophed).

Factors that decrease contractility

- **Hypoxemia**, resulting from ventilation/perfusion abnormalities in the lung, occurs in early shock and decreased contractility. In late shock it worsens, and becomes "malignant" or irreversible because of the low perfusion state.
- **Acidosis** results from anaerobic metabolism with release of lactate and pyruvic acids accompanied by decreased renal perfusion and accumulation of organic acids. Myocardial ischemia develops when arterial pressure falls and further decreases contractility. This situation is compounded in the patient with pre-existing coronary artery disease.
- **Drugs**: Negative inotropes like barbiturates, beta blockers, calcium blockers, ganglionic blockers, and lidocaine
- **Electrolyte imbalances**
- **Myocardial remodeling** as seen with chronic volume overload or following acute myocardial infarction.
- **Myocardial depressant factor** (MDF) is thought to be a low molecular weight peptide released from damaged cells in a hypoxic pancreas which markedly decreases contractility and compounds shock.

Heart rate

The other side of the equation determining cardiac output is heart rate (HR)**.** As a general rule, an increased HR will increase CO by up to three times normal. At high rates (≥ 150) the filling time (diastole) is compromised so the ventricle fills with less blood and stroke volume decreases so that CO falls.

The intrinsic HR is a function of the excitability and rhythmicity of pacemaker cells (SA node, AV node etc.). The heart's electrical conduction system has extensive neural regulation from the autonomic nervous system.

Although the heart initiates its own beat (automaticity), the autonomic nervous system can accelerate or slow the HR. The two divisions are both always on and usually balance to give an average HR of 60 to 100 beats per minute (BPM).

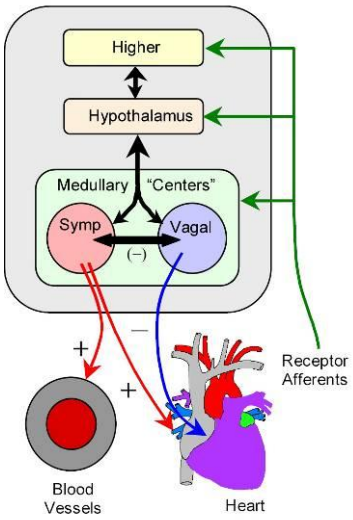
However, if the body senses an internal or external threat or anger, the sympathetic side dominates. In states of rest or sleep the parasympathetic dominates.

Sympathetic NS (SNS) activation of B-1 receptors produces an increase in HR (+ **chronotropic effect**), increase in contractile force (+ **inotropic effect**), and increases the speed of impulse conduction through the electrical conduction system (+ **dromotropic** response). The SNS can be likened to the heart's accelerator.

Stimulation of beta 2 receptors causes bronchodilation and vasodilation. Stimulation of alpha receptors causes intense vasoconstriction.

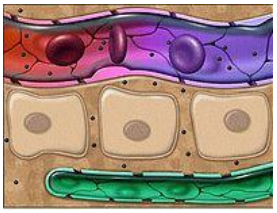
	Alpha	Beta
Heart		**Increased rate Increased force Speeds conduction**
Bronchioles	**Constricts**	**Dilates**
Arterioles	**Constricts**	**Dilates**

Parasympathetic NS (PNS) stimulation via the Vagus nerve heavily influences atrial pacemaker cells causing them to slow down. Think of PNS stimulation as the heart's brake.



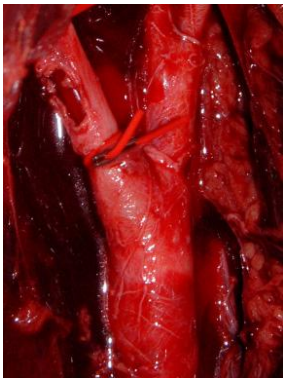
Factors affecting fluid volume

The body normally maintains a constant intravascular volume through neurogenic, endocrine, cardiovascular, microcirculatory, renal, and metabolic mechanisms. Hypovolemia can result from loss of blood plasma or fluid to the exterior of the body, or to the exterior of the vascular tree into body cavities or interstitial spaces resulting in decreased circulating volume and diminished venous return. The patient can suffer a relative hypovolemia if the size of the vascular compartment enlarges without any extra blood volume to fill it. This absolute or relative hypovolemia decreases venous return, thus decreasing preload and cardiac output.



Factors affecting vessels

Total peripheral resistance (TPR)



Since the circulatory system is a closed system, increasing either cardiac output or peripheral vascular resistance will increase blood pressure. Likewise, a decrease in cardiac output or a decrease in peripheral vascular resistance will decrease blood pressure. Arterioles are the resistance vessels. They can change diameter up to 5 fold.

TPR is a function of **blood viscosity** and the cross sectional area **(diameter)** + **length** of the vessel. Changes in vessel diameter will affect resistance. The calculated resistance is inversely proportional to the fourth power of the radius of the vessel.

There are a large number of factors influencing the diameter of vessels in the microcirculation, which ultimately determine resistance. Manipulation of these factors allows the system to tolerate or compensate for reduced CO.

Local auto-regulatory vascular control

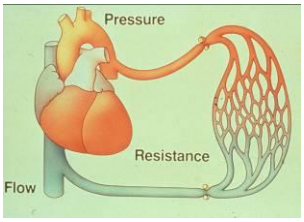
Vessels have an intrinsic ability to autoregulate tone and maintain blood flow over a wide range of perfusion pressures - independent of neurogenic or humoral influences. Different vascular beds vary in their capacity to auto-regulate flow but the cerebral, coronary and renal circulations are most potent.

The specific mediators of these local responses are not known, but they are most likely triggered by changes in osmolality, accumulation of metabolic waste and hypoxia resulting from local ischemia due to low perfusion states.

Brain (via SNS) auto-regulation protects cerebral tissues from low flow states - however, this ability is lost in the presence of hypoxia or severe hypercarbia (↑ CO_2).

Hemodynamics

Just because the patient has a blood pressure, does not mean that tissues are being perfused. This concept is explained by pressure, flow, and resistance relationships.



Blood flow = Pressure/resistance

Pressure = Flow X resistance

Resistance = Pressure/flow

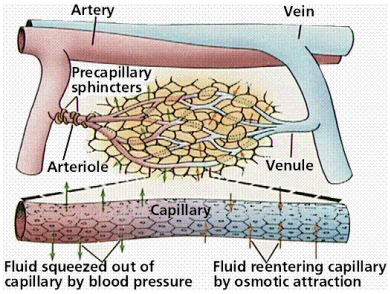
Another way of calculating this relationship is with the following equation:

$$F = \frac{P_A - P_V}{R}$$

An increase in resistance will decrease flow at any given perfusion pressure, in fact, a change in resistance (vessel diameter) is the primary means of blood flow regulation.

Cellular metabolism: normal to hypoperfused

Normal flow in the microcirculation

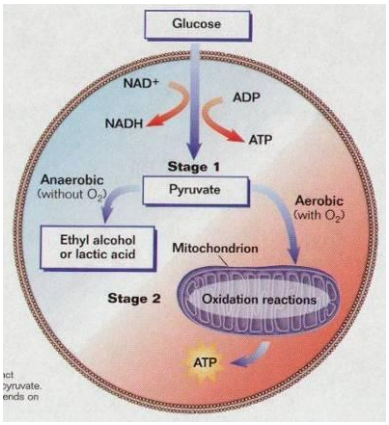


The microcirculation is composed of arterioles, capillaries and venules. There is a sphincter at the origin of the capillary between the arteriole and capillary (pre-capillary sphincter) and another at the end of the capillary between the capillary and venule, called the post-capillary sphincter. The arteriole component is concerned with homeostasis and is innervated by adrenergic (SNS) fibers that control muscular sphincters. These sphincters maintain peripheral vascular resistance and determine blood flow through the capillaries.

Each arteriole feeds a series of capillaries. Capillaries open in rotation on demand of cells adjacent to them. The opening of **precapillary sphincters** is facilitated by histamine secretion in response to local tissue conditions, such as acidosis and hypoxia. They open as more arterial blood is needed.

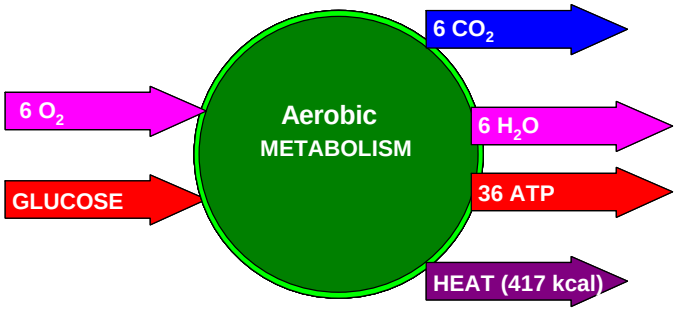
When the arteriole is widely opened flow is rapid, the pH drop is minimal, and the arterio-venous (AV) shunt is closed. Oxygen and waste products are exchanged across the capillary membrane based on hydrostatic and osmotic pressure gradients. The post-capillary sphincter opens when blood is to be emptied into the venule. Thus, blood flow to cells is regulated by peripheral resistance and pressure within the system.

Aerobic metabolism



The primary energy source for cells is glucose. Glucose must be broken down through a process called **glycolysis**. This step does not require oxygen. Glycolysis produces pyruvate but very little energy.

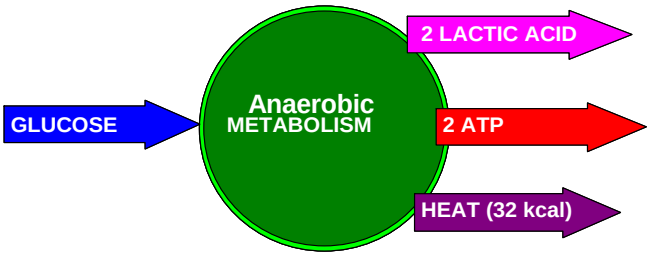
The second stage of metabolism is **aerobic**. In the presence of O_2, calcium (Ca^{++}) and ADP, the mitochondria of the cell (through the Krebs cycle) metabolizes pyruvate to produce CO_2, water, and 36 moles of ATP (adenosine triphosphate) per mole of glucose.



The cell uses ATP to maintain the **sodium/ potassium pump** at the cell wall membrane to regulate its intracellular water component. Oxygen consumption is not dependent on oxygen delivery under aerobic metabolism. When needed, cells can extract extra oxygen necessary for energy production.

Shock occurs when oxygen and nutrient delivery to cells throughout the body occurs at a rate below that of total O2 consumption (**oxygen debt**) *and* waste products (CO_2 and acids) are not effectively removed.

Cells start to change from aerobic to anaerobic metabolism.



Start connecting the dots…Causes of hypoperfusion:

- Inadequate pump
  - Inadequate preload
  - Inadequate cardiac contractile strength
  - Inadequate HR
  - Excessive afterload
- Inadequate fluid volume (absolute or relative)
- Inadequate container (container failure)
  - Dilated vessels without change in fluid volume
  - Leak in the vessels

Stages of shock & compensatory mechanisms

The stages of shock reflect the severity of disruption in tissue perfusion and the degree of cellular membrane damage. If precipitating factors are promptly reversed, compensatory mechanisms can usually restore perfusion. The longer a patient remains in shock, the longer vital organs are deprived of O_2. After cellular destruction begins, shock cannot be reversed and organs will fail.

Stages of shock

Initial stage (compensated/reversible)

Something occurs to cause a perfusion deficit with an early drop in cardiac output that alters cellular function. The body attempts to maintain hemodynamic stability through compensatory mechanisms and by neutralizing elevated lactate levels. Interrelated neural, hormonal, and chemical mechanisms restore cardiac output and perfusion to keep the circulatory system functioning at normal or near normal levels so there are no early clinical signs or symptoms.

Neural compensation - **homeostatic neuroreflexes**

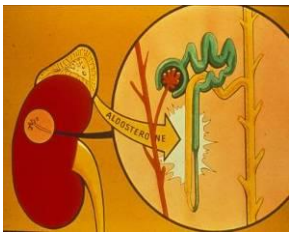
The **vasomotor center in the medulla** receives impulses from various receptor mechanisms in the body, which either suppress or stimulate neural tone in the sympathetic nervous system and adrenal glands in an attempt to stabilize the BP.

Types of peripheral receptors

Baroreceptors (pressure receptors in the aortic arch and carotid sinuses) are triggered by a decrease in cardiac output. The carotid sinuses respond to pressures of 60-180 mmHg. The aortic arch has a higher threshold and is less sensitive than the carotid bodies. Impulses travel to the medulla. The vasomotor center responds by increasing sympathetic and decreasing parasympathetic outflow. The SNS releases epinephrine and norepinephrine that increase HR and strength of contractions and cause venous then arterial vasoconstriction.

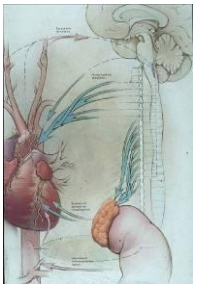
- **Chemoreceptors** detect hypoxia (pO_2 < 80), high pCO_2 levels or a low pH (< 7.4). An increase in carbon dioxide level will usually trigger ventilations. If respiratory activity cannot correct the pH, chemoreceptors activate the vagus nerve resulting in bradycardia and coronary vasodilation. Thus, patients with severe hypoxia may present with bradycardia.
- **Osmoreceptors** in the hypothalamus sense the concentration of body fluids.
- **Stretch receptors** in the ventricles sense the volume of blood return to the heart.

Hormonal compensation



In shock, the combination of hypoxia, acidosis, hypotension and volume abnormalities cause simultaneous and synergistic stimulation of all these receptors to activate the sympathetic nervous system and adrenal glands as well as other hormonal responses.

Adrenal glands



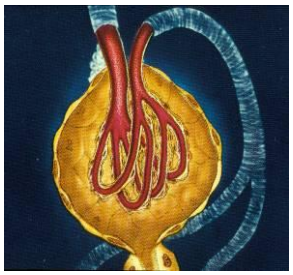
Stimulation of the SNS leads to the release of epinephrine and norepinephrine from the adrenal medulla. **Venoconstriction** precedes arterial constriction during the initial stage of shock. Given that the majority of the blood volume is stored in the veins (capacitance vessels), constricting the veins may adequately restore vascular volume. If the perfusion deficit worsens, causing further O_2 debt and acidosis, additional compensation is required and more catecholamines are released.

The SNS releases nor-epinephrine from nerve endings

Further activation of the SNS triggers the *"**fight or flight**" response*. Heart rate and myocardial contractility increase to augment cardiac output. Coronary arteries dilate to supply additional O_2 to heart muscle. Peripheral vessels constrict to redistribute blood flow to the protected vital organs (heart and brain) and shunt blood away from non-priority organs. Constriction of dermal capillary beds causes the skin to be pale and cool. Sweat glands are activated to vent off heat. Pupils dilate to enhance vision. Decreased GI perfusion slows peristalsis.

While norepinephrine secreted from sympathetic nerve endings is rapidly dissipated, adrenal catecholamines help to sustain the stress response for hours to days.

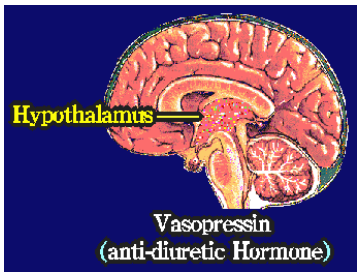
Renin - Angiotensin - Aldosterone cycle



Renin is released from the kidneys in response to hypoperfusion and to input from the SNS. Renin reacts with alpha-2 globulin in the liver to release **angiotensin I.** Angiotensin converting enzyme (**ACE**) converts angiotensin I to **angiotensin II.** Angiotensin II is a potent vasoconstrictor, helping to maintain the BP. It also causes the adrenal gland to secrete **aldosterone**. Aldosterone causes sodium retention and potassium excretion by the kidneys. The net effects are to conserve water by decreasing urinary output and to increase BP by augmenting blood volume and prompting vasoconstriction.

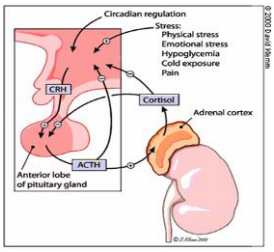
Antidiuretic hormone (ADH or vasopressin)

ADH is made in the hypothalamus and stored in the posterior pituitary gland. It is released in response to hypovolemia or hyperosmolality sensed by receptors in the carotid bodies and atria and by osmoreceptors in the hypothalamus. When released, ADH stimulates water reabsorption in the distal renal tubules and inhibits urinary output. ADH is also a potent vasoconstrictor helping to maintain the BP.



So if you are counting, this is the 3rd mechanism for vasoconstricting the vessels and maintaining mean arterial pressure (MAP):
#1 Catecholamines
#2 Angiotensin II
#3 Vasopressin

Adrenocorticotropic hormone (ACTH)



During periods of stress or trauma, the anterior hypothalamus is affected by input from the ascending reticular activating system (ARAS), brain stem, subcortex, and limbic system. This causes the hypothalamus to secrete a releasing factor that acts on the anterior pituitary to secrete ACTH. This hormone causes the adrenal cortex to increase production of glucocorticoids, like **cortisol,** that stimulate metabolic processes in the liver and kidneys to **increase blood glucose levels.** Expect adults in shock to have higher blood glucose levels due to this mechanism.

Gonadotrophins are inhibited. Women under prolonged stress may experience amenorrhea.

Kinins (Bradykinin) are potent vasodilators .They are felt to be responsible for the dramatic hypotension and hyperemia associated with anaphylactic shock.

Serotonin and histamine: These vasoactive substances are released from platelets and mast cells respectively and regulate local vascular tone and capillary permeability. Anaphylaxis and complement activation trigger their release.

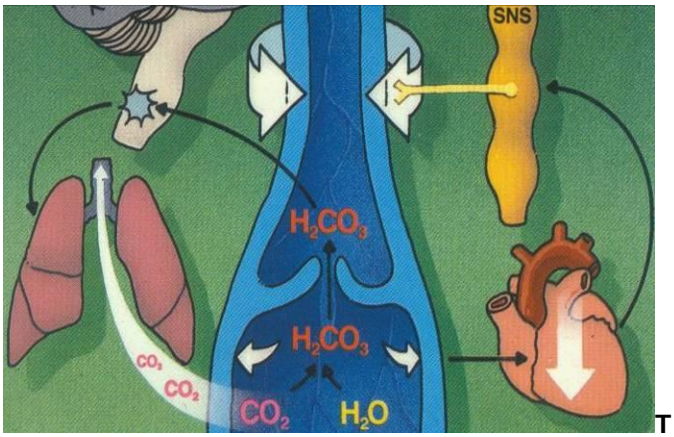
Prostaglandins are acidic lipid soluble materials distributed widely in the body. They are generally released in response to ischemia or hypoxia from the endothelial tissue or platelets and may **cause intravascular platelet aggregation, clumping and vasoconstriction**.

Chemical (respiratory) compensation

Redistribution of blood to priority organs causes hypoperfusion of the lungs. Vasoconstriction in hypoxic pulmonary beds results in alveoli that are ventilated but not perfused, thus increasing alveolar dead space. This produces a ventilation/perfusion (V_A/Q) mismatch, impaired gas exchange, and hypoxemia.

The body attempts to correct the acid-base imbalance by increasing the ventilatory rate and depth in an effort to exhale excess CO_2 (acid to the body). On room air, there is a 1:1 inverse correlation between pCO_2 and pO_2 levels. For each 1 torr the pCO_2 goes down, there is a corresponding rise of 1 torr in pO_2. Thus, **one of the earliest S&S of shock is an increase in RR**.

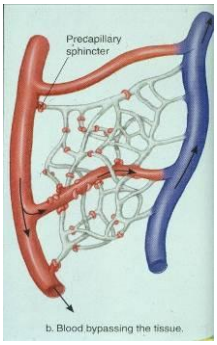
The combination of hypoxemia and respiratory alkalosis affects mental status resulting in restlessness, agitation, excitability, confusion, and lethargy (Rice, 1997).



The total net result of compensatory mechanisms in reversible shock is to successfully restore cardiac output and tissue perfusion to vital organs at the expense of the non-vital organs. If SNS fibers are intact, the patient will have an increased heart rate, increased myocardial contractility, increased diastolic BP; increased RR, pale, cool, moist skin, and decreased peristalsis.

Decompensated or progressive shock

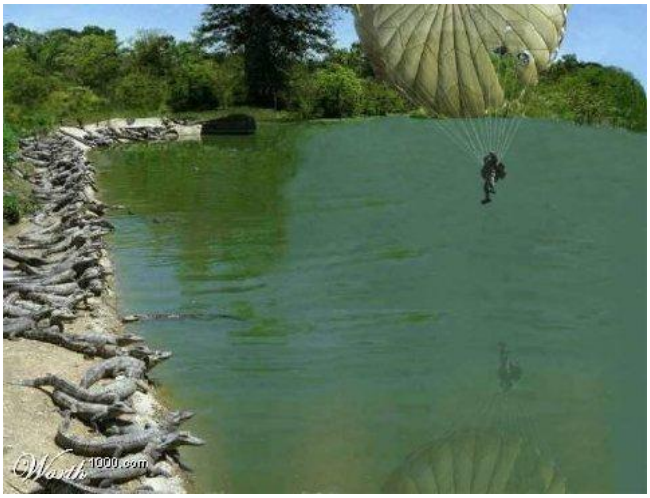
Decompensated or progressive shock occurs when the circulatory system starts to fail despite the body's maximum efforts to compensate and the **systolic BP falls below 100.**



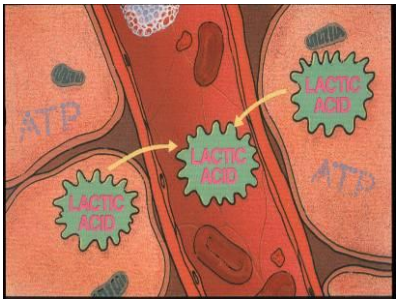
This leads to global hypoperfusion and multiple organ dysfunction syndrome (MODS). Arterioles are constricted and AV shunts open further reducing O_2 delivery to cells. There is slow flow in the upper capillary and other capillaries may open. When there is slow flow in all of them, the pH drop is marked. Blood vessels are unable to sustain vasoconstriction. Vasodilation results in decreased peripheral vascular resistance, hypotension, and capillary flooding.

Anaerobic metabolism becomes widespread and is only tolerated for only a limited amount of time. Anaerobic metabolism is much less efficient than aerobic and leads to **systemic acidosis** and **depletion of** high energy reserves (**ATP**) producing only two moles of ATP (5-10% of normal). Hypoxia will decrease the rate of ATP synthesis in the cell but will not damage the mitochondria unless it is sustained, severe, and associated with ischemia.

You're up to your ** in alligators now!***



Pathophysiology of acidosis



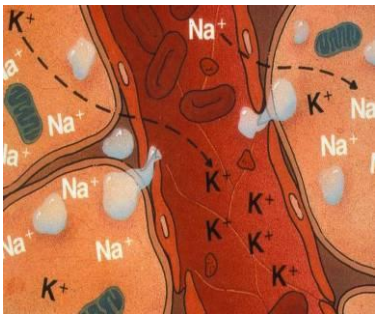
During anaerobic metabolism, glucose breakdown can only complete the first stage. This causes an accumulation of pyruvic acid. Pyruvic acid cannot be converted to Acetyl Coenzyme A without O_2 so is transformed in greater amounts to lactate and other acid by-products. Acidosis develops because ATP is hydrolyzed to ADP and phosphate with the release of a proton. Hydrogen ion accumulates, decreasing the pool of bicarbonate buffer. **Lactate** also buffers protons and **lactic acid** accumulates.

At the same time, ischemia causes an increased CO_2 production by tissues. CO_2 levels rise in the sublingual area, esophagus, stomach, duodenum, jejunum, brain, liver, and kidneys. The higher the organ's metabolic rate, the higher the CO_2 level in hypoperfused states. Excess CO_2 combines with intracellular water to produce **carbonic acid**. Thus, acidosis can be used as a measure of tissue perfusion.

The acidic condition of the blood reduces the ability of hemoglobin in red blood cells to bind with and carry oxygen. This adds to the cellular oxygen debt (shifts the oxyhemoglobin dissociation curve to the right).

Circling the drain…game over.

Micro-circulatory failure & cell membrane injury



Sodium (Na) is more abundant outside of the cell than inside. It is naturally inclined to diffuse into the cells. The sodium-potassium pump is like a "bouncer" at the cell membrane that sends the sodium back out against its concentration gradient (active transport mechanism), but needs an ample supply of ATP to fuel the process.

Reduced levels of **ATP** result in a dysfunctional Na/K pump and alterations in cell membrane function. Loss of the Na/K pump allows sodium to diffuse into the cell and stay there. **Water follows the sodium and shifts into the cell, causing the cell to swell**.

Intracellular enzymes that usually help to digest and neutralize bacteria introduced into a cell are bound in a relatively impermeable membrane. Cellular flooding explodes that membrane and allows these **lysosomal enzymes** to be released. Their job is to digest all intra and extracellular proteins, and once released, they autodigest the cell. If enough cells are destroyed, organ failure will become evident. **The release of the lysosomes heralds the onset of irreversible shock.**

Sluggish blood flow and pooling in the vessels coupled with acidic blood leads to platelet agglutination and formation of microthrombi in the **capillary stagnation phase**.

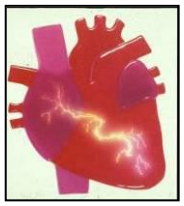
Just to compound the problem, accumulating acids and waste products act as potent vasodilators of post-capillary sphincters, releasing hydrogen ion, lactic acid, carbon dioxide and columns of coagulated red blood cells (rouleaux formations) into the venous circulation.

This is known as **capillary washout**. Rouleaux formations microembolize in the lungs.

Arterial pressure falls to the point that even the "protected organs" such as the brain and heart are not perfused. Trend pulse pressures (normal 30-50) and mean arterial pressure (MAP) (normal 70-110). When aortic root pressures fall below a mean arterial pressure (MAP) of 60 mmHg, the coronary arteries do not fill, the heart is weakened, and cardiac output falls. Myocardial depressant factor is released from an ischemic pancreas, further decreasing the pumping action of the heart and decreasing CO.

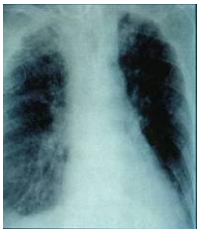
Reduced blood supply to the vasomotor center in the brain results in a slowing, then stopping of sympathetic nervous system activity.

Ischemia and necrosis lead to **Multiple Organ Dysfunction Syndrome (MODS)** where each organ system begins to fail in turn like falling dominos.



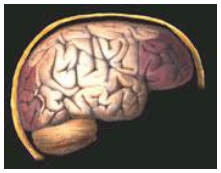
Heart: Hypoperfusion may stun even a healthy heart and result in dysrhythmias, muscle ischemia, infarction, and pump failure with ejection fractions falling far below 40%. Peripheral pulses are weak or absent, extremities become cyanotic and cold.

Lungs: Perfusion failure is evidenced by acute lung injury (ALI) **or non-cardiogenic pulmonary edema**.



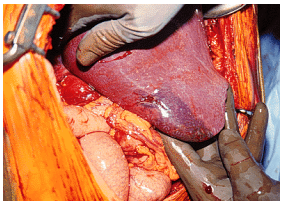
Hypoxic vasoconstriction of pulmonary beds increases pulmonary arterial pressures producing pulmonary hypertension and high afterload pressures. This puts a strain on the right ventricle. Pulmonary capillary blood flow reduction results in impaired gas exchange, reduced pO_2 and increased pCO_2. levels. Alveolar cells become ischemic and **decrease production of surfactant** resulting in massive atelectasis and a reduction in pulmonary compliance (stiff lungs).

At the same time, pulmonary capillaries become leaky resulting in interstitial and intra-alveolar edema. The net result is respiratory failure, severe hypoxemia, and respiratory acidosis.

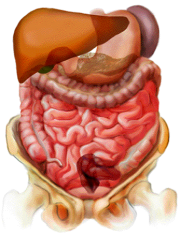


CNS: Decreased cerebral perfusion pressure (CPP) and cerebral blood flow results in confusion, reduced responses to stimuli (verbal and painful), and coma.

Kidneys: Reduced renal blood flow produces **acute tubular necrosis** (ATN) that results in oliguria (< 20 mL/hr). Toxic waste products (urea and creatinine) cannot be excreted and are retained in the blood. Metabolic acidosis worsens as kidneys are unable to excrete acids or retain bicarbonate.



Liver: Impaired metabolic function and alterations in clotting factors produce coagulation problems like disseminated intravascular clotting disorder (DIC) where the patient is clotting and bleeding at the same time. The liver fails to filter bacteria so the patient becomes vulnerable to infections. Failure to metabolize waste products (ammonia and lactate) causes markedly increased blood levels. Cell death is reflected at the hospital by an increase in enzymes such as LDH, AST, and ALT. The net result is ischemic hepatitis, hypoxic hepatitis, or shock liver.



GI tract: Hypoperfusion results in ischemic gut syndrome. Release of vasodilating endotoxins contributes to the worsening of shock.

The total oxygen deficit and its rate of accumulation are both critical determinant of survival. Inability to repay the oxygen debt to tissues invariably leads to death. Irreversible shock is diagnosed at the point when the patient is refractory to therapeutic management.

- Profound hypotension despite vasopressors
- Severe hypoxemia despite oxygen therapy
- Acute renal failure
- Multiple emboli, diffuse clotting, severe coagulopathy
- Infections
- Decreased responsiveness
- Bradycardia, hypotension, circulatory failure
- Tissue damage extensive and incompatible with life
- Multi-system organ dysfunction syndrome (MODS) evident → **patient dies**

TYPES of SHOCK

To recap: All forms of shock are due to failure of one or more of the three separate, but related factors necessary to maintain perfusion: adequate pump, circulating volume (with oxygen carrying capacity), and/or intact vascular container capable of regionalizing blood flow. Shock is classified by its primary etiology, even though multiple dysfunctions often occur in response to the primary insult.

Hypovolemic/hemorrhagic shock

This form of shock is caused by an intravascular volume deficit of either plasma or whole blood.

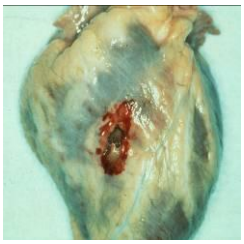
Precipitating factors

Hemorrhage

Most prevalent in trauma patients due to the following:

- Blunt or penetrating injury to vessels and/or organs
- Long bone or pelvic fractures
- Major vascular injuries including traumatic amputation
- Multi-system injury

Organs and organ systems with high incidence of exsanguination from penetrating injuries:



- Heart
- Thoracic vascular system
- Abdominal vascular system: abdominal aorta, superior mesenteric artery
- Inferior vena cava, portal vein
- Liver, spleen

Trunkey defines a severe hemorrhage as a blood loss of greater than 150 mL/min. Others site a rate of 250 mL/min as leading to exsanguination, which will cause the patient to lose ½ of their entire blood volume in approximately 10 minutes.

Fluid (plasma) shifts: Plasma shifts from the intravascular to interstitial spaces as a result of increased capillary permeability in crush or burn injuries.

Other causes of body fluid deficits

- Dehydration
- Excess GI drainage; diarrhea
- Ascites
- Diabetes insipidus
- Excess wound drainage
- Acute renal failure; high output phase
- Losses through skin and lungs
- Osmotic diuresis secondary to hyperosmolar states (DKA, HHNS)

Assessment/management

Shock resuscitation begins in the prehospital environment and continues through the ED and possibly the OR and ICU. Everyone knows when it begins, but the end points of effective resuscitation are in the process of being redefined. Classic end points were considered to be a normalizing heart rate and BP and good urine output.

Traditional markers are global measures that reflect the general circulation to large tissue beds and may be slow to exhibit signs of severe perfusion deficits.

Another limitation is that preexisting diseases or the aging process may alter a patient's response to volume losses and blunt changes in vital signs or renal function.

Cardiogenic shock (pump failure)

Etiology

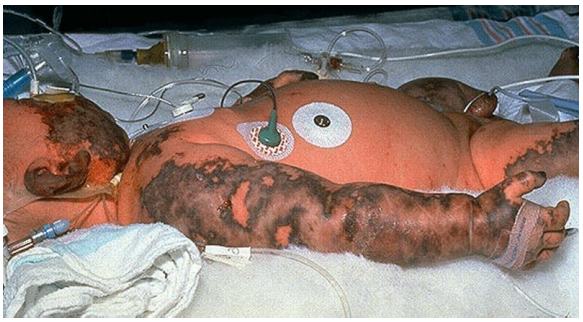
Cardiogenic shock is usually caused by extensive myocardial infarction of the LV, diffuse ischemia, or decompensated CHF resulting in primary **pump failure**. It is also seen with cardiomyopathy, valvular abnormalities, and dysrhythmias. A s*pecial type is compressive cardiac shock* due to an inadequate venous return to the heart caused by extrinsic compression, i.e., tension pneumothorax, pericardial tamponade. There is a poor prognosis when > 40% of the LV is destroyed. Historically, about 7.5% of patients with AMI develop cardiogenic shock and mortality rates range as high as 80% even with appropriate therapy.

Pathophysiology

Left ventricular function is so compromised that the heart cannot meet the metabolic needs of the body and compensatory mechanisms are maximized and ineffective. Mean arterial pressures less than 60 mmHg decrease coronary perfusion further suppressing cardiac performance, and ultimately result in total pump failure. A cardiogenic component to shock should be suspected when **hypoperfusion persists** after correcting existing dysrhythmias, hypovolemia, or altered vascular tone.

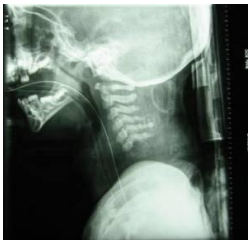
Distributive, vasogenic, low resistance, or container failure shock: Loss of peripheral vascular resistance (vasodilation) causes a relative fluid volume deficit and maldistribution of blood flow to cells.

Septic shock



Sepsis comes from the Greek word meaning "*to putrefy*". Septic shock is defined as the presence of sepsis syndrome plus a systolic BP < 90 mmHg or a decrease from the baseline BP of more than 40 mmHg. Those with sepsis develop a higher degree of shock. It is usually due to gram negative organisms but gram +, fungi, viruses and rickettsia can also be causative agents. The infection activates the inflammatory/immune response (IIR) that involves humoral, cellular, and biochemical pathways. This causes increased microvascular permeability (leaky capillaries), vasodilation, third-space fluid shifts, and microthrombi formation. In some patients an uncontrolled and unregulated IIR occurs, resulting in hypoperfusion to the cell due to opening of AV shunts, tissue destruction, and organ death. Support ABCs.

Neurogenic shock (low resistance)



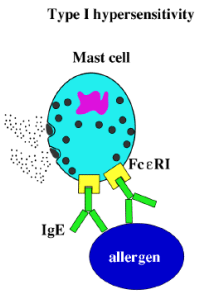
There may be **marked hemodynamic and systemic effects** in high spinal lesions. Sympathetic nervous system fibers exit at the thoracic and lumbar levels of the spinal cord before traveling to the heart, lungs, peripheral blood vessels, and sweat glands. These fibers are disrupted in SCI above T6.

Shock is caused by massive vasodilation owing to lack of sympathetic tone. Once vessels dilate, there is not enough blood to fill the new volume capacity. There may be no actual blood loss – hypoperfusion results from a blood distribution problem.

Anaphylactic Shock

Pathophysiology

The onset of anaphylaxis is generally acute. Symptoms can develop from 30-60 seconds to up to 20 minutes post-exposure and cause death within minutes.



IgE-mediated reactions occur as a result of an immune response. The immune system is exposed to an allergen (antigen) and a specific antibody (IgE) is formed and stored in mast cells and basophils. With further exposure, the antigen will bind with the IgE antibody, triggering release of vasoactive mediators. Non-IgE reactions (anaphylactoid reactions) are associated with nonsteroidal anti-inflammatory agents and aspirin.

When a hypersensitivity reaction occurs, mast cells and basophils rupture or secrete histamine, leukotrienes, and other substances that cause **systemic vasodilation,** increased capillary permeability, bronchoconstriction, coronary vasoconstriction, and skin reactions. The relative decrease in vascular volume owing to the enlarged vascular space results in a decreased cardiac output and inadequate tissue perfusion.

Risk: The severity of a reaction is affected by the quantity of the antigen; the route and rapidity of absorption (highest risk is produced by parenteral exposure; least risk by topical exposure; oral ingestion is in between); a past medical history of asthma or cardiac disease; and in patients taking beta blocker drugs.

Signs and Symptoms

A severe systemic reaction can rapidly lead to **respiratory failure and cardiovascular collapse** evidenced by BP < 90, cardiac dysrhythmias, shock and coma.

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Homework Questions

1. Define shock:

_____

_____

2. List two causes of hypoperfusion due to an inadequate pump:

_____

_____

3. List three causes of hypoperfusion due to loss of vascular tone:

_____

_____

_____

4. What is the most common etiology of **shock** in trauma patients?

   A. Brain injury
   B. Hemorrhage
   C. Respiratory failure
   D. Cardiac insufficiency

5. What two factors are multiplied to determine the cardiac output in one minute?

   _____ X _____

   Insert the numbers for the above equation.

   _____ X _____ = _____ L/min

6. Define: Stroke volume

_____

_____

7. List three major factors that all influence stroke volume:

_____

_____

_____

8. Define: Preload

_____

_____

9. Paraphrase the Frank-Starling mechanism or Starling's law:

_____

_____

10. List one EMS intervention that increases preload in a patient with hypovolemic shock.

_____

11. List one drug that is used by EMS personnel to decrease preload._____

12. Define: Afterload

_____

_____

13. List two drugs EMS personnel can give to increase afterload particularly in patients with a low resistance forms of shock (list drug name, dose, and route).

_____

_____

14. Independent of preload and afterload, cardiac contractile force is affected by:

_____

_____

14. What specific sympathetic nervous system receptors are stimulated to increase heart rate, force of contractility and speed of conduction?_____

15. Which nerve is stimulated to decrease heart rate?_____

16. What fuels are needed and how much ATP is produced during aerobic metabolism?

Fuels needed _____

Amount produced: _____

16. List the four peripheral receptors that all send feedback to the central nervous system in an effort to maintain adequate perfusion:

_____

_____

_____

_____

17. Hormonal compensation in shock includes release of two catecholamines from the adrenal gland:

_____ and _____ which

cause peripheral blood vessels to _____.

18. Activation of the renin-angiotensin-aldosterone system causes blood vessels to

Constrict / dilate (circle one) and the kidneys to reabsorb _____ and

_____.

19. Release of ADH causes blood vessels to Constrict / dilate (circle one) and the renal tubules to reabsorb _____

20. Chemical compensation is achieved through an ↑ in respiratory rate in an effort to blow off:

_____.

21. What two substances accumulate to create the acidosis experienced by a patient in shock?

_____

_____

22. What happens to hemoglobin's ability to bind with oxygen in an acidosis?

_____

23. Anaerobic metabolism only produces _____(#) moles of ATP. This results in a dysfunctional _____(cell transport mechanism) that causes the movement of water from the _____ fluid compartment to the _____ fluid compartment.

This fluid shift will cause the release of enzymes within the cell called: _____

What effect does the release of these enzymes have on the cell?

24. Which of these can hinder the clotting process?

A. Immobilization
B. Dehydration
C. Fever
D. Aspirin

25. Which of these is present if a patient's blood pressure decreases after changing from a supine to a sitting position?

A. Compensatory vasoconstriction
B. Orthostatic hypotension
C. Supine hypotension
D. A negative "tilt test"

26. What is the minimum MAP needed to maintain aortic root pressures and perfuse coronary arteries?

27. What happens to the vasomotor center in the brain when it no longer receives adequate blood flow?

28. An adult presents with chest pain, dyspnea, dusky skin, and bilateral crackles. VS: BP 60/30; P 90; R 24; SpO_2 86%; $EtCO_2$ 22 with square waveform. What etiology of shock should be suspected?

_____

29. An adult presents following an MVC with paralysis of the arms and legs and loss of sensation below the shoulders. VS: BP 70/40; P 48; R 16 and shallow with only abdominal and no chest wall movement. There are beads of sweat on the patient's upper lip but the skin is warm and dry below the shoulders. What etiology of shock should be suspected?

_____

30. An adult presents with extreme respiratory distress following the unsuspected ingestion of peanut oil. Family members state that the patient is very allergic to peanuts. The patient's tongue, lips and eyelids are extremely swollen. There is audible stridor and wheezes. VS: BP 72/44; P 110; R 32 and labored; SpO_2 88%; EtCO2 18 with sharkfin waveform. What etiology of shock should be suspected?

_____

Differentiation of Shock

Origin	Etiology	BP	P	Skin	Lungs	EMS Treatment
↓ Pump performance	Cardiogenic	↓	↓ or ↑	Pale, cool, moist	Crackles	Dopamine low dose
↓ Fluid / Volume	Hypovolemic	↓	↑	Pale, cool, moist	Clear	IV fluids
Vessels / Container dilates: maldistribution of blood; low peripheral resistance	Neurogenic	↓	↓	Flushed, dry, warm	Clear	IV fluids, atropine, high dose dopamine
	Septic		↑	Flushed/pale, hot/cool, moist	Crackles if pulmonary origin	IV fluids, high dose dopamine
	Anaphylactic		↑	Flushed/warm/moist	May have wheezes; may be ↓ w/ no sounds	IVF, epinephrine, Benadryl (diphenhydramine), albuterol, ipratropium

Assessment Parameters in Shock				
PARAMETER	**HYPOVOLEMIC**	**CARDIAC**	**NEUROGENIC**	**SEPTIC**
MAP (BP)	↓	↓	↓	↓
HR	↑	↑ or ↓	↓	↑
CO	↓	↓	↓	↑ then ↓
PVR	↑	↑	↓	↑
SpO_2	↓	↓	↓	↓
$EtCO_2$	↓	↓	↑ w ↓ RR	↓

Notes: