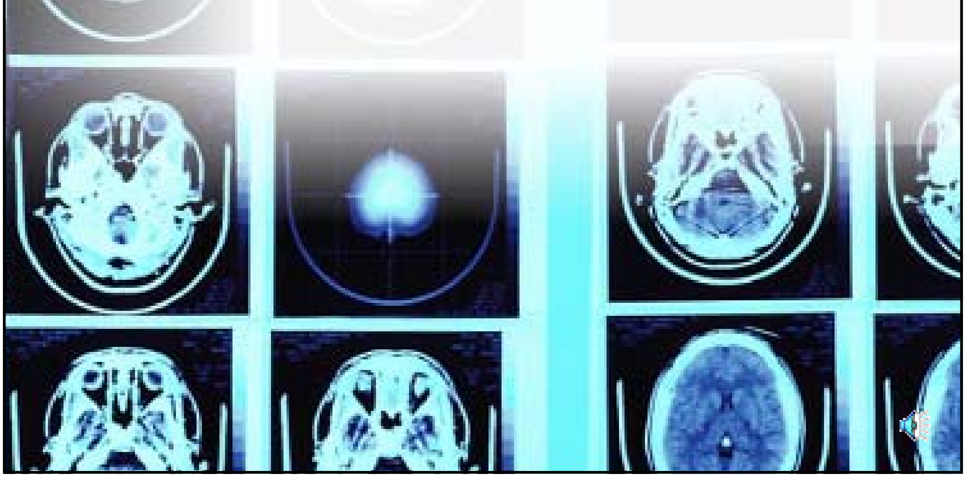
EMC 410
Trauma Management

Chapter 9. Pathophysiology of Shock



Unit Objectives

- **Upon completion of this chapter, you should be able to:**
  - Describe the anatomy and physiology of the heart and explain its autonomic innervation.
  - Describe the formulas for cardiac output and blood pressure.
  - Explain how preload, afterload, and contractility affect cardiac stroke volume.
  - Discuss the factors that determine oxygen delivery.
  - Explain the 3 phases of cellular metabolism: glycolysis, the TCA cycle, and electron transport.

Unit Objectives continued

- Discuss the pathophysiology of shock due to respiratory failure, hypovolemia, vascular failure, and cardiac failure.
- Describe the compensatory mechanisms of respiratory compensation, sympathetic nervous system response, neuroendocrine response, and transcapillary refill.
- Understand the role of systemic inflammation in the pathophysiology of shock and the cascade of events culminating in death.
- Describe interventions that improve oxygen delivery.
- Explain the common complications of shock.

Introduction

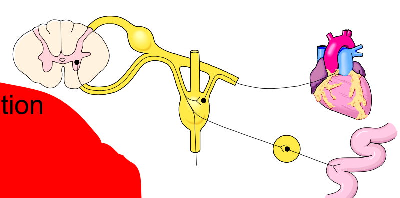
- Various early theories attempted to define and describe the presentation of shock.
- Shock is now defined as the generalized failure of the body to deliver sufficient amounts of oxygen to its tissues.
- Many of the commonly accepted signs and symptoms of shock represent compensatory measures used by the body to maintain oxygen delivery to the heart, lungs and brain.

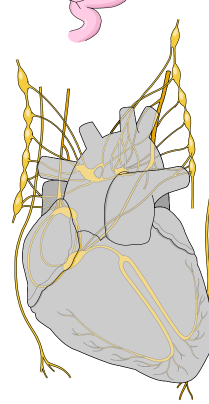
Introduction continued

- The most important aspects of prehospital management of shock are:
  - Recognition of shock in the early stages
  - Appropriate airway management and oxygen administration
  - Rapid transportation to a definitive care facility

Cardiovascular Physiology

- Heart
  - Autonomic Innervation
    - Parasympathetic (acetylcholine)
    - Sympathetic (norepinephrine; beta effects)

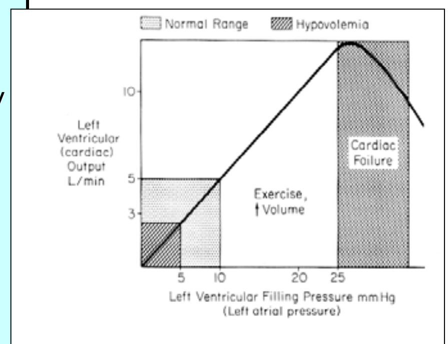




Cardiovascular Physiology continued

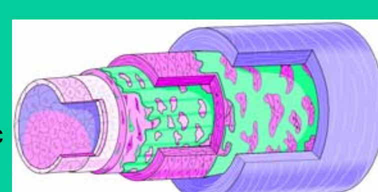
– **Cardiac Output and Blood Pressure**
  - CO (L/min) = HR x SV
  - BP ≈ HR x SV x SVR

– **Factors affecting stroke volume**
  - Preload
  - Aftlerload
  - Contractility



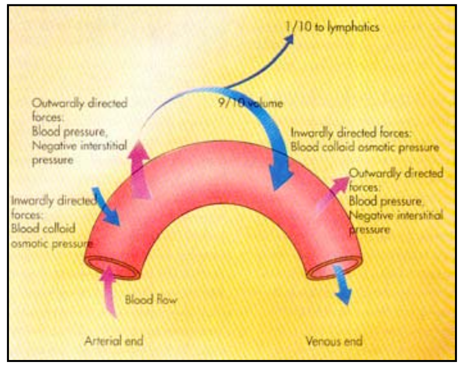
Cardiovascular Physiology continued

- **Blood Vessels**
  – Arteries and arterioles have a thicker muscular layer than veins or venules
  – Muscular layer under control of the sympathetic nervous system
  – During normal circulation, 2/3 of the blood volume is in the veins and venules
  – Venoconstriction increases blood return to the heart



Cardiovascular Physiology continued

- **Blood Vessels continued**
  - Oncotic pressure from large protein molecules draws fluid and waste products into the vascular system in the distal capillaries
  - Hydrostatic pressure drives nutrients and fluid out of the proximal capillaries and into the interstitial space

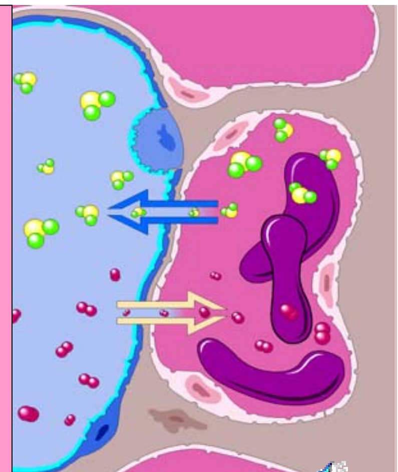


Cardiovascular Physiology continued

- Blood
  - Delivers oxygen and nutrients to the tissues and transports wastes to the liver and kidney
  - Plays a role in the immune system
  - Blood is 7% of adult TBW and 8%-9% of child’s TBW
  - Average blood volume is 5 liters
  - Formed cells accounts for 35%-45% of blood volume (Hct)

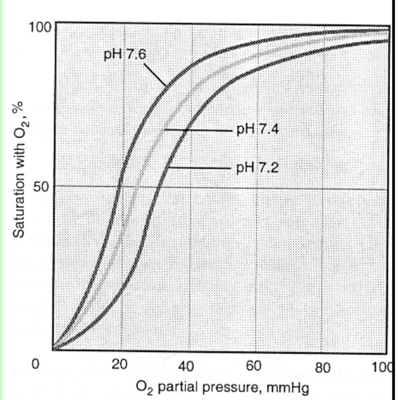
Cardiovascular Physiology continued

- **Blood continued**
  - Each RBC contains many molecules of hemoglobin which bind with oxygen for transport
  - In the lung where oxygen content is high, oxygen avidly binds to the hemoglobin
  - In the tissue where oxygen content is lower, oxygen is released to the tissues by the hemoglobin



Cardiovascular Physiology continued

- **Blood continued**
  - This uptake and release of oxygen is represented by the oxyhemoglobin dissociation curve
  - Acidosis causes a shift of the curve to the right, meaning oxygen is readily released to the tissues
  - Alkalosis causes a shift to the left whereby oxygen release to the tissues is decreased
  - The blood also contains elements essential for clotting



Factors Affecting Oxygen Delivery

- **Arterial Oxygen Delivery**
  - Arterial oxygen content (CaO_2) = (1.34 x Hgb x SaO_2)+ (0.0031 x PaO_2)
    - Each gram of hemoglobin can carry 1.34 ml of oxygen
    - Very little oxygen is dissolved in plasma
    - Reflects the role of adequate Hgb and O_2 saturation
  - Oxygen delivery (DO_2) = CO x CaO_2 x 10
    - By substitution, DO_2 = HR x SV x CaO2 x 10
    - Therefore, the heart rate, stroke volume, hemoglobin concentration, and oxygen concentration are the factors that contribute to oxygen delivery

Factors Affecting Oxygen Delivery continued

Arterial Oxygen Delivery Example

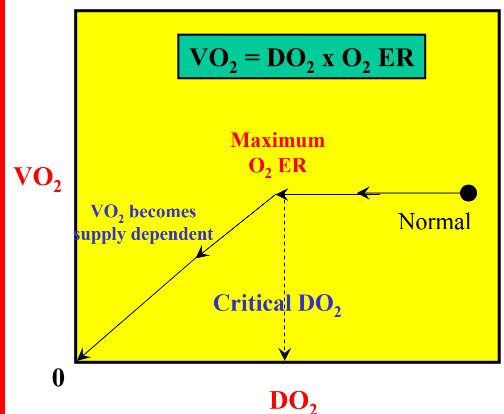
Hgb	12 g/dL
SaO_2	96%

C.O.	7 L/min
PaO_2	100 mm Hg

- Arterial oxygen content (CaO_2) = (1.34 x 12)(0.96) + 0.0031 (100) = 15.75 g/dL
- Oxygen delivery (DO2) = 7 L/min x 15.75 g/dL x 10 = 1,103 g/min

Cardiovascular Physiology continued

- **Arterial Oxygen Delivery continued**
  - Under normal circumstances, only 20% of delivered oxygen is extracted by the tissues, but in shock, this increases to 50%.
  - The ratio at which delivered oxygen is extracted by the tissue varies with flow.
  - Up to a point, increases in oxygen delivery are rewarded with increases in oxygen consumption.
  - After this point, further increases in oxygen delivery do not result in further oxygen consumption because the cells are using oxygen at their maximum rate.



Cellular Metabolism

- Oxygen plays a crucial role in the body's ability to utilize carbohydrates (sugars), amino acids (proteins), and lipids (fats) to produce energy.
- When oxygen is not present in sufficient quantities, energy production must come from anaerobic metabolism which is very inefficient.

Classifications of Shock

- **Respiratory Failure**
  - Reduces amount of oxygen delivered to the hemoglobin, and subsequently, to the tissues
  - Causes include FBAO, anoxia, and hypoxia, which may be the result of :
    - Mechanical failure
      - Open pneumothorax or tension pneumothorax
    - Diffusion failure
      - Pulmonary contusion, CHF, inhalation injuries, ARDS
    - Toxic exposures
      - Carbon monoxide and cyanide

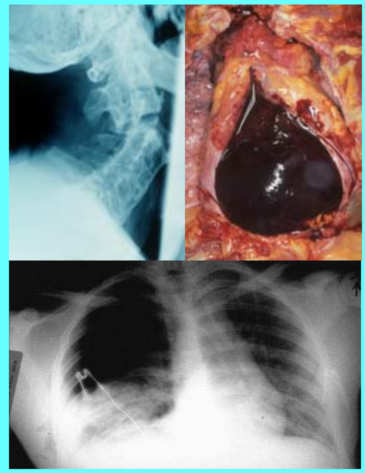
Classifications of Shock continued

- **Perfusate Failure**
  - Loss of blood (and hemoglobin) to transport oxygen to the tissues.
  - Reduction in venous return.
  - Cannot rely on hematocrit and hemoglobin levels to <u>initially</u> assess hemorrhagic shock and blood loss.

	Class I	Class II	Class III	Class IV
Blood loss (%)	< 15	15 - 30	30 - 40	> 40
Blood loss (mL)	< 750	750 – 1,500	1,500 – 2,000	> 2,000

Classifications of Shock continued

- **Vascular Failure**
  - Loss of sympathetic tone results in vasodilation and a relative hypovolemia.
  - Causes include spinal cord injury, sepsis, and anaphylaxis.
- **Cardiac Failure**
  - MI, valvular injury, rupture, dysrhythmias, tension pneumothorax, cardiac tamponade



Compensatory Mechanisms

- **Respiratory Compensation**
  - Chemoreceptors monitor the blood for oxygen and carbon dioxide levels and acidosis and alter respiratory rate and depth accordingly.
- **Sympathetic Nervous System Activation**
  - Baroreceptors in carotid sinus and aortic arch monitors blood pressure and increase sympathetic activity in response to hypotension.
  - Alpha stimulation produces vasoconstriction and shunts blood away from the periphery and GI tract to vital organs. This produces the cool, clammy skin and N&V often seen in shock.
  - Beta 1 stimulation increases heart rate and SV while Beta 2 stimulation results in bronchodilation.

Compensatory Mechanisms continued

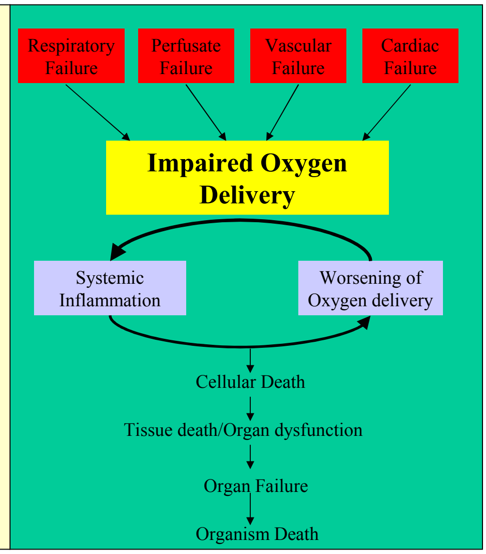
- **Neuroendocrine Response**
  - ACTH released by the pituitary stimulates the renal cortex to produce aldosterone and cortisol. Aldosterone causes reabsorption of sodium and water in the kidney.
  - Reduced perfusion of the kidney causes the release of renin which ultimately results in the lung tissue converting angiotensin I to angiotensin II which is a potent vasoconstrictor.
  - Corstisol stimulates metabolism.
  - Epinephrine and norepinephrine are secreted by the adrenal medulla.
  - Vasopressin (ADH) is released by the pituitary.

Compensatory Mechanisms continued

- **Transcapillary Refill**
  - Following hypovolemia, osmosis allows for movement of fluid from the intracellular and interstitial spaces into the intravascular space.
  - Slow process, taking up to 2 hours, and results in the movement of less than 2 liters of fluid.
  - Hgb values reflect hemorrhage only after transcapillary refill takes place.

Decompensation

- Compensatory mechanisms function quite well, but once overwhelmed, oxygen delivery will fall.
- Host Factors Affecting Compensation
  - Age
  - Medications
  - Alcohol intoxication
  - Physical conditioning
- Systemic inflammation process results in further damage to capillary membranes.
- Staged death
  - Cells, tissues, organs, organ systems, organism



Improving Oxygen Delivery

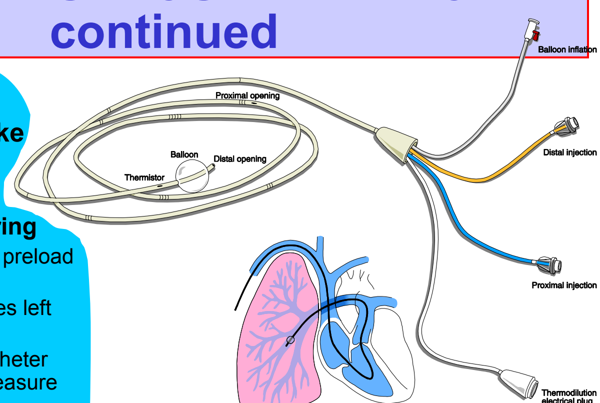
- **Improve oxyhemoglobin saturation**
  - Given C.O. of 7.0 L/min, Hgb of 12 g/dL
    - If SpO_2 is 92, DO_2 is 1035
    - If SpO_2 is 98, DO_2 is 1103
    - Larger changes in DO_2 for smaller changes in SpO_2 on the steeper portion of the oxyhemoglobin curve.
- **Ensure an adequate heart rate.**
- **Increase hemoglobin concentration**
  - Increasing hemoglobin from 8 g/dl to 12 g/dl increases DO_2 by 50%.

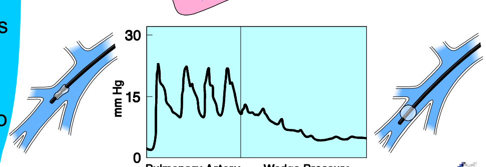
Improving Oxygen Delivery continued

- **Manipulating Stroke Volume**
  - **Improving Preload**
    - 30 ml/kg crystalloid
    - If vital signs and oxygen delivery improve, bleeding has probably ceased.
    - If vital signs improve only transiently, the patient is experiencing continued blood loss and needs rapid surgical intervention.
  - **Fluid Choice**
    - Larger volumes of crystalloids needed to achieve the same hemodynamic effects as smaller volumes of colloids
    - In shock, capillary wall permeability increases resulting in leakage of colloids out of intravascular space.
    - Cyrstalloids are inexpensive, easier to store, and have longer shelf life.

Improving Oxygen Delivery continued

- **Manipulating Stroke Volume continued**
  - **Invasive Monitoring**
    - CVP measures preload of right atrium
    - PCWP measures left atrial pressure
    - Swan-Ganz catheter also used to measure cardiac output
    - Fluid is administered as long as C.O. improves. When C.O. only minimally improves and/or PCWP begins to increase, further fluid resuscitation results in LV failure.



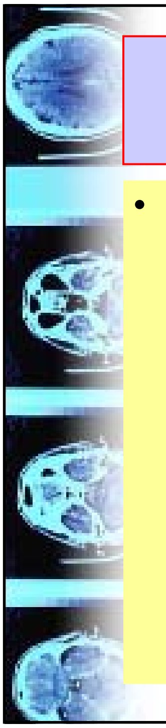


Improving Oxygen Delivery continued

- **Manipulating Stroke Volume continued**
  - **Afterload**
    - Once preload is maximized, afterload should be considered.
    - SVR = [(MAP-CVP)/CO] x 80
    - Normal SVR is 800 - 1200 dynes/second/cm^5
    - Spinal shock presents with a low SVR
    - Hemorrhagic shock initially presents with a high SVR

Improving Oxygen Delivery continued

- **Manipulating Stroke Volume continued**
  - **Contractility**
    - Inotropic agents can be used to increase contractilty.
    - Rarely used in trauma care.
  - **Trendelenburg position**
    - No longer recommended as it may impair breathing.
    - Venous capacitance vessels already depleted of blood volume and raising the legs simply increases afterload on the heart.



Improving Oxygen Delivery continued

- **Cautionary Notes**
  - **Vasoconstrictors have virtually no use in shock.**
    - $DO_2 = HR \times SV \times CaO_2 \times 10$
    - Blood pressure ≠ blood flow and oxygen delivery
    - Peripheral vasoconstriction increases SVR and decreases C.O.
  - **Bicarbonate Therapy**
    - Does nothing to improve metabolic acidosis in shock
    - Bicarbonate increases CO_2 production
    - Excessive bicarbonate shifts the oxyhemoglobin dissociation curve to the left further impairing oxygen delivery