Shock

Percipitous drop in systemic BP resulting in widespread impairment of cellular metabolism → organ failure → Death

5 types:

Cardiogenic shock - results from heart failure

Hypovolemic shock - excessive loss of blood begins with a loss of 20-25% of total blood volume (750ml)

Neurogenic shock - massive ↑ in parasymp activity & massive ↓ in symp activity
widespread vasodilation ↓
percipitous drop in systemic BP

results from trauma to spinal cord
or medulla. Also can be result of
severe emotional stress.

Anaphylactic shock — results from
a widespread hypersensitivity
reaction known as anaphylaxis

↓

systemic inflammation

↓

widespread vasodilation

↓

percipitous drop in systemic BP
Septic shock - bacterial infection in the blood

↓ widespread inflammation

↓ widespread vasodilation

↓ precipitous drop in systemic BP

Treatment:
- deal with the cause
- intravenous fluid → ABV → ABP
- vasopressor drugs can be used to try to counteract the vasodilation (not for hypovol. shock)
- O₂ is given to support the tissues and prevent organ failure
Figure 25-1 Structures of the pulmonary system. The enlargement in the circle depicts the acinus, where oxygen and carbon dioxide are exchanged. (From Thibodeau GA, Patton, KT: Anatomy & physiology ed 5. St. Louis, 2003, Mosby.)
Pulmonary System

Primary function is the exchange of CO₂ + O₂ between the environment and the blood.

Exchange of these gases occurs primarily across the walls of the alveoli.

Two characteristics that facilitate gas exchange:

- Wall of the alveoli consists of squamous epithelial cells (single layer), minimizes gas exchange distance.
- 300 million alveoli in the lungs that provide a large surface area for gas exchange.
These characteristics create two problems:

- The thin walls of the alveoli tend to collapse in on themselves due to surface tension created by fluid lining the surface of the alveoli.

\[ \text{polar molecule} \]

\[ H^+ \quad O^- \]

\[ \text{Surface tension pulls inwards} \]

\[ \text{alveoli on the walls of the alveoli} \]

causing them to collapse unless the force is disrupted.
Fig. 25-6 *Section through the alveolar septum (gas-exchange membrane).* Inset shows a magnified view of the respiratory membrane composed of the alveolar wall (fluid coating, epithelial...
- The thin, large, wet surface area of the alveoli provides an excellent route for entering the body pathogens.

To solve the first problem, there are type II alveolar cells (surfactant cells) in the alveoli that secrete a lipoprotein called surfactant that mixes with the fluid lining the alveoli and disrupts the surface tension created by the attraction between the water molecules. Surfactant cells develop at 36 months of fetal development, but don't secrete enough surfactant to prevent
alveolar collapse until 8 months of fetal development.
Babies born before 8 months have difficulty with gas exchange because their alveoli are collapsing reducing the surface area for gas exchange.
Condition is called "Neonatal Respiratory Distress Syndrome" (NRDS).

Treatment:
- Using positive pressure ventilation to provide positive pressure in the lungs to hold the alveoli open.
- To provide aerosolized surfactant in the inhaled air that can supplement the surfactant being produced by the babies' surfactant cells.
If premature birth is anticipated, antenatal corticosteroid therapy can be initiated, in which the mother is given corticosteroid injections 7-10 days before delivery. The corticosteroids stimulate surfactant production and maturation of the surfactant cells.

Acute Respiratory Distress Syndrome (ARDS) occurs in adults as a result of inhalation of noxious gases (e.g., chlorine gas), smoke, hot air that damages the alveolar-capillary exchange surface.
Results in decreased surfactant production
↓
inflammation in lung tissues
↓
pulmonary edema + bleeding
↓
↓ gas exchange
↓
Hypoxemia (low arterial O2)
+ can progress to respiratory failure and death.

Treatment:
support respiratory system until lung function recovers:
- mechanical ventilation with positive pressure ventilation
New treatment strategies being tried include use of anti-inflammatory and surfactant replacement therapy.

Second problem: Exposure of the thin wet surface area of alveoli to pathogens.

Dealt with in a couple of ways:

- Alveolar macrophages phagocytose pathogens that reach the alveoli

- URT + LRT are lined by pseudostratified ciliated columnar epithelium (Respiratory epithelium)
**Figure 24-2 The Respiratory Epithelium**

(c) Sketch and light micrograph showing the sectional appearance of the respiratory epithelium (LM × 932)

(b) A surface view of the epithelium, as seen with the scanning electron microscope (SEM × 1647)

Cilia in LRT push the mucus up towards the pharynx.

Cilia in URT push the mucus back towards the pharynx.

Mucus in pharynx is swallowed and passes into stomach, where pathogens are killed.
The mucus produced by the goblet cells is thick and requires water to thin it out. This water comes from the body across the respiratory epithelium.

Water is drawn out of the tissues by creation of an osmotic gradient. This osmotic gradient is created by transport of Cl⁻ ions across the epithelium by transport proteins called cystic fibrosis transmembrane conductance regulators (CFTRs). Transport Cl⁻ into the mucus that creates an osmotic gradient that draws water & Na⁺ into the mucus across the epithelium.
In cystic fibrosis, an autosomal recessive gene that codes for the CFTR protein is mutated and so the person can’t make functional CFTR proteins, so can’t transport Cl- ions and can’t create osmotic gradient to draw water into the mucus of respiratory tract. Mucus remains thick and can’t be moved by the cilia.

- Mucus obstructs airways resulting in difficulty breathing
- Trapped pathogens in mucus increase incidence of respiratory tract infections.
Treatment:
- Postural drainage + percussion of the lungs to help move the mucus along
- Bronchodilators making it easier for mucus to move along dilated airways
- Use of mucolytics (breakup mucus) + expectorants
- Prompt and aggressive treatment of pulmonary infections with antibiotics.

No cure, some effort at gene therapy to introduce CFTR gene into epithelial cells has been successful, but it has not been successful in treating CF.
Lung Ventilation

- Diaphragm pulls downward during inhalation.
  - Expansion of the volume of the thoracic cavity creates a situation where \( P_i < P_o \).
  - Air moves down the pressure gradient and fills the lungs.

- During normal, quiet breathing, air is moved out during exhalation due to elastic recoil of the muscles and connective tissue of the lungs and thoracic cavity.
  - Creates a situation where \( P_i > P_o \).
  - Air moves out of the lungs down the pressure gradient.
The movement of air into and out of the lungs requires that the thoracic cavity be a closed chamber.

If the thoracic wall is damaged allowing air to enter the thoracic cavity, then the negative pressure required to move air in can't be generated.

This kind of damage results in a pneumothorax.

Most commonly caused by a puncture wound of the thoracic wall.
Tension pneumothorax - the air brought in through the puncture wound doesn't move back out, pushes against the lung causing it to collapse → Atelectasis (collapsed lung)

Rupture of a bleb can also cause a pneumothorax. Bleb is like a blister on the lung, that may
rupture allowing air to move from the lung into the thoracic cavity. This is known as a spontaneous pneumothorax.

Treatment:
- Repair of chest wall or bleb to prevent air from moving into the thoracic cavity.
- Needle aspiration to remove air in the thoracic cavity.
- When air is removed the lung will inflate if atelectasis has occurred.

Hemothorax — bleeding into the thoracic cavity preventing lung inflation.
- Requires locating and stopping the bleeding and draining the blood from the thoracic cavity by inserting a tube through the chest wall.
Diseases that affect lung ventilation.

Two categories:

- Obstructive Lung (Pulmonary) Diseases (OPDs)

- Restrictive Lung Diseases (RLDs) (aka Interstitial Pulmonary Diseases) (IPDs)

OPDs

Characterized by difficulty in expiration (trouble getting air out of the lungs)

Acute OPDs and Chronic OPDs

Ex. of acute OPD - Asthma
inflammatory disease involving a hypersensitivity reaction of the airways with spasmodic contraction of the bronchial smooth muscle.

Bronchiole diameter is decreased

\[\downarrow\]

Increases resistance to airflow through bronchioles

\[\downarrow\]

Airflow is reduced.

So, air gets trapped in the lungs.

Asthma attack triggers

- Allergens
- Cold air
- Exercise
- Emotional upset

Any trigger that causes inflammation of airways with spasmodic contraction of bronchial smooth muscle
Results:
- Dyspnea → increased rate + depth of breathing
- Hypoxemia
- Hypocapnia (drop in CO₂ levels in the blood)

As the attack worsens and spreads, hypocapnia occurs (increase CO₂ levels in the blood)

↓
Respiratory acidosis (upsh levels in blood)

↓
Respiratory Failure + Death
Treatment:

- Avoiding triggers

- Pharmacologic treatments to relieve bronchoconstriction:
  - Bronchodilators (β2 adrenergic agonists) that stimulate β2 receptors on bronchial smooth muscle → bronchodilation

- Anticholinergic drugs to block parasymp. input to bronchial smooth muscle → bronchodilation

- Antihistamines, anti-leukotrienes, and corticosteroids may be used to reduce inflammation in the airways.

- O2 may be administered