Respiration
Functions

• **Gas exchange:** Grab $O_2$, eject $CO_2$
• **Regulate blood pH:** Alters $CO_2$ levels
• **Voice production:** air movement past vocal cords
• **Olfaction:** in nasal cavity
• **Innate immunity:** physical protection of blood
Anatomy

- **Upper respiratory system**
  - Nose, nasal cavity, pharynx
- **Lower respiratory system**
  - Larynx, trachea, bronchi, lungs
Pharynx

- Common passageway for respiratory & digestive tract
  - Nasopharynx
    - Auditory tubes empty here
    - **Soft palate** = floor of nasopharynx; posterior portion is **uvula**
    - This is **elevated** (closed) when we swallow & **held open** when we sneeze
    - Pharyngeal tonsils located here
Oropharynx & Laryngopharynx

- **Oro** extends from uvula to epiglottis
  - Oral cavity opens into oropharynx
    - Food passes through here
  - Lined with *stratified* epithelium (protects against abrasion)
  - Two tonsils

- **Laryngo** extends from epiglottis to esophagus
  - Food and drink
Larynx

- Complex assortment of cartilages
  - Maintains open air tube
  - Voice production
  - Allow us to hold breath
  - Closes air tube during swallowing to prevent food from entering trachea
Trachea & Bronchi

- **Trachea** = windpipe
  - CT & smooth muscle, supported by cartilage
    - Cartilage protects trachea & maintains an open airway; smooth muscle constricts airway
    - Effect of smoking: Destroys ciliated cells; mucous collects; microorganisms are not ejected

- **Bronchi**
  - Symmetrical branches from trachea
  - Extensions with same tissue types
Lungs

• Divided into lobes (R=3, L=2), divided by septa
• Bronchi subdivide further into bronchi &:
  - **Bronchioles**, which subdivide further, ending in alveolar ducts and alveoli (small air sacs)
  - As bronchi & bronchioles divide, cartilage decreases & smooth muscle increases
    • During exercise alveolar duct diameter increases
    • During asthma attack, diameter decreases
Respiratory membrane

- Characterized by extreme thinness
- Thin fluid layer lines alveolus
- Alveolar epithelium = simple squamous
- Interstitial space = THIN
- Capillary epithelium = simple squamous
Ventilation

• Inspiration and expiration
• Caused by changes in thoracic volume, which produce changes in air pressure in lungs
  - Muscles of expiration: abdominals, internal intercostals
  - Muscles of inspiration: diaphragm, external intercostals, pectoralis minor, scalenes, sternocleidomastoid
End of expiration

Muscles of inspiration
- Sternocleidomastoid
- Scalenes
- Pectoralis minor
- External intercostals
- Diaphragm (cut)

Clavicle (cut)

End of inspiration

Quiet breathing: The external intercostal muscles contract, elevating the ribs and moving the sternum.

Labored breathing: Additional muscles contract, causing additional expansion of the thorax.

Muscles of expiration
- Internal intercostals
- Abdominal muscles

Diaphragm relaxed

The diaphragm contracts, increasing the superior-inferior dimension of the thoracic cavity.

Abdominal muscles relax.
Pressure changes & Airflow

- At end of expiration:
  - $P_{atm} = P_{alv}$
  - No air flow
Pressure changes & Airflow

- **During inspiration:**
  - increased thoracic volume decreases $P_{alv}$
  - $P_{atm} > P_{alv}$
  - Air flows into alveoli
Pressure changes & Airflow

- End of inspiration:
  - $P_{\text{atm}} = P_{\text{alv}}$
  - No air flow
Pressure changes & Airflow

- **During expiration:**
  - Decreased thoracic volume = increased pressure in side alveoli
  - $P_{atm} < P_{alv}$
  - Air flows out of lungs
Passive expiration

- Passive expiration occurs due to lung recoil:
  - Lung recoil has two causes:
    - Elastic fibers in CT of lung
    - Surface tension of H₂O molecules pull on alveolar walls
  - **Surfactant opposes lung recoil**
    - Secreted by cells of alveolar epithelium
    - Consists of lipoproteins & interferes with surface tension produced by water
Lung volumes

• **Tidal Volume** - 500 mL
• **Inspiratory Reserve Volume** - 3000 mL
  - What you can forcefully inspire after TV
• **Expiratory Reserve Volume** - 1100 mL
  - What you can forcefully expire after TV
• **Residual Volume** - 1200 mL
  - Air remaining in lungs & respiratory passage after max expiration = anatomical dead space
Vital Capacity

- Sum of Tidal, Inspiratory, Expiratory Reserve volumes
  - Maximum amount of air one can forcefully inspire and expire
- Influenced by age, sex, body size, and training
  - Trained athlete may have 40% higher VC than untrained
- Clinical importance - used to assess resistance to airflow; high resistance may indicate fluid build-up, inflammation, alveolar collapse, smooth muscle constriction, etc.
  - Emphysema patients, asthmatics, chronic bronchitis:
Why is gas exchange efficient?

- Substantial differences in PP of gases across respiratory membrane
- Short distances for diffusion
- Gases are lipid soluble
- $SA_T$ is large
- Coordinated blood flow & air flow
Gas Exchange

• **Mainly** across respiratory membranes of alveoli
  - some across alveolar ducts & respiratory bronchioles; the rest = functional dead space

• **THIN** - gas exchange occurs **easily**
  - ANY increase in thickness decreases gas exchange rate
    - **Ex:** pulmonary edema = increased fluid layer in alveoli; rate of gas exchange plummets; $O_2$ diffusion is disproportionately affected
Surface Area (SA)

- Total SA of respiratory epithelium = 70 m²
  - Why so large?
- During strenuous exercise, ANY reduction in SA severely affects gas exchange
  - Other examples: surgical removal of lung tissue; destruction by cancer or emphysema (alveoli expand but capillaries deteriorate); reduced production of surfactant (premature infants may not have enough yet)
Partial Pressure (PP) aside

- **PP** = pressure exerted by ONE gas in a mixture of gases
  - Total pressure = 760 mm Hg (sea level) & 21% is O$_2$
  - $P_{O_2} = (760 \text{ mm Hg} \times 0.21) = 160 \text{ mm Hg}$
  - $P_{O_2}$ in alveoli = 100 mm Hg
  - At liquid/air interface, gasses diffuse so that PP of each gas is **equal** in both media
Gas Diffusion

• At respiratory surface of lungs:
  - In pulmonary capillaries: $P_{O_2}$ is LOW & $P_{CO_2}$ is HIGH
  - In alveolar sacs: the reverse is true
    • This leads to $O_2$ diffusing into capillaries and $CO_2$ diffusing into alveoli

• At tissues:
  - In interstitial fluid: $P_{O_2}$ is LOW & $P_{CO_2}$ is HIGH
  - Inside cells, reverse is true
    • $O_2$ diffuses into cells and $CO_2$ diffuses into interstitial fluid
O₂ Transport

- 98.5% of O₂ in blood is bound to hemoglobin (iron-containing protein)
  - O₂ binds reversibly with hemoglobin
  - Depends on P O₂ in neighboring areas
  - Hemoglobin releases O₂ at tissues, where P O₂ is LOW, it binds O₂ at alveoli, where P O₂ is HIGH
**CO$_2$ Transport**

- 7% as dissolved CO$_2$
- 23% attached to hemoglobin
- 70% as bicarbonate ions

  - Carbonic anhydrase (enzyme) converts CO$_2$ to carbonic acid; these dissociate into bicarbonate ions

  Carbonic anhydrase exists on surface of capillary epithelia and inside RBCs.
$CO_2$ Transport

- In lungs:
  - the reverse reaction occurs, converting bicarbonate ions to $CO_2$ and allowing it to diffuse into alveoli
PO₂ effects binding to Hb

- As PO₂ increases, so does binding to Hb
- Virtually ALL Hb is saturated with O₂ when it leaves the lungs
- On Mt. Everest, air pressure ~ 253 mm Hg
  - PO₂ = 253 * 0.21 = 53 mm Hg
Temp & pH changes shift this saturation curve

- As pH declines Hb saturation decreases
- As temp. increases Hb saturation decreases
Ventilation rates

- Normal **adult** rate = 12-20 cycles/min
- **Children** = 20-40 cycles/min
- Primary control at **medulla oblongata**
  - Neurons stimulate muscles of respiration
  - Deeper, more rapid breathing results from stimulating **more** muscle fibers **more frequently**.
    - More APs & increased frequency of APs
Respiratory Areas

- 2 medullary centers
  - **Dorsal nuclei** contract diaphragm = ??
  - **Ventral nuclei** stimulate both sets in intercostals and abdominal muscles = ??

- 1 **pontine center**
  - Role in switching from inspiration to expiration
Respiration Cycle

- **Beginning Inspiration**: Some neurons of medullary center are always excited
  - Integrate info (mechano & chemoreceptors & voluntary inputs) & fire AP at threshold
- **Increasing**: Positive feedback increases stimulation of resp. muscles
- **Stopping**: Medullary neurons are inhibited
  - Stretch receptors in lungs, pontine neurons
Modification of Ventilation

- **Nervous control and feedback**
  - *Higher brain centers*: speech, breath holding, sobbing & gasping (limbic system)
  - *Reflexes*: Sneeze, cough, Hering-Breuer
    - H-B limits extent of inspiration; prevents overventilation in infants & in adults during heavy exercise
  - *Touch, thermal pain receptors* in skin cause rapid inspiration
Modification of Ventilation

- **Chemical control** (chemoreceptors)
  - MO monitors **pH** level of blood (proxy for CO$_2$ levels)
    - Increases in CO$_2$ = decrease in pH
    - Hypercapnia = high CO$_2$ concentration
  - Carotid artery & aorta monitors O$_2$ level
    - Hypoxia = low O$_2$ levels $\rightarrow$ excitation
    - Emphysema, high altitude, asphyxiation reduce O$_2$, leaving CO$_2$ unchanged
(a) Higher centers of the brain (speech, emotions, voluntary control of breathing, and action potentials in motor pathways)

(b) Medullary chemoreceptors
   \[ \downarrow pH, \uparrow CO_2 \]

(c) Carotid and aortic body chemoreceptors
   \[ \downarrow O_2 \]

(d) Hering-Breuer reflex
   (stretch receptors in lungs)

(e) Proprioceptors in muscles and joints

(f) Receptors for touch, temperature, and pain stimuli

Input to respiratory centers in the medulla oblongata and pons modifies respiration.
Immediate Effect of Exercise

- **Abrupt increase in ventilation rate**
  - *Induced* by collateral *motor neurons* that activate skeletal muscle
  - *Increased* by *sensory feedback* from collateral fibers reporting joint movement
  - *Learned* through training to match respiration rate to level of activity

- **Gradual increase**
  - Levels of 4-6 min after onset
Immediate Effect of Exercise

- As long as exercise remains aerobic, average arterial $O_2$, $CO_2$ and pH levels remain close to resting levels.
- At anaerobic threshold our ability to exercise indefinitely ends, due to accumulation of lactic acid (pH changes), and ventilation rate increases further.
Adaptations to Exercise

• Increased efficiency of cardiovascular function: $O_2$ delivery and $CO_2$ expulsion (i.e. ventilation is not the rate limiting step)
• Tiny increase in VC & decrease in RV
• TV during exercise increases lots
• Respiratory rate & ventilation rate increase lots
Changes with age and smoking

![Graph showing respiratory performance changes with age and smoking habits.](image-url)