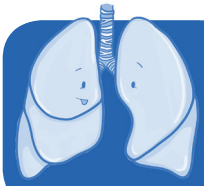

NOTES
NORMAL VARIATIONS

PULMONARY CHANGES DURING EXERCISE

osms.it/pulmonary_changes_during_exercise

RESPIRATORY RESPONSE TO EXERCISE

- Exercise → muscle workload increase → consumption of significant O_2 amounts, above baseline production of CO_2, lactic acid
- Increased O_2 demand → hyperpnea (ventilation increases 10–20x to compensate)
- Hyperpnea vs. hyperventilation
  - *Hyperpnea:* aims to maintain homeostasis → blood O_2 ,CO_2 levels remain relatively constant
  - *Hyperventilation:* excessive ventilation, blowing off too much CO_2 → low P_{CO2}, respiratory alkalosis
- Exercise-induced ventilation not initially prompted by alterations in blood gases (rising P_{CO2} , declining P_{O2}, pH)
- Ventilation increases abruptly as exercise begins due to neural factors
  - Psychological stimuli (conscious exercise anticipation)
  - Simultaneous cortical motor activation of skeletal muscle, respiratory centers
  - Proprioceptors moving muscles, tendons, joints → stimulate respiratory centers
  - Initial neural regulation → early compensation to exercise as opposed to waiting for change in blood values
- Initial abrupt increase in ventilation is followed by gradual increase (reflective of lung CO_2 delivery rate) → eventually, steady state of ventilation appropriate for intensity achieved
- Exercise cessation → initial small abrupt decline in ventilation (higher neurological stimulation ends) → followed by gradual decrease to pre-exercise respiratory rate (gradual decrease in CO_2 flow to lungs)

PULMONARY CIRCULATORY RESPONSE

- Cardiac output increases to meet tissue O_2 demand → increased right heart output → increased blood flow through pulmonary circulation → increased blood return to left heart → increased output to systemic circulation → increased O_2 tissue delivery
- Exercise → pulmonary resistance decrease → perfusion of more pulmonary capillary beds → more even distribution of pulmonary perfusion, ventilation → improved V/Q ratio (decreased physiological dead space) → increased gas exchange efficiency

HEMATOLOGICAL RESPONSE

Bohr effect

- Hemoglobin's oxygen binding affinity is inversely related to acidity, carbon dioxide concentration
  - Exercise → increased tissue P_{CO2}, decreased tissue pH, increased temperature → right shift of O_2-hemoglobin dissociation curve → decreased affinity of hemoglobin for O_2 → greater unloading of oxygen to exercising muscle

Regulation of blood gases during exercise

- Arterial P_{CO2}, P_{O2} remain nearly constant during exercise
- Venous P_{CO2}, P_{O2} may change significantly during exercise
  - Ventilation increases sufficiently to blow off all excess CO_2, maintain arterial homeostasis

Anaerobic respiration

- Leads to rise in lactic acid levels
- Not due to inadequate respiratory function
- Alveolar ventilation, pulmonary perfusion remain well matched during exercise → hemoglobin fully saturated
- Cardiac output limitation/limits of skeletal muscle to utilize oxygen → rising lactic acid

RESPIRATORY RESPONSE TO EXERCISE OVERVIEW

	RESPONSE
VENTILATION RATE	↑
PHYSIOLOGIC DEAD SPACE	↓
V/Q RATIO	More equal distribution throughout lungs
PULMONARY BLOOD FLOW, CARDIAC OUTPUT	↑
O_2 CONSUMPTION	↑
CO_2 CONSUMPTION	↑
ARTERIAL P_{O2}, P_{CO2}	No change
ARTERIAL pH	Light exercise: no change

PULMONARY CHANGES AT HIGH ALTITUDE & ALTITUDE SICKNESS

osms.it/pulmonary_changes_high_altitude_altitude_sickness

RESPIRATORY RESPONSE TO ALTITUDE

- Humans typically live at altitudes between sea level and 2400m/7800ft
- Altitudes > 2400m/7800ft → lower overall atmospheric pressure → lower P_{O2} → hemoglobin less saturated at baseline
  - At rest at sea level hemoglobin typically unloads 20–25% O_2 content on a single trip through the circulatory system
  - Significant functional reserve allows for survival due to further hemoglobin unloading when poorly saturated

ACCLIMATIZATION

- Long-term, slow steady move from sea level to higher altitude → respiratory, hematopoietic adaptation
- Decrease in arterial P_{O2} → peripheral chemoreceptors more responsive to increases in P_{CO2} → chemoreceptors stimulate medullary inspiratory center → increased breathing rate

Initial (fast) adaptation

- Some changes occur immediately, others over course of days
- Pulmonary
  - Minute ventilation → 2–3L/min higher than sea level
  - Increased ventilation → decreased arterial CO_2 (<40mmHg) → respiratory alkalosis → increased blood pH → inhibition of central, peripheral chemoreceptors → offset increase in ventilation rate (initial effect)
  - As adaptation occurs → HCO_3^- excretion increases → HCO_3^- concentration in cerebrospinal fluid (CSF) decreases → CSF pH decreases toward normal → increased ventilation rate resumes
  - Respiratory alkalosis as result of rapid ascent to high altitude managed with carbonic anhydrase inhibitors → increased HCO_3^- excretion → mild compensatory metabolic acidosis
- Hematological
  - Increase in 2,3-bisphosphoglyceric acid (2,3-BPG) concentration → hemoglobin affinity for O_2 reduced → increased unloading of O_2 at tissue level (also decreases efficiency of oxygen loading in lungs)
- Cardiac
  - Increased heart rate
  - *Right heart hypertrophy:* low P_{O2} alveolar gas → pulmonary vasculature vasoconstriction → increase in pulmonary vascular resistance → increased right heart strain → right ventricular hypertrophy
- Oxygen conservation
  - Non-essential body functions suppressed → reduction in food digestion efficiency (decreased circulation in favor of perfusing more important organs)

Late (slow) acclimatization

- Occurs over weeks to months
- *Hematological:* hypoxia → kidneys produce more erythropoietin → stimulates bone marrow production of red blood cells → total O_2 carrying capacity of blood increased
  - Essential compensation for living at altitude
  - Increases blood viscosity → greater blood flow resistance → greater heart workload
  - *Full acclimatization:* increase in red blood cell plateaus
- Effect on complete blood count parameters
  - *Total red cells:* ↑
  - *Hemoglobin:* ↑
  - *Hematocrit:* ↑

- *Mean corpuscular volume:* unchanged
- *Mean corpuscular hemoglobin concentration:* ↑

Exercise at altitude

- Adaptations normally serve to achieve homeostasis at rest → unless fully acclimatized intense physical activity → homeostasis loss → severe hypoxia
- This transient intentional hypoxia can be exploited by athletes → further adaptive changes to altitude → blood with greater oxygen carrying capacity → improved performance at lower altitude
- Late phase acclimatization of skeletal muscle includes: increased capillary concentration, increased myoglobin amount, increased mitochondria number, increased aerobic metabolism enzyme concentration

PHYSIOLOGICAL ACCLIMATIZATION TO HIGH ALTITUDE OVERVIEW

	RESPONSE
ALVEOLAR P_{O2}	↓ (lower barometric pressure → lower atmospheric P_{O2})
ARTERIAL P_{O2}	↓ (hypoxemia)
ARTERIAL pH	↑ (respiratory alkalosis due to hyperventilation)
HEMOGLOBIN CONCENTRATION	↑ red blood cell concentration
2,3-DPG CONCENTRATION	↑
MUSCLE METABOLISM	↑ efficiency of aerobic metabolism
O_2-HEMOGLOBIN DISSOCIATION CURVE	Right shift (more oxygen unloaded to tissues)
PULMONARY ARTERIAL PRESSURE	↑ (secondary to increased pulmonary vascular resistance)
PULMONARY VASCULAR RESISTANCE	↑ (vasoconstriction)
VENTILATION RATE	↑

ACUTE MOUNTAIN SICKNESS

- AKA altitude sickness
- Commonly associated with altitudes above 2400m/7800ft
  - Minor symptoms may occur at as low as 1500m/5000ft
  - *Death zone:* 5500m/18000ft, altitude considered incompatible with human life; acclimatization not possible
- Caused by sudden transition to altitude without sufficient acclimatization → low atmospheric pressure → low P_{O2} → hypoxia
- Contributing factors
  - Rate of ascent
  - Rate of water vapor loss from lungs
  - Activity level
- Sudden increase in altitude without taking time to acclimatize

Symptoms

- Headache, shortness of breath, nausea, dizziness, peripheral edema

Complications

- Severe complications of high altitude can be fatal
- High altitude pulmonary edema (HAPE)
  - Low atmospheric pressure → decreased oxygen partial pressures, poor oxygenation → increased pulmonary arterial, capillary pressures, idiopathic increase in permeability of vascular endothelium → fluid extravasation → pulmonary edema
- High altitude cerebral edema (HACE)
  - Hypoxia → increased cerebral microvascular permeability, failure of cellular ion pumps → vasogenic, cytotoxic edema

Treatment

- Supplemental oxygen/immediate descent