Chapter 23: The Respiratory System

Respiratory System Functions

--Gas Exchange: Oxygen enters blood and CO₂ leaves
--Regulation of blood pH: altered by changing blood carbonate dioxide levels
  *also regulated by the renal system
--Voice Production: movement of air past vocal folds makes sound and speech
--Olfaction: smell occurs when airborne molecules drawn into nasal cavity
--Protection: against microorganisms by preventing entry and removing them

Anatomical Respiratory System Divisions

--Upper tract: above the larynx
  *nose, pharynx, and associated structures
--Lower tract: below the voice cords
  *larynx, trachea, bronchi, lungs

Functional Respiratory System Divisions

--Conducting system: conducts the air
  *series of cavities and tubes
  *Includes: nose, pharynx, larynx, trachea, bronchi, bronchioles, and terminal bronchioles
  *NO GAS EXCHANGE, ANATOMICAL DEAD SPACE
--Respiratory portion: where GAS EXCHANGE OCCURS
  *Includes: respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli
  *Physiological dead space: when something is wrong with some part of the respiratory portion and the alveoli d/n function properly anymore

Respiratory System Anatomy

--External Nose/Nasal Cavity
  *Functions:
    a. passageway for air
    b. cleans the air: pseudostratified ciliated columnar epithelium with goblet cells lines nasal cavity
    c. humidifies, warms air: due to high vascularity
    d. smell: olfactory epithelium for sense of smell
    e. along with paranasal sinuses are resonating chambers for speech
  *Respiratory defense system: traps virtually all particles larger than 10 um
    a. mucous moistens air and traps dust, contains lysozyme (digest bacteria cell wall) and defensins (natural antibiotics)
    b. cilli move mucous towards pharynx
    c. large quantity of dust, allergens, pathogens cause a rapid increase in
mucus production (running nose)

*Paranasal Sinuses
  a. found in ethmoid, sphenoid, frontal, and maxillary bones
  b. Function: lighten skull and resonate voice (voice changes when one has a stuffy nose)

--Internal Nose Structures
  *roof is ethmoid bone and floor is hard palate
  *internal nares are opening into pharynx
  *bony swellings/CONCHAE on lateral walls: slow down velocity of air/more time to collect particles, increase surface area to warm air

--Pharynx
  *common opening for digestive and respiratory systems
  *extends from internal nares to cricoid cartilage (skeletal muscle and mucous membrane)
  *Functions:
    a. passageway for food, liquid, air
    b. resonating chamber for speech production
    c. tonsil (lymphatic tissue) in the walls protects entryway into body

*Three regions
  a. nasopharynx: from conchae to soft palate (opening of auditory tube from middle ear, adenoids/pharyngeal tonsil in roof)
    --passageway for air only
  b. oropharynx: from soft palate to epiglottis (fauces is opening from mouth into oropharynx, palatine tonsils in side walls, lingual tonsils in tongue)
    --common passage for food and air
    ---**stratified squamous epithelium** (b/c of constant abrasion from food)
  c. laryngopharynx: from epiglottis to cricoid cartilage
    --common passageway for food and air

--Larynx
  *cartilage and CT tube, anterior to C4-C6, constructed of 3 single and 3 paired cartilage
  *Functions:
    a. maintain an open passageway for air movement
    b. epiglottis and vestibular folds (false vocal cords) prevent swallowed material from moving into larynx
    c. vocal folds are primary source of sound production

*Cartilages of the Larynx
  a. Thyroid cartilage: Adam’s apple
  b. Epiglottis: leaf-shaped piece of elastic cartilage (during swallowing,
epiglottis bends to cover glottis)
c. Cricoid cartilage: ring of cartilage attached to top of trachea
d. Pair of arytenoid cartilages: many muscles responsible for their movement, partially buried in the vocal folds

*Vocal Cords
a. False vocal cords/ventricular folds: found above true vocal cords
b. True vocal cords: attach to arytenoid cartilages

*The Structures of Voice Production
a. True vocal cord contains both skeletal muscle and an elastic ligament (vocal ligament)
b. When 10 intrinsic muscles of larynx contract, move cartilages and stretch vocal cord tight
c. When air is pushed past tight ligament, sound is produced (the LONGER and THICKER vocal cord in male produces a LOWER pitch)
d. The tighter the ligament, the higher the pitch
e. to increase volume of sound, push air harder
f. cords open to breath, close together for sound production

*Speech and Whispering
a. Speech: modified sound made by the larynx
b. Requires pharynx, mouth, nasal cavity, and sinuses to resonate sound produced by the larynx
c. Tongue and lip forms words
d. Pitch: controlled by tension on vocal folds (pulled tight produces higher pitch)
e. Whispering: forcing air through almost closed rima glottidis, oral cavity alone forms speech, controlled by force of air through cords

--Trachea
*5” long, 1” in diameter
*from larynx to T5
*anterior to esophagus and splits into bronchi

*Layers:
a. mucosa: pseudostratified columnar with cilia (that point to pharynx) and goblet cells
b. submucosa: loose CT and seromucous glands
c. hyaline cartilage: 16-20 incomplete rings (loose in the back to allow food to flow down esophagus, must have cartilage to keep airways open at all times), closed by trachealis m., internal ridge called carina (high number mechanorecepotors → cough reflex)
d. adventitia: binds trachea to other organs/holds it in place

--Tracheobronchial Tree
   *Conducting Zone: trachea to terminal bronchioles
      a. ciliated for removal of debris
      b. passageway for air movement
      c. cartilage holds tube system open and smooth muscle controls tube diameter
   *Respiratory Zone: respiratory bronchioles to alveoli
      a. site for gas exchange

--Bronchi and Bronchioles
   *primary bronchi supply each lung (Right is wider and straighter than left)
   *secondary bronchi supply each lobe of the lungs (3R, 2L)
   *tertiary bronchi supply each bronchopulmonary segment
   *repeated branching called bronchioles (have smooth muscle, not cartilage)

--Histology of Bronchial Tree
   a. epithelium changes from pseudostratified ciliated columnar to nonciliated simple cuboidal as air passes deeper into lungs
   b. incomplete rings of cartilage replaced by rings of smooth muscle (spasm of distal bronchiole sm=asthma) then CT
   c. SNS and adrenal gland release epi that RELAXES airway smooth m. and dilates airways

--Respiratory bronchiole
   *simple squamous epithelium

--Alveolar ducts
   *surrounded by alveolar sacs (2 or more alveoli sharing a common opening) and alveoli

--Alveoli
   *surface area is about 75 m²
   *Cell Types of Alveoli
      a. Type I/squamous pneumocytes: simple squamous cells where gas exchange occurs, 95% of alveolar surface
      b. Type II/septal or great pneumocytes: free surface has microvilli, secrete alveolar fluid containing surfactant, 5% of lveolar surface, can differentiate into type I to repair damaged pulmonary membrane
      c. Alveolar dust cells: wandering macrophages that remove debris, removed by mucociliary escalator upward to the pharynx (clear and swallow over 2 million alveolar macrophages/hour)

Alveolar-Capillary Membrane (the blood/gas interface)
--0.5um thick respiratory membrane
--4 layers of membrane to cross
  *dissolve in aqueous fluid in alveoli (with surfactant)
    a. alveolar epithelial wall of type I cells
    b. alveolar epithelial basement membrane
    c. capillary basement membrane
    d. endothelial cells of capillary (continuous)
  *through RBC membrane to bind with Hb
--if thick layer of fluid in alveoli this will lead to hypoxia before hypercapnia
--vast surface area

**Pleural Membranes/Cavity**

--Pleural Membrane: serous membranes 2 layers
  *visceral pleura: covers lungs
  *parietal pleura: lines ribcage and covers upper surface of diaphragm
--Pleural cavity: potential space between ribs and lungs
  *vacuum b/w 2 pleuras w/ a lil bit of fluid → when move it opens the lungs
  *Pleura fluid: produced by pleural membranes, acts as lubricant, helps hold parietal and visceral pleural membranes together
  → loss of fluid causes pleurisy (painful condition where 2 pleural membranes rub against one another)
--Intrathoracic Pressure: always subatmospheric (756mmHg)
  *helps keep parietal and visceral pleura stick together (keeps vacuum, keeps lungs open at all times)
--Function of Pleural Fluid
  1. Decrease surface tension and prevent rubbing
  2. Keep vacuum via intrathoracic pressure to prevent lung collapse

**4 Stages of Respiration**

1. *Pulmonary Ventilation*: movement of air into and out of lungs/based on pressure differences
   *air moves into lungs when pressure inside lungs is LESS than atmospheric pressure (negative pressure breathers)
   *air moves out of lungs when pressure inside lungs is GREATER than atmospheric pressure (760mmHg)

2. *External Respiration*: gas exchange between air in lungs and blood

3. *Transport* of oxygen and carbon dioxide in the blood
4. **Internal Respiration/Cellular Respiration**: gas exchange between the blood and tissues

**Changing Alveolar Volume**

--Lung recoil: causes alveoli to collapse resulting from elastic recoil and surface tension
  *surfactant: reduces tendency of lungs to collapse
--Pleural pressure: negative pressure can cause alveoli to expand
--Boyle’s Law: $P_1V_1 = P_2V_2$
  *as the size of closed container decreases, pressure inside is increased (inverse relationship) V down, P up
  *thoracic volume is increased by sternum going anterior and ribs moving laterally, CONTRACTION of diaphragm flattens the dome and increase vertical dimension of chest

*INSPIRATION*: volume increases, pressure decreases below atmospheric pressure, air moves from high to low, active process b/c diaphragm must contract
  → quiet inspiration: diaphragm moves 1cm and ribs lifted by contraction of external intercostal mm., requires neuronal input…phrenic n./intrathoracic pressure falls and 2-3 liters inhaled
  *EXPIRATION*: relaxation of diaphragm, dome shape returns, decreased volume, increased pressure, pressure higher than atmospheric pressure, air exits lungs
  → quiet expiration: passive process with no muscle action, elastic recoil and surface tension in alveoli pulls inward, alveolar pressure increases and air is pushed out

--Labored Breathing
  *Forced inspiration: use accessory inspiration muscles (SCM, scalenes, pec minor to lift chest upward)
  *Forced expiration: use accessory expiration muscles (abdominal mm for diaphragm up, internal intercostals depress ribs)

**Respiratory Pressures**

1. Alveolar: within alveoli
  *pressure decreases and air rushes in
  *pressure increases and air rushes out
2. Pleural/intrapleural: within pleural cavity, most negative at end of inspiration (biggest vacuum, most tension needed to keep lungs from collapsing), least negative at end of expiration

3. Transpulmonary: the difference between the two; a measure of elastic forces/recoil

**Compliance of the Lungs**

--Ease with which lungs and chest wall expand depends upon elasticity of lungs and surface tension
   *the greater the compliance, the easier it is for a change in pressure to cause expansion
   *a lower-than-normal compliance means the lungs and thorax are harder to expand
--Some diseases reduce compliance (c/n easily expand lungs—inhale)
   *tuberculosis forming scar tissue
   *pulmonary edema: fluid in lungs
   *reduced surfactant
   *paralysis
   *lung fibrosis

**Elasticity of the Lungs**

--2 Basic forces determine the elasticity
   1. Elastic forces of lung tissue itself: determined by amount of elastin and collagen fibers in lung parenchyma
   2. elastic forces caused by surface tension: fluids that line alveolar walls
--quiet exhalation

**Alveolar Surface Tension**

--Law of Laplace P=2T/r
   *”critical closing pressure”
   *inverse relationship between pressure and radius (greater the radius the lesser the pressure needed to keep alveoli open)
   *surfactant: decreases tension which causes pressure to decrease
--If no surfactant: air moves from smaller to larger alveoli and the smaller collapses
--Surfactant Functions
   a. decrease surface tension proportionally to surface area b/c have different size alveoli
   b. decrease overall surface tension
--The decrease in surface tension by surfactant is proportionate to surface area
--Pneumothorax
  *injuries to chest wall that let air enter intrapleural space
  *leads to a collapsed lung (atelectasis) on the same side

**Airway Resistance**

--resistance to airflow depends upon airway size
  *increase size of cheat: airways increase in diameter
  *contract smooth muscles in airways: decrease in diameter
--Disorders increasing resistance
  *airway obstructions or collapse (emphysema, chronic bronchitis)

**Work of Breathing**

1. Compliance/elastic work
   *the GREATEST amount of work
   *work required to expand lung against chest and lung elastic forces

2. Tissue resistance work
   *the LOWEST amount of work expended
   *work required to overcome viscosity of lung and chest

3. Airway resistance work
   *a little higher than tissue resistance work
   *work required to overcome airway resistance during air movement
      w/I the airways

4. Work Varies during heavy breathing or diseases
   *Normal exhalation: no muscle work
   *heavy breathing: muscle work does occur
   *disease: have different work requirements

**Pulmonary Volumes**

--Tidal Volume: volume of air inspired or expired during a normal inspiration or expiration
  *inc. during exercise
--Inspiratory Reserve Volume: amount of air inspired forcefully after inspiration of normal tidal volume
  *dec. during exercise
--Expiratory Reserve Volume: amount of air forcefully expired after expiration of normal tidal volume
  *dec. during exercise
--Residual Volume: volume of air remaining in respiratory passages and lungs after the most forceful expiration
  *same

**Pulmonary Capacities**

--Inspiratory Capacity (2 volumes)
  *tidal volume plus inspiratory reserve volume
--Functional residual capacity (2 volumes)
  *expiratory reserve volume plus the residual volume
--Vital capacity (3 volumes)
  *sum of inspiratory reserve volumes plus expiratory reserve volumes plus tidal volume
  *same during exercise
--Total Lung capacity (4 volumes)
  *total of vital capacity plus residual volume
  *same during exercise

**Minute and Alveolar Ventilation**

--Minute Ventilation: total amount of air moved into and out of respiratory system/minute
  *Respiratory rate/frequency: number of breaths taken/minute
  *Anatomic dead space: part of respiratory system where gas exchange does not take place
  *Physiological dead space: part of respiratory zone where there is no gas exchange due to ventilation/perfusion deficiencies
--Alveolar ventilation: how much air/minute enters the parts of the respiratory system in which gas exchange takes place
  *difference from minute ventilation b/c some volume is in physiological dead space and not available for gas exchange
--minute > alveolar
--Spirometer: measure volumes of air

**Dynamic Component of Exhalation**

--Forced Vital Capacity (FVC): total volume that is forcibly expired (vital capacity)
--FEV\(_1\): the forcefully expired volume (as a % of FVC) in the first second of exhalation
--FEV\(_1\) is about 80% of FVC
  *Obstructive Disease: decrease in elasticity
    ⇒ lung volumes will inc. b/c body is still making gas (CO\(_2\)) the problem is c/n blow air out b/c dec. in elasticity and c/n recoil and push air out...leads to inc. in residual volume (asthma)
  → FEV\(_1\) is decreased to 66%
*Restrictive Disease: decrease in compliance
  → not being able to open up/expand lungs to INHALE
  → ALL volumes are decreased, but proportionally
  → FEV1 stays pretty much the same (may be slightly increased)

**Blood Flow Through Lungs**

--Double blood supply: lungs receive blood via pulmonary (gas exchange) and bronchial (provides oxygenated blood to organs)
  *lower pressure in pulmonary system than in systemic circulation
  *pulmonary blood vessels constrict in response to hypoxia
--Venous drainage returns all blood to heart
--Pulmonary Blood Flow (uneven throughout lung due to gravity)
  1. Zone 1: no blood flow during cardiac cycle
     --alveolar pressure exceeds arterial pressure and venous pressure
     --only seen during severe hemorrhage (drop in blood hydrostatic pressure) and during positive pressure ventilation → when alveolar pressure is increased or arterial pressure is reduced
  2. Zone 2: intermittent blood flow (flow during systole and less/no flow during diastole)
     --under normal condition seen in upper 2/3 of lungs
     --arterial pressure exceeds alveolar pressure and alveolar pressure exceeds venous pressure
  3. Zone 3: Continuous blood flow during all phases
     --both arterial and venous pressures exceed alveolar pressure
     **pressure greater at base than apex
     **perfusion/blood flow greater at base than apex
     **ventilation greater at base
     **high altitudes → alveolar pressure decreases → easier for blood to pass → deeper zones

--V/Q Ratios
  *At apex: ventilation the greatest therefore high V/Q ratio
  *At base: perfusion the greatest therefore low V/Q ratio
  *Conditions with LOW V/Q ratio
    a. loss of VENTILATION, normal perfusion
    b. physiological dead space: space not ventilated V/Q = 0
    c. Ex: partially collapsed lung (atelectasis) → dec. V → dec. ratio
  *Conditions with HIGH V/Q ratio
    a. loss of PERFUSION, normal ventilation
    b. physiological dead space: space not perfused V/Q = infinity
c. Ex: embolism → dec. Q → inc. ratio
generalized hypoxia → dec. Q → inc. ratio

Shunts

--Matching lung airflow and blood flow is not perfect (wasted air and wasted blood)
--Types of Shunts
  a. Anatomic: area where no gas exchange can occur (conducting zones)
  b. Physiological: blood flow to unventilated area of the lung (pneumothorax, pneumonia)
  c. Right-to-Left: blood bypasses pulmonary circulation (transposition of great vessels, atrial septal defect, atelectasis)
  d. Left-to Right: blood bypasses systemic circulation, can cause pulmonary hypertension (patent ductus arteriosus)
--Venous admixture: mixing of oxygenated and non-oxygenated blood (2 types)
  1. Anatomical shunts: blood bypasses alveoli through a channel
     *In R-to-L shunt bronchial circulation drains deoxygenated blood directly into oxygenated pulmonary veins (ALWAYS causes hypoxemia which c/n be correct with oxygen administration)
     --Ex: heart interatrial or interventricular defect (signs are cyanosis)
     *L-to-R shunts d/n cause hypoxemia, oxygenated blood returns to lungs, may cause ventricular hypertrophy
     --Ex: patent ductus arteriosus
  2. Low regional ventilation/perfusion ratio
     *insufficient ventilation fails to fully oxygenate blood

Laws

1. Poiseuille’s law: \( Q = \frac{\Delta P}{R} \) (resistance inversely effects flow)
   *Pulmonary vascular resistance FALLS with INCREASED arterial pressure and INCREASED cardiac output (opposite of systemic circulation)
   *Due to 2 local mechanisms
     a. Capillary recruitment: vessels in parallel, add more channels so total resistance decreases
     b. Capillary distension: dec. velocity of blood flow to have more time for efficient gas exchange
   *low oxygen (hypoxemia)INCREASES pulmonary vascular resistance by vasoconstriction → increased pulmonary vascular resistance may lead to right sided heart failure (cor pulmonae)
     a. Regional Alveolar Hypoxia: hypoxia is localized to specific region and vasoconstriction in that area diverts blood from poorly ventilated area
b. Generalized Alveolar Hypoxia: causes vasoconstriction throughout both lungs (high altitude or chronic disease when pO$_2$ is decreased) leads to pulmonary hypertension and right heart hypertrophy

2. General Gas Law: PV = nRT

3. Laplace’s Law: \( P = \frac{2T}{r} \)

4. Boyle’s Law: \( P_1V_1 = P_2V_2 \)
   * a special case of the general gas law

5. Dalton’s Law: partial pressure
   * the pressure exerted by each type of gas in a mixture
   * total pressure is the sum of ALL partial pressures (O$_2$ is about 20%)

   * concentration of a gas in a liquid is determined by its partial pressure and its solubility coefficient
   * quantity of a gas that will dissolve in a liquid depends upon the amount of gas present and its solubility coefficient
   -- CO$_2$ is the most soluble
   -- N is not soluble (if deep dive and increase pressure, force N to dissolve, leads to nitrogen narcosis)
   -- O$_2$ soluble (breathing O$_2$ under pressure dissolves more O$_2$ in blood)

7. Charles’s Law: the volume of the gas is DIRECTLY proportional to its temperature

8. Fick’s Law: \( V = \frac{(D \times A \times \Delta P)}{x} \)
   * conservation of mass
   * oxygen consumed = oxygen coming in from pulmonary veins minus oxygen returning to lungs in pulmonary arteries
   * dependent on membrane’s thickness (X), diffusion coefficient of gas, surface areas of membrane, partial pressure of gases in alveoli and blood

**Physical Principles of Gas Exchange**

-- diffusion of gases through the respiratory membrane depends on: membrane’s thickness, the diffusion coefficient of gas, surface areas of membrane, partial pressure of gases in alveoli and blood

-- relationship between ventilation and pulmonary capillary flow
  a. increased ventilation or increased pulmonary capillary blood flow increases gas exchange
  b. physiologic shunt is deoxygenated blood returning from lungs
Rate of Diffusion of Gases

--Depends upon partial pressure of gases in air
--Large surface area of alveoli
--Small diffusion distance
--Solubility and molecular weight of gases

*CO₂ dissolves 24x more easily in water than O₂ so net outward diffusion of CO₂ is much faster
*disease produces hypoxia before hypercapnia

--External Respiration
*gases diffuse from areas of high partial pressure to areas of low partial pressure → exchange of gas between air and blood → deoxygenated blood becomes saturated

--Internal Respiration
*exchange of gases between blood and tissues → conversion of oxygenated blood into deoxygenated → diffusion of O₂ inward → diffusion of CO₂ outward

Oxygen Transport in Blood

--98.5%: oxyhemoglobin: oxygen and hemoglobin inside RBC
*different efforts needed to remove oxygen based on how many hemes have oxygen
*people are OK at high altitudes and with some disease b/c big change in oxygen leads to little change in saturation levels
*hemoglobin and oxygen partial pressure dissociation curve is sigmoid
*during exercise: hemoglobin releases more oxygen (25% release to 75%)
*Oxygen binging to Hb is allosterically regulated by:
  a. O₂ binding/cooperativity
  b. pCO₂
  c. pH/[H+]
  d. Temperature (inc. in temp shifts curve to right)
  e. Activators and inhibitors (2,3-BPG)

*left shift of curve due to:
  a. increase in pH, decrease in H+
  b. dec. in CO₂
  c. decreased in temperature
  →results in an INCREASE in the ability of hemoglobin to hold oxygen

*Right shift of curve due to:
  a. decrease in pH, increase in H+
  b. increase in CO₂
c. increase in temperature
d. increase in 2,3-BPG
→ results in DECREASE in the ability of hemoglobin to hold oxygen
→ fetal hemoglobin has higher affinity for oxygen than does maternal
--1.5%: transported dissolved in blood

**Bohr Effect**

--Effect of pH on binding Affinity
--As acidity increases, O\(_2\) affinity for Hb decreases
  *H+ binds to hemoglobin and alters it
  *Oxygen left behind in needy tissues

**Carbon Monoxide Poisoning**

--Binds to Hb heme group more successfully than O\(_2\)
  *much higher affinity
--bright red coor of lips, oral mucosa
--treatment: administer pure O\(_2\) to speed up the release of CO

**Carbon Dioxide Transport**

--Carried by the blood in 3 different ways
  1. dissolved in plasma (7%)
  2. combined with the globin part of Hb molecule forming carbaminohemoglobin (23%)
  3. as part of bicarbonate ion (70%)
--Haldane effect: hemoglobin that has released oxygen binds more readily to carbon dioxide than hemoglobin that has oxygen bound to it
--Cl- Shift: see picture

**Respiratory Areas in Brainstem**

--Medulla Rhythmicity Respiratory Center
  *Dorsal Group: INSPIRATORY, 2 seconds, stimulate the diaphragm and external intercostals via phrenic nerves (C3-C5)
    → autorhythmic cells active for 2 seconds then inactive
  *Ventral group: EXPIRATORY, 3 seconds, stimulate the internal intercostal and abdominal muscles
--Pons
  *Pneumotaxic respiratory center
    a. inhibits inhalation
    b. controls transition between inspiration and expiration
    c. provides constant inhibitory impulses to inspiratory area to shorten the inspiration preventing overinflation of lungs
d. neurons trying to turn off inspiration before lungs become too expanded
e. active pneumotaxic area tends to increase rate of respiration

*apneustic respiratory center
  a. promotes inhalation
  b. stimulatory signals to inspiratory area to prolong inspiration

--Hering-Breuer Reflex (inflation reflex)
  *big deep breath stretches receptors in bronchi and bronchioles producing urge to exhale
  *limits the degree of inspiration and prevents overinflation of the lungs
    a. Infants: plays a role in regulating basic rhythm of breathing and preventing overinflation of lungs
    b. Adults: important only when tidal volume is large (exercise)

Chemical Regulation of Respiration

--Central chemoreceptors in medulla
  *respond to changes in H+, pCO$_2$

--Peripheral chemoreceptors
  *respond to changes in H+, pCO$_2$, and pO$_2$
  *found in the aortic body (CN X) and carotid bodies (CN IX)

--pCO$_2$ is major stimulus for breathing

Regulation of Ventilation Rate and Depth

--Ventilation rate and depth increases with:
  a. increase in arterial blood H+ levels or pCO$_2$ levels (central and peripheral)
  b. decrease in pO$_2$ arterial blood levels (peripheral chemoreceptors)
  c. decrease in blood pressure (baroreceptors)
  d. increase in body temperature
  e. prolonged pain

--Ventilation rate and depth decreases with:
  a. decrease in arterial blood H+ levels or pCO$_2$ levels (central and peripheral)
  b. increase in pO$_2$ levels (peripheral)
  c. increase in blood pressure (baroreceptors)
  d. decrease in body temperature
  e. severe pain causes apnea