What is the Function of the Pulmonary System?
Pulmonary Function

- Attain O2 from environment
- Deliver O2 to circulatory system
- Remove CO2 from the body
- Acid-base balance
- Phonation
- Olfaction
- Modification of inspired air
- Pulmonary defense
- Metabolism
PULMONARY FUNCTION

- VENTILATION
  - Moving O\textsubscript{2}/CO\textsubscript{2} b/t lungs & environment

- DIFFUSION
  - Moving O\textsubscript{2}/CO\textsubscript{2} b/t lungs & capillaries

- TRANSPORT
  - Moving O\textsubscript{2}/CO\textsubscript{2} b/t capillaries & tissues

- REGULATION
CONTENTS

- VENTILATION
- DIFFUSION
- STRUCTURE & FUNCTION
- PULMONARY FUNCTION TESTS
- MECHANICAL VENTILATION
VENTILATION

What muscles work during normal quiet inspiration?
VENTILATION

- Normal quiet breathing is performed by the diaphragm only.
  - Diaphragm contracts down
  - Lengthens the chest
  - Increases negative pressure
  - Draws air into the lungs
What muscles work during normal quiet expiration?
VENTILATION

There are NO muscles that WORK during normal quiet expiration.

Expiration occurs when the diaphragm RELAXES or RECOILS upward:
- shortens the chest
- reduces the negative pressure
- expels the air
VENTILATION

What muscles assist with heavy breathing?
VENTILATION

- **Rib Cage**
  - Natural position: ribs slanted down
  - External intercostal muscles contract → Inspiration
    - Rib cage elevates
    - Pulls the sternum away from the spine
    - Increases the A-P diameter by 20%
  - Internal intercostal muscles contract → Expiration
    - Rib cage depresses
    - Pulls the sternum down
    - A-P diameter decreases
VENTILATION

- **Muscles of Heavy Inspiration: Raises the Rib Cage**
  - External Intercostals
  - SCM (lifts the sternum)
  - Anterior Serratus (lifts the ribs)
  - Scalene Muscles (lifts 1\textsuperscript{st} two ribs)

- **Muscles of Heavy Expiration: Depresses the Rib Cage**
  - Abdominal Recti
  - Internal Intercostals
Muscles can change the shape and therefore the pressure inside the thoracic cavity to assist with breathing.
VENTILATION

Pleural Pressure

- Continual suction of excess fluid into lymphatics
- Normally a slightly negative pressure
  -5 cm H₂O
  (The amount required to keep lungs open at the resting level)

- Normal inspiration: ↑ pleural pressure
  -7.5 cm H₂O
VENTILATION

Alveolar Pressure

Pressure inside the lung alveoli

- With no airflow and open glottis,
  alveolar pressure = atm pressure = 0

- Normal inspiration→ -1 cm H$_2$O
- Normal expiration→ +1 cm H$_2$O
VENTILATION

- Alveolar Pressure change of -1 cm H$_2$O AND
- Pleural Pressure change of -2.5 cm H$_2$O

→ Moves 500 mL air into the lungs
VENTILATION

Transpulmonary Pressure
Alveolar Pressure – Pleural Pressure
Measure of the elastic forces
≈ Recoil Pressure
What is the measure of how well the lungs expand for the pressure applied?
VENTILATION

COMPLIANCE

- Normal compliance for the lung = 200 ml / cm H₂O

- Two Factors:
  - Elastic Forces of the Lung Tissue
  - Elastic Forces of the Surface Tension
Elastic Forces of Lung Tissue

only 1/3 contribution to compliance

depends on elastin & collagen fibers in the lung parenchyma
Elastic Forces of Surface Tension

2/3 contribution to compliance

What is Surface Tension?

Water on the surface sticks together by contracting together

ex: Raindrops
VENTILATION

- What about water in the alveoli??

  The thin layer of water inside the alveoli:
  - contracts together
  - tries to push the air out
  - collapses the alveoli

- Surface Tension $\approx$ Elastic Force
What agent decreases the surface tension inside alveoli??
VENTILATION

Surfactant
Made by Type II alveolar epithelial cells
Contains: phospholipids, proteins, ions
Key Ingredients:
Dipalmitoylphosphatidylcholine
Apoproteins
Calcium ions
Dipalmitoylphosphatidylcholine:

- One portion hydrophilic—spreads over alveolar water
- One portion hydrophobic—orient toward air in alveoli
- Does not dissolve in fluid
- Decreases the normal surface tension by 1/12 to 1/2
VENTILATION

- Apoproteins & Calcium ion
  - Allow phospholipid to spread faster over alveolar surface.
  - Surfactant not functional without them
VENTILATION

- **Surface Tension**
  - Pure Water: 72 dynes / cm
  - Alveoli: 50 dynes / cm
  - Alveoli w/ Surfactant: 5-30 dynes / cm
## VENTILATION

### Factors of Inspiration
- Muscles
- Pleural Pressure
- Alveolar Pressure
- Compliance
- Surfactant

### Factors of Expiration
- Collapse Pressure
- Surface Tension
- Recoil Pressure
- Muscles

### Measures
- Lung Volumes / Capacities
- Minute Ventilation
- Alveolar Ventilation
- Dead Space
VENTILATION

Collapse Pressure

\[ \text{Collapse Pressure} = 2 \times \text{Surface Tension} \times \frac{1}{\text{Radius}} = 4 \text{ cm H}_2\text{O} \]

The smaller the alveolus, the greater the collapse pressure
In premature babies:
- Alveoli are usually ¼ the normal size
- Surfactant is secreted b/t 6th & 7th gestational month
- Therefore, a premature baby could have a collapse pressure of > 30 mm Hg

- RESPIRATORY DISTRESS SYNDROME OF THE NEWBORN
VENTILATION

- Compliance of Lung-Thorax system is \( \frac{1}{2} \) that of lungs alone (200 ml / cm H\(_2\)O)

110 ml / cm H\(_2\)O

Therefore, it takes 2x the energy for normal inspiration
VENTILATION

- Compliance Work
- Tissue Resistance Work
- Airway Resistance Work
VENTILATION

- Compliance Work
  - Overcomes elastic forces
  - $= \Delta \text{Volume} \times \Delta \text{Pressure} \div 2$
VENTILATION

- Tissue Resistance Work
  - Overcomes the viscosity of the lungs

- Airway Resistance Work
  - Overcomes resistance to airflow thru respiratory passageways
VENTILATION

- Usually compliance >> airway > tissue resistance work
- Usually there is no work during expiration…
- Heavy Breathing: ↑↑airway resistance work
- Pulmonary disease: all work ↑
- Fibrosis: ↑ compliance & tissue resistance work
- Airway Obstruction: ↑ airway resistance work
- Asthma: ↑ airway resistance work in expiration > inspiration
VENTILATION

- Measurements
  - Volumes (4)
  - Capacities (4)
What are the four lung volumes?
VENTILATION

- **Tidal Volume (TV or $V_T$)**
  - Normal breath 500 ml

- **Inspiratory Reserve Volume (IRV)**
  - Maximum Inspiration p $V_T$ 3000 ml

- **Expiratory Reserve Volume (ERV)**
  - Maximum Expiration p $V_T$ 1100 ml

- **Residual Volume (RV)**
  - Air left in lungs p ERV 1200 ml
What are the four lung capacities?
VENTILATION

- Capacities are combos of lung volumes
  - **Inspiratory Capacity (IC)**
    - $V_T + IRV$ \(= 3500\ ml\)
  - **Functional Residual Capacity (FRC)**
    - $ERV + RV$ \(= 2300\ ml\)
  - **Vital Capacity (VC)**
    - $IRV + V_T + ERV$ \(= 4600\ ml\)
  - **Total Lung Capacity (TLC)**
    - $IRV + V_T + ERV + RV$ \(= 5800\ ml\)
VENTILATION

- **Minute Ventilation** \( (V_E) = V_T \times RR \)
  - Normal = 6 L / min

- **Alveolar Ventilation** \( (V_A) \)
  - Normal tidal volume gets most of the air only to the terminal bronchioles
  - The velocity of the air molecules moving a short distance to the alveoli occurs by diffusion
  - Rate at which new air reaches respiratory bronchioles
VENTILATION

- Alveolar Ventilation
  \[ \text{Alveolar Ventilation} = \text{RR} \times (V_T - V_D) \]
  
  Normal = 4200 ml / min

One of the major factors determining concentration of \( O_2 \) and \( CO_2 \) in the alveoli
VENTILATION

- **Dead Space (V_D)**
  - **Dead Space Air**
    - Air that stays in the nose, pharynx, trachea, terminal bronchioles
  - **Dead Space**
    - Respiratory passages where no gas exchange occurs
    - Normal V_D is 150 ml (increasing with age)
VENTILATION

- Anatomic Dead Space
  - Volume of the respiratory passages not in the gas exchange areas

- Physiologic Dead Space
  - Additional volume of air in gas exchange areas that are not functional
VENTILATION

What conditions can increase the physiologic dead space?
VENTILATION

- Things that decrease perfusion:
  - Pulmonary Embolus
  - Pulmonary HTN
  - Decreased Cardiac Output
  - High PEEP
  - ARDS

- Causes high $P_a \text{CO}_2$
VENTILATION

- Alveolar Ventilation
  \[ = RR \times (V_T - V_D) \]
  
  Normal = 4200 ml / min

One of the major factors determining concentration of \( O_2 \) and \( CO_2 \) in the alveoli
# VENTILATION

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DIFFUSION

- Gas Exchange between alveoli and capillaries

- Hydrostatic Pressure difference between the apex and the base of the lung
  \[ \approx 23 \text{ mm Hg difference} \]
Three Zones of Blood Flow

- **ZONE 1**—No Flow: $P_A > P_a$
- **ZONE 2**—Intermittent Flow: $SBP > P_A$ but $DBP < P_A$
- **ZONE 3**—Continuous Flow: $P_a > P_A$
DIFFUSION

- Exercise increases blood flow to the top of the lung ~ 700-800% and to the bottom of the lung ~ 200-300% creating equalization.

- The entire lung becomes Zone 3 blood flow
DIFFUSION

- **V/Q Ratio**
  - Usually 0.8 to 1.2
  - If V/Q ratio increases: (vice versa)
    - There is more O2 in the alveoli that are not delivered to the blood volume
    - $\uparrow P_AO_2$ and $\downarrow P_AC0_2$

- **Shunt**
  - Perfused, but not ventilated
  - Mixed venous blood from the right heart shunted to the left heart without gas exchange
  - Not correct by administration of oxygen
DIFFUSION

- Similar molecular weights b/t O₂ and CO₂
- Diffusion rate of O₂ in the gaseous phase is 1.2 x that of CO₂
- Diffusion rate of CO₂ in the liquid phase is 20 x that of O₂
- CO₂ diffuses until the partial-pressure gradient is equalized
- O₂ diffuses into the blood combining with Hb maintaining the original partial-pressure gradient to saturate all the Hb
DIFFUSION

- Fick’s Law
  - Rate of Gas Diffusion = Rate of Gas Diffusion = Area x Gas Diffusion Coefficient x Pressure Gradient / Barrier Thickness

- \[ V_{\text{gas}} = \frac{A \times D_{\text{gas}} \times \Delta P}{T} \]
DIFFUSION

- Easier to measure diffusion capacity of Carbon Monoxide than Oxygen:
  - $DL_{CO}$
  - Divide uptake of CO by measure $P_aCO$
  - Based on
    - Diffusing Capacity of Alveolar-Capillary Membrane
    - Hb-CO reaction
    - Pulmonary Capillary Blood Flow
DIFFUSION

- O2 content of blood

\[ C_aO_2 = (Hb \times 1.34 \times O_2\text{sat}) + 0.0003 \ P_aO_2 \]
DIFFUSION

- Oxyhemoglobin Dissociation Curve

O$_2$ % saturation vs $P_a$O$_2$

Function of Hb’s changing affinity for O$_2$

Normal p50 = 50% O$_2$sat ~ 27 mmHg
DIFFUSION

- Right Shift:
  - Low pH
  - High temperature
  - High pCO$_2$
  - High levels of 2,3-DPG (↓ affinity of Hb for O$_2$)
  - High ATP

- Encourages O$_2$ offloading making it more available for tissue
DIFFUSION

- Alveolar gas equation
  \[ P_A O_2 = P_{1O_2} - P_A CO_2/0.8 + F \]

- A-a gradient equation
  \[ P_{(A-a)O_2} = P_A O_2 - P_{aO_2} \]
Causes of Hypoxemia

- V/Q mismatch (most common)
- Shunt
- \( \downarrow P_{A\text{O}_2} \)
- Hypoventilation
- \( \uparrow \) Diffusion gradient
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Airways--Nasopharynx

- 10,000 L of Toxic, Infected, Irritating Air
- Functions:
  - Filter
  - Humidify
  - Warm

- **SNIFFING**: abrupt inspiration allowing olfactory system to detect hazard before full breath
Airways—
Tracheobronchial Tree

- 23 Generations of airway branching
  - Conducting Zone: 1-16 generations
  - Transition Zone: 17-19 generations
  - Respiratory Zone: 20-23 generations

- Anatomic Dead Space =
  - Conducting Zone of the airway
  - Nasopharynx, Larynx, Trachea
Tracheobronchial Tree

- Three groups of histology / function
  1. Trachea, major lobar, and segmental bronchi
  2. Membranous and terminal bronchioles
  3. Respiratory bronchioles, alveolar ducts, and alveolar air sacs
Tracheobronchial Tree

- Trachea, Major Lobar Bronchi & Segmental Bronchi
  - Cartilaginous Walls
  - Ciliated, Pseudostratified, Columnar Epithelium
- Mucus-secreting goblet cells
- MUCOCILIARY ESCALATOR
- Submucosal glandular cells secrete lactoferrin (bacteriostatic agent)
Tracheobronchial Tree

- Membranous and Terminal Bronchioles
  - No cartilage
  - Large amt of smooth muscle & elastic fibers
  - Neuroendocrine (APUD) cells
    - Serotonin, Dopamine, Norepinephrine, VIP
  - Mast cells
  - Autonomic Nervous System
Tracheobronchial Tree

- Mast cells
  - Histamine, Lysosomal Enzymes, Leukotrienes, Platelet-Activating Factor, Chemotactic Factors, Serotonin
  - Bronchoconstriction
  - Anaphylactic Reactions
- Immune/Inflammatory Responses of the lung
- Cardiopulmonary Reflexes
Tracheobronchial Tree

- Autonomic Nervous System
  - Vagus → Bronchoconstriction, Glandular Secretion
  - SNS → Bronchodilation
  - Inhibit Glandular Secretion
  - α-receptors: Bronchoconstriction
  - β-receptors: Bronchodilation
  - Circadian Rhythm of Bronchial Smooth Muscles by PSNS: bronchoconstriction in the a.m., bronchodilation in the p.m.
## Tracheobronchial Tree

### Bronchiodilators
- SNS
- \( \beta \)-receptors
- \( \uparrow \text{CO}_2 \)
- \( \downarrow \text{O}_2 \)

### Bronchoconstrictors
- PSNS
- \( \alpha \)-receptors
- thromboxane (plts)
- histamine (mast cells)
- cool temperature
- arterial chemoreceptors
- \( \downarrow \text{CO}_2 \)
Tracheobronchial Tree

- Respiratory Bronchioles & Alveoli
  - Kohn pores
  - Clara cell
  - Type I pneumocytes
    - 95% simple squamous layer on alveolar surface
    - Primary diffusion surface
  - Type II pneumocytes
    - Cuboidal cells
    - Surfactant
    - Regeneration of Type I pneumocytes
- Alveolar Macrophage
Chest Wall

- Bony thoracic cage for protection
- Accessory muscles of respiration
- Maintains negative intrathoracic pressure by the intrinsic outward elastic recoil of the curved ribs
Pleura

- Parietal Pleura
  - Corpuscular sensory endings
  - Free somatic nerve endings
  - Primary Function: absorption via lymphatics
- Visceral Pleura
  - PSNS, SNS innervation
  - Primary Function: lubrication
    - Microvilli produce hyaluronic acid-glycoprotein
- Pleural Fluid
  - Typical Volume = 5-15 ml
  - Typical Turnover = 1-2 L / day
Pulmonary Vasculature

- Pulmonary arteries
  - Entire right ventricle output of mixed venous blood
  - Distensible, low-pressure system
  - SNS—vasoconstriction
  - PSNS—vasodilation
  - Branches follow the airways into alveolar-capillary units for gas exchange
Pulmonary Vasculature

- Inspiration increases total pulmonary vascular resistance
- Resistance is the result of passive forces
  - Distension
  - Recruitment—opening of new, previously closed capillaries from increased perfusion pressure overcoming the critical opening pressure
Pulmonary Vasculature

- **Vasoconstrictors**
  - Histamine
  - Thromboxane
  - Hypoxia
  - Hypercapnia
  - Prostaglandins F & E

- **Vasodilators**
  - Isoproterenol
  - Acetylcholine
  - PGE$_1$
  - PGI$_2$
Pulmonary Vasculature

- Bronchial Arteries
  - Supplied from the aorta, intercostal arteries
  - Oxygenated arterial blood
  - Systemic pressure

- Venous Drainage
  - Bronchial unsaturated blood drains to
    - Azygous-hemiazygous veins
    - Pulmonary veins (anatomic right-to-left shunt)
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PULMONARY FUNCTION TESTS

- Static Lung Volumes
  - Decreased in restrictive lung dz
  - Increased RV, FRC, TLC in obstructive dz (air trapping on expiration)

- Dynamic Lung Characteristics
  - FEV$_1$ Decreased in COPD
  - FVC
  - FEV$_1$/FVC Decreased in COPD, NI/Increased in Restrictive Dz
Pulmonary Function Assessment

- Forced Vital Capacity (FVC)
  - Assessment of muscle strength & airway resistance
  - Measures ability for deep breathing & coughing postoperatively
- Forced Expiratory Volume (FEV)
- $\text{FEV}_1$/FVC
  - Abnormal if < 0.7
  - Increased risk for major surgery if < 0.5
PULMONARY FUNCTION TESTS

- Requirement for thoracic surgery
  - Predicted post-op values
    - $\text{FEV}_1 > 0.8$ (>40% predicted)
    - $\text{DL}_{CO} > 11-12$ ml/min/mmHgCO (>50% predicted)
    - FVC > 1.5 L
  - Pre-op values
    - $\text{PCO}_2 < 45$
    - $\text{PO}_2 > 50$
    - $\text{VO}_2 \text{ max} < 10$
  - V/Q scan for lung contribution to FEV$_1$ if < 2L
MECHANICAL VENTILATION

- Indications
  - Dysfunction:
    - CNS
    - Chest Wall
    - Airway
    - Respiratory Muscles
    - Alveoli
MECHANICAL VENTILATION

- CNS dysfunction
  - GCS < 8 for airway protection
    - Narcotic Overdose
    - Closed Head Injury
- Chest Wall dysfunction
  - Flail chest
  - Open PTX
  - Marked Scoliosis
MECHANICAL VENTILATION

- Airway Dysfunction
  - Facial Trauma
  - Anaphylaxis
  - Foreign Body / Mass
- Respiratory Muscle Fatigue / Disease
- Alveolar Dysfunction
  - Pulmonary Edema
  - Pneumonia
MECHANICAL VENTILATION

- Also
  - PaCO2 > 45
  - PaO2 < 55 (w/ supplemental oxygen)
  - RR > 35

- Always accepted criteria: clinical judgment
MECHANICAL VENTILATION

- Modes
  - Assist Control (AC)
  - Intermittent Mandatory Ventilation (IMV)
  - Pressure Support (PS)
  - Pressure Control (PC)
MECHANICAL VENTILATION

- **AC**
  - Volume given at preset intervals
  - Patient breath triggers volume delivery

- **IMV**
  - Volume given at preset intervals
  - Patient breath not mechanically supported
  - Synchronous setting prevents stacking

- **PS**
  - Positive pressure with each patient effort
  - Volume depends on PS, patient effort, compliance

- **PC**
  - Peak pressure, inspiratory time, RR are set
  - Volume depends on compliance
  - Good for ARDS to decrease barotrauma
MECHANICAL VENTILATION

- **PEEP (Positive End-Expiratory Pressure)**
  - Increases FRC
  - Improves compliance
  - Best way to improve oxygenation

- **Pressure Support**
  - Decreases Work of Breathing
MECHANICAL VENTILATION

- To decrease $P_aCO_2$:
  - Increase Tidal Volume
  - Increase Respiratory Rate

- To increase $P_aO_2$:
  - Increase PEEP
  - Increase $F_iO_2$
MECHANICAL VENTILATION

- Complications
- Too much PEEP
  - Decrease Preload
  - Decrease CO
  - Decrease Renal Blood Flow & UOP
  - Increase PVR
- Too much Oxygen
  - Oxygen radical toxicity if $F_iO_2 > 60-70\%$ for $> 24$ hours
- Barotrauma
  - Plateau pressure $> 30$, Peak pressure $> 50$
MECHANICAL VENTILATION

- Complications
- ET Tube
  - Tracheal Stenosis
  - Laryngeal Injury
  - Tracheomalacia
  - Tracheo-innominate Fistula
- Infection
  - Sinusitis
  - Pneumonia
MECHANICAL VENTILATION

- Weaning Parameters
  - Settings:
    - FiO2 < 35%
    - PEEP 5
    - PS 5
    - RR < 24
  - ABG:
    - pH 7.35-7.45
    - pO2 > 60
    - pCO2 < 50

- Measurements:
  - $V_E < 10$ L/min
  - $V_T > 5$ mL/kg
  - VC > 10 mL/kg
  - Neg Insp Force > 20 cmH2O

- Vital Signs:
  - HR < 120
  - O2 Sat > 93%

- Misc:
  - Off pressors
  - Follows commands
  - Can protect airway
CONCLUSION

- VENTILATION
- DIFFUSION
- STRUCTURE & FUNCTION
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