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#### **MECHANICS OF RESPIRATION**

**Understanding Airway Management....or Not** 

## **Goals of Respiration**

#### **Primary Goals Of The Respiration System**

- Distribute air & blood flow for gas exchange
- Provide oxygen to cells in body tissues
- Remove carbon dioxide from body
- Maintain constant homeostasis for metabolic needs

#### **Functions of Respiration**

#### **Respiration divided into** *four* **functional events:**

Mechanics of pulmonary ventilation
Diffusion of O2 & CO2 between alveoli and blood
Transport of O2 & CO2 to and from tissues
Regulation of ventilation & respiration

# **External & Internal Respiration**

#### **External Respiration**

- Mechanics of breathing
- The movement of gases into & out of body
- Gas transfer from lungs to tissues of body
- Maintain body & cellular homeostasis

#### **Internal Respiration**

- Intracellular oxygen metabolism
- Cellular transformation
- Krebs cycle aerobic ATP generation
- Mitochondria & O2 utilization

### **Pulmonary Ventilation**

# The main purpose of ventilation is to maintain an optimal composition of alveolar gas

- Alveolar gas acts a stabilizing buffer compartment between the environment & pulmonary capillary blood
  - Oxygen constantly removed from alveolar gas by blood
  - Carbon dioxide continuously added to alveoli from blood
  - O2 replenished & CO2 removed by process of ventilation, by simple diffusion.
- The two ventilation phases (inspiration & expiration) provide this stable alveolar environment
- Breathing is the act of creating inflow & outflow of air between the atmosphere and the lung alveoli

# **Physiological Lung Structure**

- Lung weighs 1.5% of body weight
  - 1 kg in 70 kg adult
  - Alveolar tissue is 60% of lung weight
- Alveoli have very large surface area
  - 70 m<sup>2</sup> internal surface area
  - -40 x the external body surface area
- Short diffusion pathway for gases
  - Permits rapid & efficient gas exchange into blood
  - $-1.5 \ \mu m$  between air & alveolar capillary RBC
  - Blood volume in lung 500ml (10% of total blood volume)

# **Respiratory Mechanics**

#### <u>Multiple factors required</u> <u>to alter lung volumes</u>

- Respiratory muscles generate force to inflate & deflate the lungs
- Tissue elastance & resistance impedes ventilation
- Distribution of air movement within the lung, resistance within the airway
- Overcoming surface tension within alveoli

# **The Breathing Cycle**

- Airflow requires a pressure gradient
- Air flow from higher to lower pressures
- During inspiration alveolar pressure is subatmospheric allowing airflow into lungs
- Higher pressure in alveoli during expiration than atmosphere allows airflow out of lung
- Changes in alveolar pressure are generated by changes in pleural pressure

# Inspiration

#### **Active Phase Of Breathing Cycle**

- Motor impulses from brainstem activate muscle contraction
- <u>Phrenic nerve (C 3,4,5)</u> transmits motor stimulation to <u>diaphragm</u>
- <u>Intercostal nerves (T 1-11)</u> send signals to the <u>external intercostal</u> <u>muscles</u>
- Thoracic cavity expands to lower pressure in pleural space surrounding the lungs
- Pressure in alveolar ducts & alveoli decreases
- Fresh air flows through conducting airways into terminal air spaces until pressures are equalized
- Lungs expand passively as pleural pressure falls
- The act of inhaling is <u>negative-pressure ventilation</u>

### **Muscles of Inspiration: Diaphragm**

#### **Most Important Muscle Of Inspiration**

- Responsible for 75% of inspiratory effort
- Thin dome-shaped muscle attached to the lower ribs, xiphoid process, lumbar vertebra
- Innervated by <u>Phrenic nerve (Cervical segments 3,4,5)</u>
- During contraction of diaphragm
  - Abdominal contents forced **downward** & **forward** causing <u>increase</u> in vertical dimension of chest cavity
  - Rib margins are lifted & moved outward causing <u>increase</u> in the transverse diameter of thorax
  - Diaphragm moves **down** 1cm during normal inspiration
  - During forced inspiration diaphragm can move down 10cm
- Paradoxical movement of diaphragm when paralyzed
  - Upward movement with inspiratory drop of intrathoracic pressure
  - Occurs when the diaphragm muscle is denervated

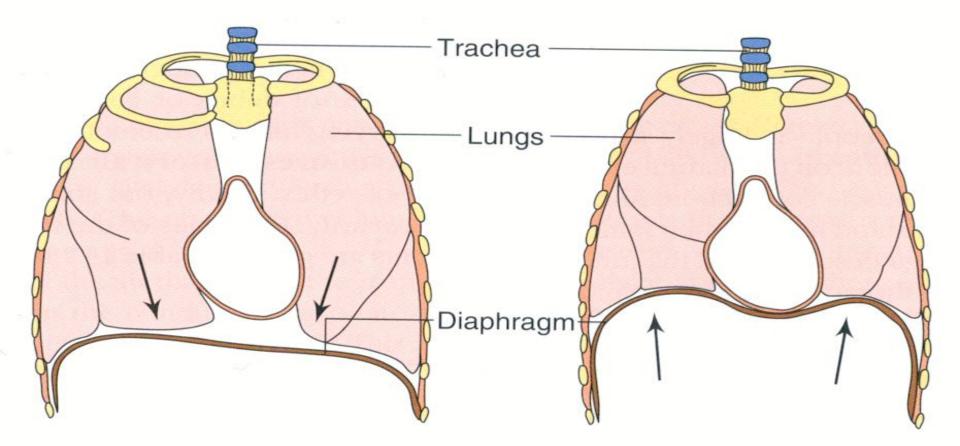
# Diaphragm



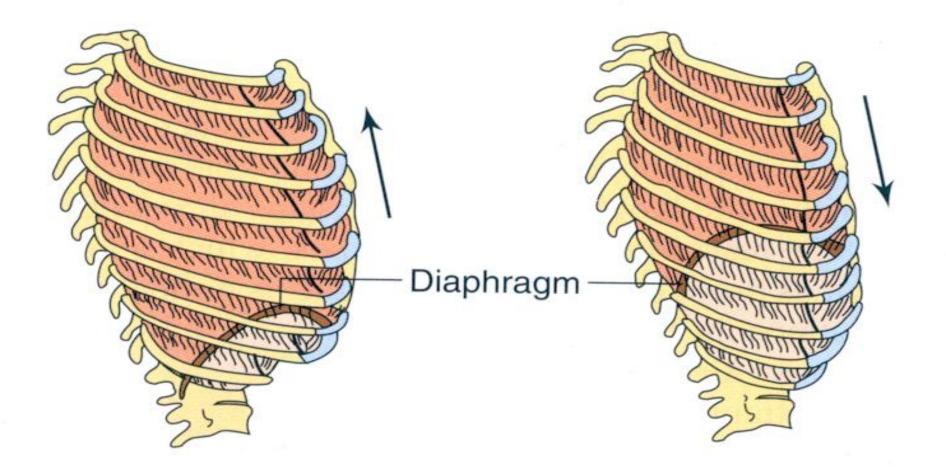
### Movement of Thorax During Breathing Cycle

(a) Inspiration

(b) Expiration



#### **Movement of Diaphragm**



(a) Inspiration

(b) Expiration

### **Transdiaphragmatic Pressure**

- Effect of abdominal pressure on chest wall mechanics is transmitted across the diaphragm
  - Abdominal pressure equal atmospheric pressure in supine position *when* respiratory muscles are relaxed
  - Increasing abdominal pressure pushes diaphragm cephalad into thoracic cavity, decreasing FRC.
    - (FRC=Functional Reserve Capacity)
- FRC reduced by increased intra-abdominal pressure situations
  - <u>Examples</u>: Pregnancy, Obesity, Bowel obstruction, Laparoscopic surgery, Ascites, Abdominal mass, Hepatomegaly, Trendelenburg position, Valsalva maneuver

### **Muscles of Inspiration External Intercostal Muscles**

- The external intercostal muscles connect to adjacent ribs
- Responsible for 25% of inspiratory effort
- Motor neurons to the intercostal muscles originate in the respiratory centers of the brainstem and travel down the spinal cord. The motor nerves leave the spinal cord via the intercostal nerves. These originate from the ventral rami of T1 to T11, they then pass to the chest wall under each rib along with the intercostal veins and arteries.
- Contraction of EIM pulls ribs upward & forward
  - Thorax diameters **increase** in both lateral & anteroposterior directions
  - Ribs move outward in "bucket-handle" fashion
  - Intercostals nerves from spinal cord roots innervate EIMs
- Paralysis of EIM does not seriously alter inspiration because diaphragm is so effective but sensation of inhalation is decreased

### Muscles of Inspiration Accessory Muscles

# These muscles assist with *forced* inspiration during periods of stress or exercise

#### **Scalene Muscle**

- Attach cervical spine to apical rib
- <u>Elevate</u> the first two ribs during forced inspiration <u>Sternocleidomastoid Muscle</u>
- Attach base of skull (mastoid process) to top of sternum and clavicle medially
- <u>Raise</u> the sternum during forced inspiration

### **Expiration**

#### **The Passive Phase Of Breathing Cycle**

- Chest muscles & diaphragm relax contraction
- Elastic recoil of thorax & lungs return to equilibrium
- Pleural & alveolar pressures rise
- Gas flows passively out of the lung
- Expiration active during hyperventilation & exercise

### **Muscles of Active Expiration**

Active expiration requires abdominal & internal intercostals muscle contraction

#### Rectus abdominus/abdominal oblique muscles

- Contraction raises intra-abdominal pressure to move diaphragm upward
- Intra-thoracic pressure raises and forces air out from lung

#### • Internal intercostals muscles

- Assist expiration by pulling ribs downward & inward
- Decrease the thoracic volume
- Stiffen intercostals spaces to prevent outward bulging during straining

#### These muscles also contract forcefully during coughing, vomiting, & defecation

#### **Transpulmonary Pressure**

- The pressure difference between the alveolar pressure & pleural pressure on outside of lungs
- The alveoli tend to collapse together while the pleural pressure attempts to pull outward
- The elastic forces which tend to collapse the lung during respiration is Recoil Pressure

# **The Pleura Space**

- Two parts of the pleural membrane
  - <u>Visceral pleura</u> is a thin serosal membrane that envelopes the lobes of the lungs
  - <u>Parietal pleura</u> lines the inner surface of the chest wall, lateral mediastinum, and most of the diaphragm
- Pleura space enclosed by a continuous membrane
  - The two pleural membranes slide against each other
  - The pleural membranes are difficult to separate apart
  - Separated by a thin layer of serous fluid ( a large amount would be a pleural effusion as seen in CHF, CA, infection)
- Pleura sac
  - The continuous membranes fold to create a sac inferiorly
  - Both pleura line this potential space inclosing a small amount of fluid
- Pleural fluid
  - Functions as a lubricant between the membranes, prevents frictional irritation
  - Causes the visceral & parietal pleura to adhere together, maintains surface tension
  - Lymphatic drainage maintains constant suction on pleura (-5cmH2O)

## **Compliance of the Lungs**

- Compliance is a measure of the distensibility of the lungs
- Compliance = change in lung volume/ change in lung pressure
- $C_{\text{pulm}} = \Delta V_{\text{pulm}} / \Delta P_{\text{pulm}}$
- The extent of lung expansion is dependant on increase of transpulmonary pressure
- Normal static compliance is 70-100 ml of air/cm of H2O transpulmonary pressure
- Different compliances for inspiration & expiration based on the elastic forces of lungs
  - Compliance *reduced* by higher or lower lung volumes, higher expansion pressures, venous congestion, alveolar edema, atelectasis & fibrosis
  - Compliance *increased* with age & emphysema secondary to alterations of elastic fibers

# **Elastic Forces of the Lung**

#### **Elastic Lung Tissue**

- Elastin & Collagen fibers of lung parenchyma
- Natural state of these fibers is contracted coils
- Elastic force generated by the return to this coiled state after being stretched and elongated
- The recoil force assists to deflate lungs

#### **Surface Air-fluid Interface**

- <u>2/3 of total elastic force in lung</u>
- Surface tension of H2O
- Complex synergy between air & fluid holds alveoli open
- Without air in the alveoli a fluid filled lung has only lung tissue elastic forces to resist volume changes
- Surfactant in the alveoli fluid reduces surface tension, keeps alveoli from collapsing

#### **Surface Tension Elastic Forces**

The net effect on the lung is to simultaneously attempt to collapse alveoli by water tension

- Water-air interface creates tension on inner alveoli surface
- Water has strong attraction to itself resulting in a tight contraction of H2O molecules together
- Elastic force caused by water tension attempts to force air out of alveoli

#### Surfactant

- A synthesized fatty-acid product of Type II pneumocyte
- Surfactant lowers the surface tension of the alveoli fluid

#### **DPPC-Dipalmitoyl phosphatidyl choline**

- Hydrophobic & Hydrophilic opposing ends
- Alignment of intermolecular repulsive forces
- DPPC opposes water self-attractant elastic force to reduce alveolar surface tension
- Reduction of surface tension greater when film compressed closer as DPPC repel each other more

## **Multiple Functions of Surfactant**

#### • Lowers surface tension of alveoli & lung

- Increases compliance of lung
- Reduces work of breathing

#### • Promotes stability of alveoli

- 300 million tiny alveoli have tendency to collapse
- Surfactant reduces forces causing atelectasis
- Assists lung parenchyma 'interdependant' support

#### • Prevents transudation of fluid into alveoli

- Reduces surface hydrostatic pressure effects
- Prevents surface tension forces from drawing fluid into alveoli from capillary

#### **Total & Alveolar Ventilation**

#### <u>Total Ventilation or Minute</u> <u>Ventilation</u>

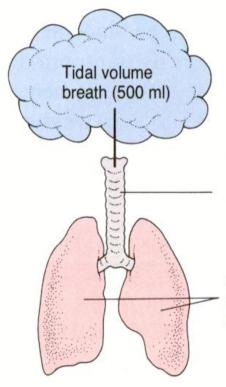
- Total volume of air conducted into lungs per minute
- Single breath = Tidal Volume (VT)
- V<sub>T</sub> varies with age, sex, body position & activity
- Normal  $V_T$  is 0.5 L
- Minute ventilation = VT x freq
- $6 \text{ L/min.} = 0.5 \text{ L} \times 12 \text{ breaths/min.}$

#### **Alveolar Ventilation**

- Volume of fresh air entering alveoli each minute (70% of total ventilation or minute ventilation)
- Alveolar ventilation is always less than total ventilation
- Anatomical dead space and its portion of tidal volume (30%) affect amount of gas exchanged in alveoli
- Alveolar O2 concentration steady state achieved when supply matches demand

# **Anatomic Dead Space**

- <u>Dead Space = ventilated but not</u> <u>perfused</u>
- The portion of tidal volume fresh air which does not go directly to the terminal respiratory units (30%)
- The conducting airways do not participate in O2 & CO2 exchange
- Dead space roughly 2 ml/kg ideal body weight or weight in pounds
- Anatomical differs from physiological dead space also described as wasted ventilation



Air in conducting airways; anatomical dead space air (150 ml)

Alveolar air; "useful" air participating in gas exchange (350 ml)

### **Wasted Ventilation**

- The concept of physiologic dead space (VPD) describes a <u>deviation from ideal ventilation relative to</u> <u>blood flow</u>
- Wasted ventilation includes anatomical dead space plus any portion of alveolar ventilation that does not exchange O2 or CO2 with pulmonary blood flow (alveolar dead space)
- Ventilation/blood flow (V/Q) mismatch where blood flow blocked ( clot or emboli)

### **Features of Laminar Flow**

- Laminar flow is parallel streams of flow
- Velocity in center of airway twice as fast than at edges of tube
- <u>Poiseuille Law</u> describes resistance to flow through a tube
  - Pressure increases proportional to flow rate & gas viscosity
  - Smaller airway radius & longer distances increase flow resistance

### **Airflow through Tubes**

- As air flows through a tube a pressure difference exists between the ends of tube
- This pressure difference depends on rate & pattern of air flow
- Airflow at low flow rates is laminar
- Turbulence occurs at higher flow rates or changes in air passageway (airway branches/diameter/velocity/direction changes)

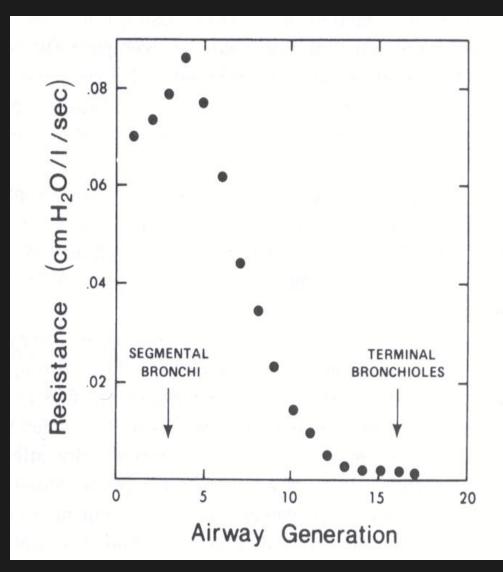
### **Turbulent Flow**

- Turbulence occurs at higher flow rates or air velocity
- Local eddies form at sides of airway & stream lines of flow become disorganized
- Pressure no longer proportional to flow
- Increases in density, velocity & airway resistance make turbulence more probable

# **Chief Site of Airway Resistance**

#### \*Major resistance is at the medium-sized bronchi

- Most of pressure drop occurs at **seventh** division
- Very small bronchioles have very little resistance
  - Less than 20% drop at airways less than 2mm
  - Paradox secondary to prodigious number of small airways in parallel
  - Air velocity becomes low, diffusion takes over



#### **Factors Determining Airway Resistance**

#### Lung Volume

- Linear relationship between lung volumes & conductance of airway resistance
- As lung volume is reduced airway resistance increases

#### Bronchial Smooth Muscle

- Contraction of airways increases resistance
- Bronchoconstriction caused by, acetylcholine, low Pco<sub>2,</sub> direct stimulation, histamine, environmental, cold
- Density & Viscosity Of Inspired Gas
  - Increased resistance to flow with elevated gas density
  - Changes in density rather than viscosity have more influence on resistance

# **Work of Breathing**

- Work is required to move the lung & chest
- Work represented as **pressure X volume (W=PxV**)
- Difficult to directly measure total work of breathing done by movement of lung & chest wall
- Oxygen consumption measurements can be used to determine work of breathing
  - O2 cost of quiet breathing is 5% of total resting oxygen consumption
  - Hyperventilation increases O2 cost to 30%
  - High O2 cost in obstructive lung disease limits exercise ability

#### A little pre-planning goes a long way...



"A mind once stretched by new Ideas never regains it original dimensions..."

# Why do we Intubate?

• Inability to protect and maintain patent airway.

• Failure of oxygenation or ventilation.

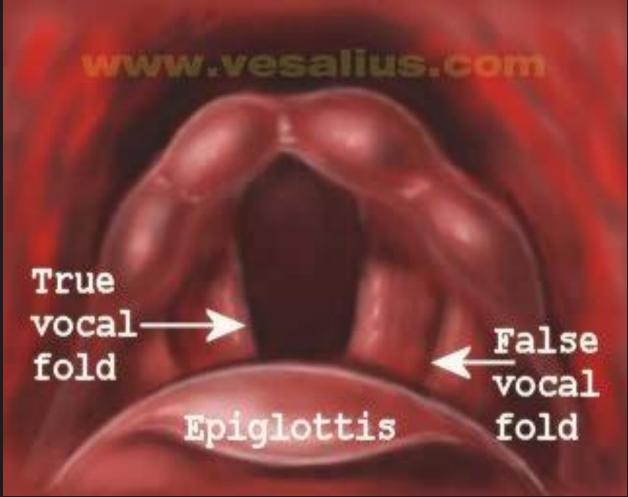
• Anticipated need based on clinical course

#### Ideal conditions for intubation

- Ideal Lighting, positioning, etc.
- Plenty of assistance
- Time to prepare, plan, discuss
- Option to Abort
- Empty Stomach
- Back up available.

## Ideal Pt. for intubation

- Intact, clear airway
- Wide open mouth
- Pre-Oxygenated
- Intact respiratory drive
- Normal dentition/good oral hygiene
- Clearly identifiable and intact Neck and Face
- Big open Nostrils
- Good Neck Mobility
- Greater than 90 KG, Less than 110 kg.



If only they looked this good...

# In Reality Our patients are:

- Immobilized
- Traumatized
- Compromised
- Prioritized
- Beer-n-Pizza-ized

### They Tend to look like This:



# And This:



## And This (after failed ETT attempt)







### OK, Here You Go!

• Mandibular Aplasia



### What does this mean to us?

- Well, many Anesthesiologist have the option to "Abort" induction, or to work through a problem with as much assistance as needed.
- In the REAL WORLD of EMS that is seldom the case for Paramedics.
- However many of the BASIC principles are valid in the clinical evaluation of Patients, and thus valuable in our education as medics.
- Knowing these principles will improve our decision making process and Patient Care;.

#### Before intubation

- Is there another means of getting our desired results BEFORE we attempt Direct Oral ETT? (Especially if we RSI)
- CPAP ?
- PPV with BVM or Demand Valve?
- Nasal ETT?
- Do we have all the help we need, all Airway equipment with us? (Suction?)

What are we going to do if we don't get the Tube?

• Plans "A", "B" and "C"

• Know this answer before you tube.

#### Plan "A": (ALTERNATE)

- Different Length of blade
- Different Type of Blade
- Different Position

# Plan "B": (BVM and BLIND INTUBATION Techniques)

• Can you Ventilate with a BVM? (Consider two NPA's and a OPA, gentile Ventilation)

• Multi-Luman Airway?

• LMA an Option?

What do we do when faced with a Can't Intubate Can't Ventilate situation?

• Plan "C": (CRIC) Needle, Surgical, P/C

# Do YOU feel ready to enact Plans A, B, C at a drop of a hat?

- Feel familiar with all those tools and techniques?
- As Paramedics we should, After all we will provide the only definitive care in these patients.

#### Thanks for Your Attention!!

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