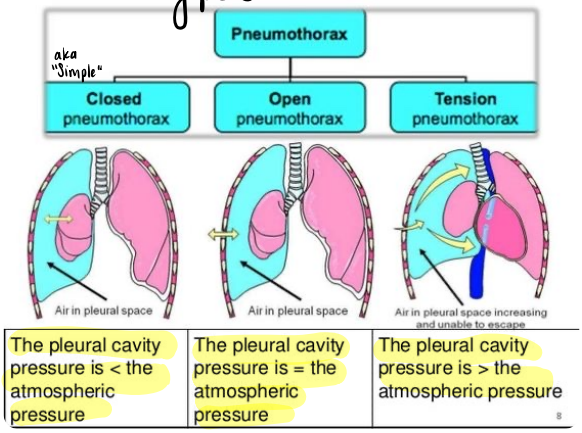
PNEUMOTHORAX

Three types:



Pneumothorax etiology is when air collects in the pleural space by occupying space and not allowing the lung to fully inflate.

Tension PTX: ↑ air pressure in the hemithorax
- Causes the trachea to deviate
- Compresses the heart (∴ cardiac output ↓)
- Compresses the other lung, reducing its ability to inflate.
- Compresses the great vessels (particularly the Vena Cava), thus reducing venous return.

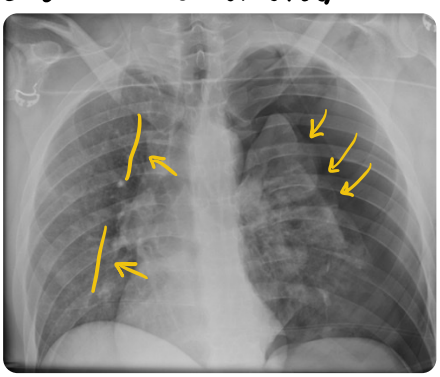
Open PTX: when the chest opening/wound diameter is ≥ 2/3 of the tracheal diameter, air will follow the pathway of least resistance → ∴ when the diaphragm contracts, air will not move through the trachea but through the wound
- this further ↑ the gas in the pleural space which will ↑ lung collapse.
  ↳ hypoxia, hypercarbia (↑CO_2)

* History/PE: sudden onset, trauma, resuscitative efforts; Marfan Syndrome; tachycardia, resp. distress, hypotension, neck vein distention; cyanosis (late manifestation), ↓/∅ breath sounds, ↓ tactile fremitus, hyperresonance on percussion, unilateral chest expansion.

* Diagnostics: chest xray, CT, ultrasound ("lung point sign")
- "lung point sign" = highly specific ultrasound sign that involves visualizing the point where the visceral pleura begins to seperate from the chest wall at the margin of a ptx.

* Treatment: decompress the chest,
- Needle or tube thoracostomy, give supplemental O_2, positive pressure ventilation (PPV) (for small traumatic ptx),
- ↳ chest tubes placed in 2nd ICS MCL

Tension Pneumothorax:



Placing tubes in a tension ptx will create an open ptx b/c pleural pressure will now = atmospheric. This is still not great, but better as less pressure on unilateral-sided organs.

ED Thoracostomy requirements: unresponsive hypotension (systolic BP < 70) despite vigorous resuscitation and...
- (for penetrating injuries): cardiac arrest with previously witnessed cardiac activity
- (for blunt injuries): rapid exsanguination (draining blood) from chest tube

- Occlusive dressing is good treatment for open ptx (air goes out, but not in)

Note: for spontaneous ptx, treatment guidelines state:

If stable & asymptomatic → small → no intervention
                         → large → intervention

If unstable → intervention

FOREIGN BODY ASPIRATION

* Symptoms / Signs: abrupt onset of stridor, cough, wheezing
  - Acute presentation: witnessed choking event
  - Chronic presentation: >10 days following aspiration; hx of prolonged cough, focal wheezing, etc.

* Diagnostics: Xray or CT (CT has higher sensitivity)
  - X-Ray Findings -
  → Due to the check valve mechanism, where air enters the bronchus around the foreign body but cannot exit, the affected lung will usually appear overinflated and hyperlucent, with concomitant rib flaring and a depressed ipsilateral hemidiaphragm.
  → Patient should be radiographed on expiration to exaggerate lung differences; patient should be in the decubitus position

WARNING: button batteries or multiple magnets = liquefaction necrosis!

→ Rule of thumb: a coin in the trachea will be better seen in the lateral view, while a coin in the esophagus is better seen in the AP view

* Treatment: Bronchoscopy (but it has a high adverse event rate in kids!)

If highly suggestive history → Admit and bronchoscopy (regardless of exam or CXR)

Equivocal history + abnormal exam or CXR → Admit and bronchoscopy

Equivocal history + normal exam / normal CXR → may forego bronchoscopy and opt for observation

ARTERIAL BLOOD GASSES

- Volatile acid: easily removed from the body (ex: H_2CO_3)
- Fixed acid: not easily removed from the body (ex: lactate, ketoacids, sulfate, phosphate)

Principles of Acid-Base regulation:

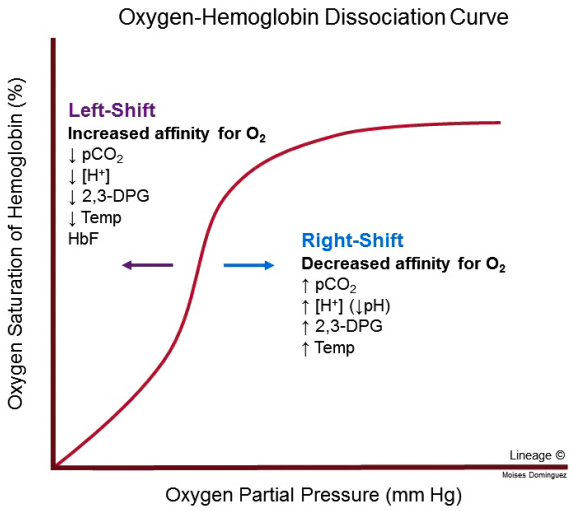
$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

$\uparrow CO_2 \leftrightarrow \uparrow HCO_3^-$

$\downarrow CO_2 \leftrightarrow \downarrow HCO_3^-$

Increase one, you'll increase the other and vice versa

Carbonic Anydrase is an enzyme in RBCs that...
- At all metabolically active tissues, converts CO_2 into H_2CO_3.
- At all pulmonary capillaries, converts H_2CO_3 into CO_2.



RESPIRATORY PHYSIO:

In normal CO_2 excretion (ventilation), ↑CO_2 is detected by the medullary respiratory center in the medulla oblongata. This leads to ↑ ventilation, a response to a change in pH or pCO_2.
- If pH falls, ↑ CO_2 excretion

RENAL PHYSIO:

Proximal tubular absorption of HCO_3^- is ↑ by: ↑pCO_2, ↓ potassium, ECF volume contraction.
HCO_3^- is generated at the distal nephron and absorbed into blood

RECAP:

LUNGS:
- Eliminate CO_2 (hyperventilation) to ↑pH
- Retain CO_2 (hypoventilation) to ↓ pH

KIDNEYS:
- Excretes H^+ and retains HCO_3^- to ↑pH
- Retains H^+ and excretes HCO_3^- to ↓pH

Acid-Base Parameters:
- pH: 7.35 - 7.45
- pCO_2: 35-45 mm Hg
- HCO_3^-: 22-26 meq/L

- **Simple Disorder**: an acid-base disturbance and associated compensatory changes (usually isolated)
- **Mixed Disorder**: more than one primary acid-base disturbance occuring at the same time

Arterial Blood Gas Values

	Acidosis	Normal	Alkalosis
pH	<7.35	7.40	>7.45
pCO_2	>45	40	<35
HCO_3^-	<22	24	>26

Note: If both pCO_2 and HCO_3^- are on the same side as pH, then it is a mixed disorder

Respiratory Acidosis:

- Acute (normal HCO_3^-)
  - CNS depression - drugs, CNS event
  - Neuromuscular disorders
  - Acute airway obstruction
  - Severe pneumonia or pulmonary edema
- Chronic (metabolic compensation)
  - Chronic lung disease
  - Chronic neuromuscular disorders
  - Chronic respiratory center depression - central hypoventilation

Anion Gap = $Na^+ - (Cl^- + HCO^{3-})$

Decreased gap = hypoalbuminemia or increase in unmeasured cation
Increased gap = retention of 1 or more unmeasured anions

Metabolic Acidosis with Respiratory Compensation

- Anion Gap Acidosis (Anion Gap >20)
  - Ketoacidosis
  - Renal failure
  - Lactic acidosis
  - Rhabdomyolysis
  - Toxins
- Non-anion Gap Acidosis
  - GI bicarb loss (ie. Diarrhea)
  - Renal bicarb loss
  - HCl administration
  - Posthypocapnia

Acronym for reasons for Anion Gap Metabolic Acidosis

- M – Methanol/metformin
- U – Uremia
- D – Diabetic ketoacidosis
- P – Paraldehyde/phenformin
- I – Iron/isoniazid
- L – Lactate
- E – Ethylene glycol
- S – Salicylates

Metabolic Alkalosis

- Low Urine Cl^- Level
  - Vomiting
  - Diuretic use in past
  - Posthypercapnia
- Normal or High Urine Cl^- Level
  - Excessive mineralocorticoid
  - Diuretic Use currently

Three Step Method for Recognizing Acid-Base Disorders

1. Look at pH
   Determine if acid or alkaline
   Look at pCO_2 (respiratory) and HCO_3^- (metabolic)
   Value on same side as pH is primary abnormality
2. Calculate the anion gap
   If anion gap $\geq$ 20, there is an anion gap metabolic acidosis regardless of pH or serum HCO_3^- concentration
3. Calculate the excess anion gap
   Excess = total AG – normal AG of 12 + measured HCO_3^-
   1. If the sum is >30, underlying metabolic alkalosis
   2. If the sum is <23, underlying nonanion gap metabolic acidosis