# *NUR5002 NOTES*

# CLINICAL SKILLS

## PATIENT POSITIONING STRATEGIES

- Place pillows behind the shoulder, head, weaker arm and/or hip of affected side
- Place feet in neutral position and ensure correct alignment of pts knees and feet (feet directly below/in line with knees)
- Minimise time pt spends sitting position in bed → better support for sitting position in a chair
- Do not pull weaker arm → can cause subluxation of shoulder and pain
- Elevate weaker arm when pt is in bed, chair, or wheelchair and place feet in neutral position
- Position hand and fingers so they are barely flexed (palm faced upwards) as it is most functional position
- Hand roll is not used if upper extremity is spastic b/c it stimulates grasp reflex → in this case use dorsal splint to allow palm to be free of pressure

## INTERCOSTAL CATHETERS (ICC) AND PERITONEAL DRAINS

Drains inserted through an incision to drain fluid or air from cavities it should not be in

### INDICATIONS

| | | |
|---|---|---|
| **Ascites** | **Peritoneal Fluid** | **Pleural Effusion** |
| **Empyema** | **Pneumothorax** | |

### NURSING CONSIDERATIONS

1) Assess for infection risks if the drain has been in for awhile
2) Assess the liquid being drained: colour, consistency, pus presence, blood presence, volume of drainage
3) Auscultate the chest, respiratory obs
4) Cardiac obs if the drain is for effusions that can push on the heart
5) On insertion increase obs to 15 minutely for the first hour, then 1 hourly for the next 4 hours
6) Assess the drain site to ensure there is no leaking, and the dressing is dry and intact
7) Do the dressings as per the dressing plan
8) Assess for pressure injuries if the tubing is in a position where it puts pressure on the skin

9) Pain management and advocation
10) Encourage patient positioning changes to help with drainage
11) Take a specimen sample
12) Note the insertion site: 2rd or 3rd intercostal for pneumothorax, 4th or 5th for pleural effusions

## UNDERWATER SEAL DRAINAGE DEVICES (UWSD)

Used to help drain fluid from the chest or to help restore negative pressure in the lungs. Can also be inserted into the pericardial cavity to drain fluid around the heart.

### INDICATIONS

| | | |
|---|---|---|
| **Pneumothorax** | **Pleural Effusion** | **Haemothorax** |
| **Post-Operative Cardiac Surgery** | **Empyema** | **Pyothorax** |

### NURSING CONSIDERATIONS

1) Assess for infection risks if the drain has been in for awhile
2) Assess the liquid being drained: colour, consistency, pus presence, blood presence, volume of drainage
3) Auscultate the chest, respiratory obs, and ensure pt goes to get a chest x-ray to ensure it is correctly placed
4) On insertion increase obs to 15 minutely for the first hour, then 1 hourly for the next 4 hours
5) Assess the drain site to ensure there is no leaking and the dressing is dry and intact
6) Do the dressings as per the dressing plan
7) Assess for pressure injuries if the tubing is in a position where it puts pressure on the skin
8) Pain management and advocation
9) Ensure suctioning is on and working appropriately
10) Assess for bubbling that would indicate and air leak somewhere
11) Ensure there is a swing in the water chamber with inhale and exhales (with respirations), however swing/tidalling will stop when the lung has successfully re-expanded
12) Encourage patient positioning changes to help with drainage
13) Take a specimen sample
14) Note the insertion site: 2rd or 3rd intercostal for pneumothorax, 4th or 5th for pleural effusions
15) Ensure there is emergency equipment by the bedside including clamps in the case of malfunctions so you can clamp the tube to prevent air from coming back into the pleural space
16) Ensure UWSD is kept lower than the patients chest and is upright

17) IF there are clots or fibrin in the tubing, rolling the tube between the fingers to break up the clot. Do not pinch the tube though as it causes high negative pressure
18) Assist with coughing and deep breathing
19) Reposition the patient every 2 hours
20) If test tube is dislodged, immediately apply an occlusive sterile dressing and escalate

## TRACHEOSTOMY CARE

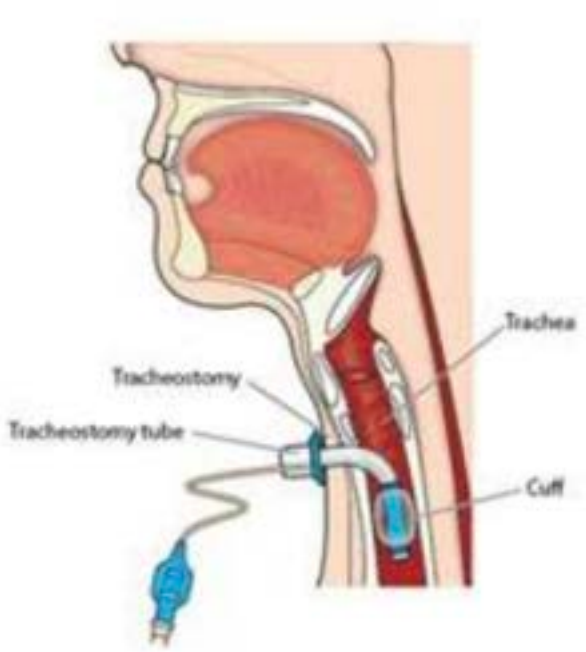
a surgical opening in the person's trachea into which an indwelling tube is placed to overcome upper airway obstruction, facilitate mechanical ventilatory support or enable removal of trachea-bronchial secretions. Can be permanent or temporary

### INDICATIONS

| **Overcome Airway Obstruction** | **Facilitate Mechanical Ventilation Support** | **Enable Removal of Trachea-Bronchial Secretions** |
|---|---|---|

### NURSING CARE

1) Suction secretions
2) O2 if required needs to go through a tracheostomy shield and must be humidified and heated to body temperature
3) Stoma care - assess the skin around the insertion site for: skin integrity, colour, dressing and tapes D+I, swelling
4) Emergency equipment is required for all tracheostomy patients to be at their bedside: spare tracheostomy tubes, dilator, air-viva bad with straight attachment, 10 ml syringe for emergency cuff deflation, Functioning O2, suction with a Y-Suction Catheters
5) Monitor for obstructions
6) Monitor for haemorrhage
7) Monitor for dislodgement or removal
8) Monitor for loss of voice, speech and language delays and decay
9) Monitor impaired swallow and risk of aspiration
10) Monitor for infections since they are at risk for increased infections and illnesses




## VENIPUNCTURE

Needle Size: 21-23

Insert 15-30% angle

Tourniquet cannot be on for more than 1 minute before venepuncture

Always remember to unlock the tourniquet before taking the needle out so you don't spurt blood everywhere

Fill up the pathology tubes in order of how they are represented on pathology charts

When collecting cultures fill up the aerobic bottle first then the anaerobic. Blue before purple, you can think of it in terms of blood pressure - BP

## INJECTIONS

| | **Subcutaneous** | **Intramuscular** | **Intradermal** |
|---|---|---|---|
| Needle Size | 25-27 Gauge | 22-25 Gauge | 26-27 Gauge |
| Injection Site | Abdomen<br>Upper gluteal region<br>Anterior fatty aspect of the arm<br>Thigh | Ventrogluteal<br>Deltoid<br>Vastus Lateralis and Rectus Femoris | Inner aspect of the arm |

| | | | |
|---|---|---|---|
| Injection Angle | 45-90 Degrees (depends on body fat) | 90 Degrees | 5-15 Degrees |
| Considerations | Pinch up if lower BMI<br>Spread skin if higher BMI | Smaller muscles can handle less volume, use larger muscle groups for larger doses | Can cause bubbling under the skin |
| Injection Location | Subcutaneous fat layer | Muscle layer | Between epidermis and dermis |
| Complications | Leakage, infection, bruising, bleeding, going too deep or not deep enough | Infection, bruising, bleeding, pain, hitting a nerve, | Skin bursting, incorrect depth for injection, bruising, bleeding |

## CENTRAL VENOUS ACCESS DEVICES

| Main types | Characteristics | Duration of Use | Advantages | Limitations | Schematic representation |
|---|---|---|---|---|---|
| **Tunneled central venous catheter** (Hickman®, Groshong®, Broviac®). | -Distal end open to the outside with subcutaneous (tunneled) way.<br>-A Dacron cuff attached to the line induces a local fibrotic reaction and anchors the cuff to the tissues. | Months to years | -Longer duration.<br>-Lower risk of infection and thrombosis. | -Access for insertion needs medical radiology, surgery or anesthesia equipment.<br>-Higher cost.<br>-Limited flow. |  |
| **Central venous access device with subcutaneous reservoir** *(Port or port-a-cath type).* | -Special tunneled venous line connected to a subcutaneous reservoir.<br>- A non-coring needle is needed to access the thick port membrane | Months to years | -Longer duration.<br>-Lower risk of infection and thrombosis.<br>-Comfortable and cosmetic (no visible line). | -Access for insertion needs medical radiology, surgery or anesthesia equipment.<br>-Need for special training/equipment for nurses to access<br>-Higher cost.<br>-Limited flows.<br>-6 weekly flushing, heparin locking<br>-Access to device requires skin puncture each time. |  |
| **Peripheral insertion central catheter** (PICC type). | -Line is inserted by a peripheral vein (basilic, cephalic, etc.) passing through the arm.<br>Correct tip location: superior vena cava/right atrium junction. | Months | - Easier to insert and remove by trained nurses.<br>-Lower cost. | -Increased risk of infection.<br>-Increased risk of thrombosis.<br>-Requires weekly care by trained nurses. |  |

## PATIENT CONTROLLED ANALGESIA (PCA)

- Often used postoperatively and contains schedule 8 opioids → morphine, fentanyl, tramadol

**Setting Up the PCA**

| | |
|---|---|
| Loading Dose | Loading dose ensures therapeutic action of med is researched immediately after set-up<br>- Not always prescribed |
| Bolus Dose | The amount of medication that the PCA pump delivers when the demand button is pressed |
| Lockout Interval | Time from end of delivery of one dose until the PCA will response to another dose |
| Continuous Infusion Rate | A background continuous infusion rate that some PCAs will have<br>- Not always prescribed |
| Lockout | Time between demands that pt cannot exceed and is documented in minute increments (e.g. 5 min) |
| Hourly limit | Max dose the PCA will deliver in any one hour |
| Attempts/Demands/Bolus Dose | Number of times the pt has pressed the button. NOT the amount of doses delivered<br>- Indicates if education or a change of the dose is required |

## DIALYSIS

**Indications**

1) eGFR is 12 or less and/or symptomatic
2) Increased levels of urea and creatinine
3) Oliguria
4) Increased K+ levels

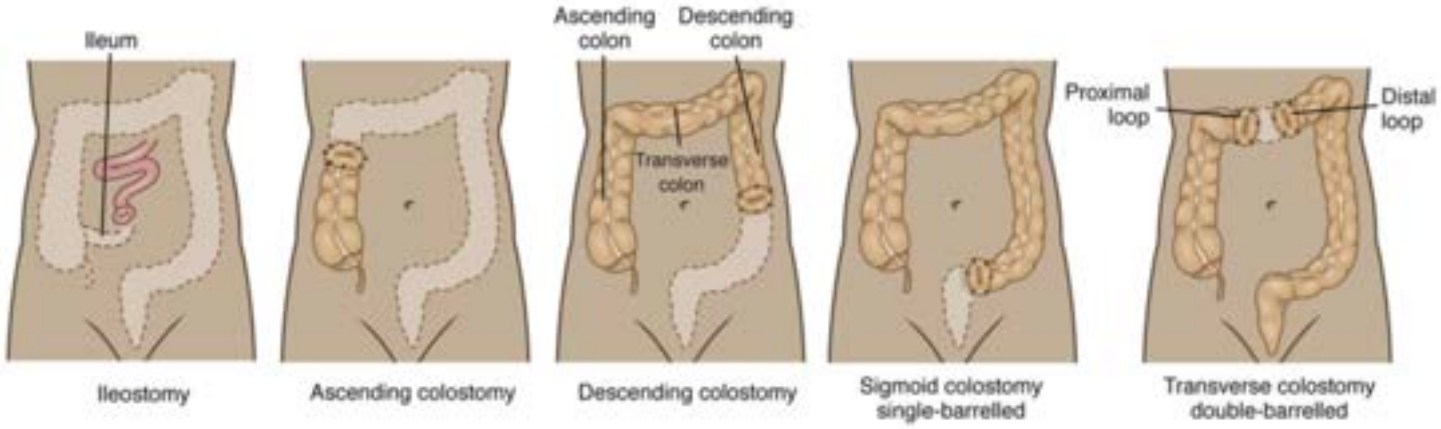
**Nursing Considerations**

1) Ensure you are not giving meds that need to be withheld for patients going for dialysis
2) Monitor their blood tests
3) Monitor how much volume has been taken off of your pt in dialysis and monitor their BP since it can be in MET call hypotensive criteria afterwards
    a. Also can get hypovolaemic shock
4) Regular weights/daily dry weights
5) Ensure nutrition is adequate

**Temporary Shunt VS AV Fistula**

| Care/Nursing Management | |
|---|---|
| **Temporary Shunt** | **AV Fistula** |
| More prone to infection, daily care required<br>Done through a CVAD<br>Dressing D+I and dressing needs a weekly change<br>Check notes for fluid taken off during dialysis, expect BP to go down (decreased vascular resistance)<br>Certain meds need to be restricted on dialysis days (e.g. beta blockers) | Performed for those needing long term dialysis<br>Avoid fistula arm for BPs, injections, phlebotomy, cannulas, CVADs<br>Auscultate fistula to see if its producing the bruit it should<br>Avoid heavy lifting<br>Check notes for fluid taken off during dialysis, expect BP to go down (decreased vascular resistance)<br>Certain meds need to be restricted on dialysis days (e.g. beta blockers)<br>Monitor for NGT need |

## STOMA CARE




- Preoperative preparation and education is important for psychological and practical preparation
- Colostomy → output is a more formed stool so a bag that is closed is used and taken off and disposed, and entire bag replaced
- Ileostomy → bag that can be drained is used, as the output is more liquid in consistency due to the section of the GI tract the stoma is made.
- Use a remover spray to take the bag off easily and without discomfort
- There are deodoriser drops you put inside the bag when you put a new bag on
- Clean the skin surrounding the stoma when replacing
    - Clean from the middle of the stoma out in a circular motion

- Note the surrounding skin condition and any break down
- Note the colour of the stoma itself – should be pinkish/mild red

- cut the stoma bag to the correct size and affix to the skin

## NASOGASTRIC TUBE AND ENTERAL FEEDINGS

Nasogastric Tube Functions:

1) a way for air and fluid to escape the stomach
2) for enteral feedings to enter

Indications for NGT insertion:

1) inadequate nutritional intake
2) swallowing difficulties
3) Decompression (wide bore NGT)

Types of Enteral Feeding Lines:

1) Nasogastric – end of tube lies in the stomach
2) Nasoduodenal – end of tube lies in the duodenum of the small intestine
3) Nasojejunal – end of tube lies in the jejunum of the small intestine

Types of Parenteral Nutrition:

1) Total parenteral nutrition (TPN) – used where the pt has central venous access, e.g. PICC line
2) Peripheral parenteral nutrition (PPN) – used if there is only peripheral IV access, e.g. cubital fossa

### NGT Insertion

Equipment:

- NGT Tube itself – check the orders for the size required and adjust as needed with the patient if they are not tolerating the size
- Drainage bag and spigot – PT can be on free drainage or on hourly aspirates
- Syringe – as you may be aspirating gastric contents
- Litmus paper
- NGT fixation device
- Lubricant

Process:

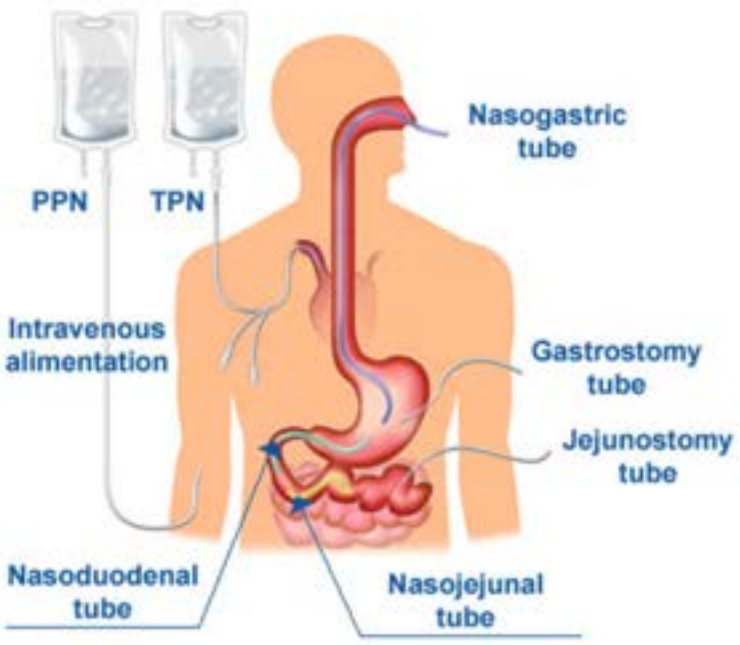
1) Education to patient – it is an uncomfortable procedure
2) Gather materials
3) Assess which nare is better
4) Measure tube – from nose, around back of ear, and then to the xiphoid process, then add another 10 cm. that end number is what you will insert until
5) Lubricate

6) Start insertion – when the NGT tube starts hitting the throat region ask the pt to put their head forwards and swallow
7) If there is a lot of resistance stop, if you're coming across a little you can try twisting around but if it's not helping stop and try the other nare
8) Once inserted to correct length, get the catheter tipped syringe and aspirate gastric contents
9) Test the contents on the litmus paper, you want the pH to be between 1-5
10) Secure the Nasogastric tube with nasofix
11) Do not use until the PT has gone for a chest x-ray to make sure it's in the right spot
12) Document how many cm it is at, if its on free drainage, or spigotted

**NGT Nursing care and considerations**

- Note the length of the NGT at the start of your shift
- Ensure the fixation device is still working
- Empty drainage bags if the NGT is for decompression
    - Note colour, consistency, and amount drained
- Ensure the correct dietitian ordered feeding supplement is ordered, since they're not all the same

| **Enteral vs Parenteral Feeds** | |
|---|---|
| **Parenteral** | **Enteral** |
| Feeding done through intravenous access | Tube placed directly into the GIT |
| Intravenous feeds bypassing the usual process of eating and digestion | Can give crushed meds through |




# FLUID & ELECTROLYTES

## FUNCTION OF ELECTROLYTES IN THE BODY

| Electrolyte | Main Function | Normal Range | Related Body System |
|---|---|---|---|
| **Sodium** | Helps maintain blood volume & blood pressure. Maintain fluid volume and osmolality. | 135-145 mEq/L | Sodium imbalances can lead to neurological changes |
| **Potassium** | Helps maintain muscles to contract (e.g. heart muscle). Maintain osmolality within the cell. Works with sodium. | 3.5-5 mEq/L | Potassium imbalances can cause cardiac dysrhythmias that can be life threatening |
| **Calcium** | Helps with heart function, blood clotting & bone formation. Influences | 9-11 mg/dL | Calcium is important for bone formation and imbalances can lead to increased risk |

| | | | |
|---|---|---|---|
| | the excitability of nerve & muscle cells. | | for pathological fractures |
| **Magnesium** | Helps muscles and nerves stay healthy & regulates energy levels. Assists both skeletal and cardiac muscles. Also assists in reactions during protein and carbohydrate metabolism | 1.5-2.5 mg/dL | Magnesium can act like a sedative. Imbalances can affect muscle health and activity |
| **Phosphorus** | Helps create/maintain teeth and bones, & helps repair cells and body tissue. An essential component in ATP cell energy and cellular metabolism & also in nucleotides | 2.5-4.5 mg/dL | Phosphorus helps the body to use vitamins to maintain tooth and bone health |
| **Chloride** | Helps maintain acid-base balance, helps control fluid levels in the cells | 95-105 mEq/L | Think cellular. Chloride helps to balance the acids and bases in the body to prevent disturbance, and maintain healthy fluid volume in the cells |

## RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM

The Renin-Angiotensin Aldosterone System responds to renal perfusion and makes adjustments that affect blood pressure by affecting fluid volume

Step 1: Juxtaglomerular Cells respond to blood pressure/volume filtering through the glomerulus to stimulate or inhibit RENIN release

- They respond through mechanoreceptors which can feel low blood pressure
- Sympathetic nerve fibres which measure immediate blood pressure from the heart which tell the juxtaglomerular cells when BP goes down in the aortic arch and carotid sinus and also stimulates the release of renin from the juxtaglomerular cells when BP is low through sympathetic stimulation

- Chemoreceptors in the juxtaglomerular cells also respond to glomerular filtration and respond to the amount of sodium and /or chloride ion being filtered. As more filtration occurs when BP raises, and filtration decreases when BP decreases. This change also triggers the release or withholding or renin (note NSAIDS and impair this response system)

Step 2: After Renin is released it then seeks out Angiotensinogen in the blood. Renin then cleaves off a portion of angiotensinogen to create Angiotensin I

Step 3: Angiotensin I interacts with angiotensin converting enzyme (largely in the lungs) to create Angiotensin II

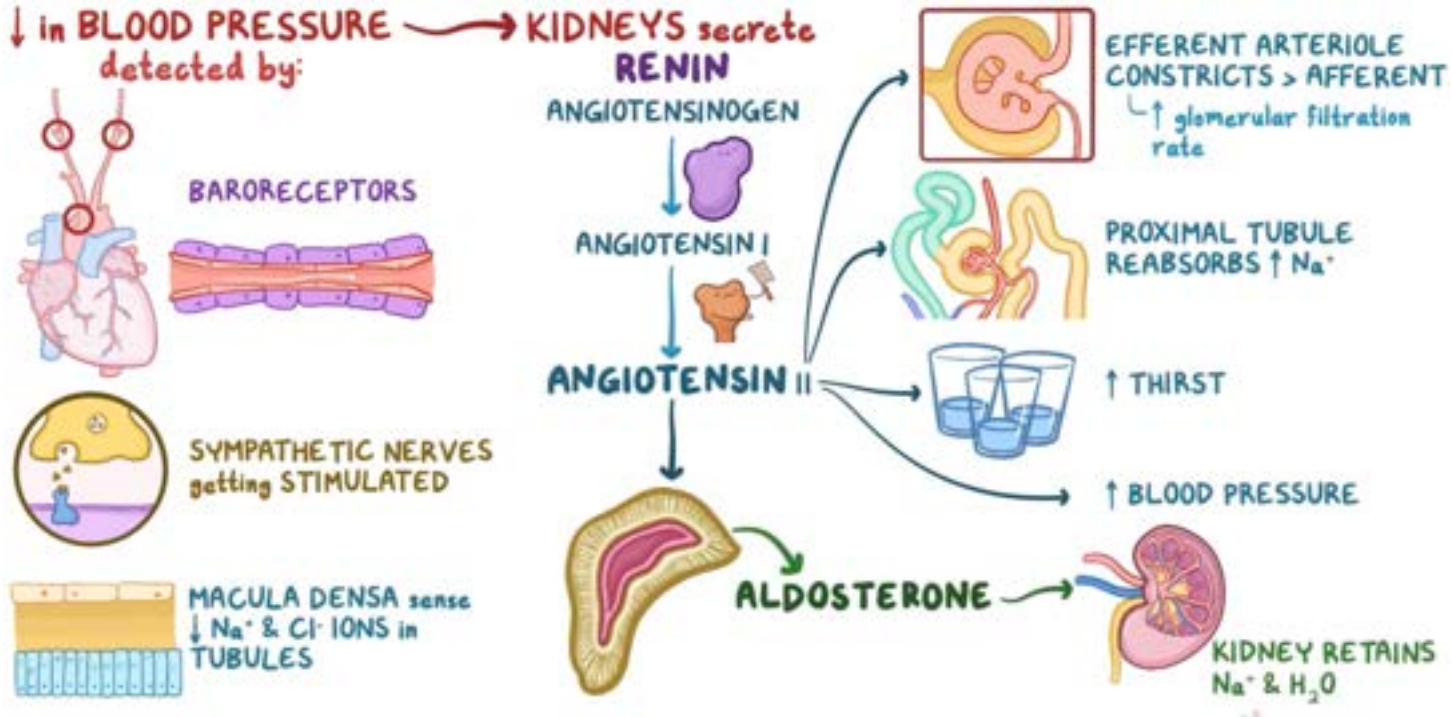
Step 4: Angiotensin II is a prominent vasoconstrictor that leads to blood vessel constriction. This increased peripheral vascular resistance, in turn increasing Blood pressure

Step 5: Increased levels of Angiotensin II cause constriction of both the afferent and efferent arterioles to decrease glomerular filtration rate, causes more sodium ion reabsorption and water retention through osmosis

Step 6: Angiotensin II acts on the hypothalamus to increase thirst and increase antidiuretic hormone. This causes you to ingest more water and causes a retention of fluids in the body to increase circulating blood volume. This also increases blood pressure.

Step 7: Angiotensin II also causes the adrenal glands to produce more aldosterone.

Step 8: aldosterone helps regulate sodium balance in the body by acting on the cells that line distal tubule and the collecting duct. It forms an aldosterone complex there and causes more sodium to be pushed into the blood and secretion of potassium. Water therefore follows into the blood and is retained, but also can create hypokalaemia. This process can take hours to days though, so this is a late result of the RAAS system.



# ACID BASE BUFFER

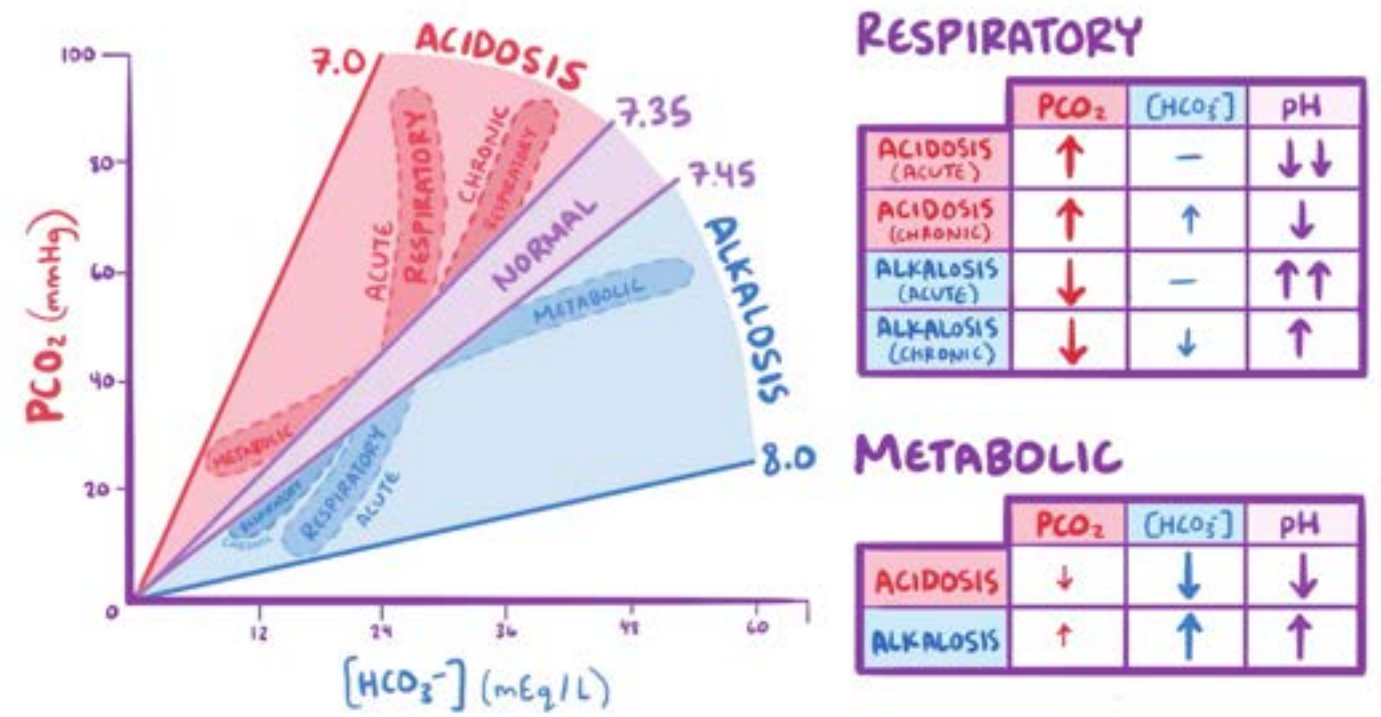



**Figure 8.1** An acid-base map shows the relationship between pH, bicarbonate concentration, and partial pressure of carbon dioxide in respiratory and metabolic acidosis or alkalosis, and how these values are adjusted when there is renal or respiratory compensation. The accompanying tables depict the changes in $PCO_2$, $[HCO_3^-]$, and pH associated with respiratory/metabolic acidosis/alkalosis.

Imbalances in pH are either due to Respiratory causes or Metabolic disorders (e.g. renal disease). There is an extreme pH disturbance if there is both an ability to compensate through the kidneys and the respiratory system.

The Mechanisms to maintain homeostatic pH are the Lungs and the Kidneys:

- By controlling the rate and depth of breathing, the chemoreceptors in the brain signal for changes in respiration to either breath out more CO2 to reduce acidity and make the body/blood more basic or slow the rate and depth to retain more CO2
- The kidneys control HCO3 (bicarbonate) to make the environment more or less basic by retaining or excreting extra bicarbonate. The kidneys also control the amount of hydrogen ions (acidic) retained or excreted through the ammonia phosphate buffer system to also change pH. They also can change acidity through phosphate buffer salts. The kidneys response is normally slower than the ability to accommodate through respiratory compensation

# RESPIRATORY

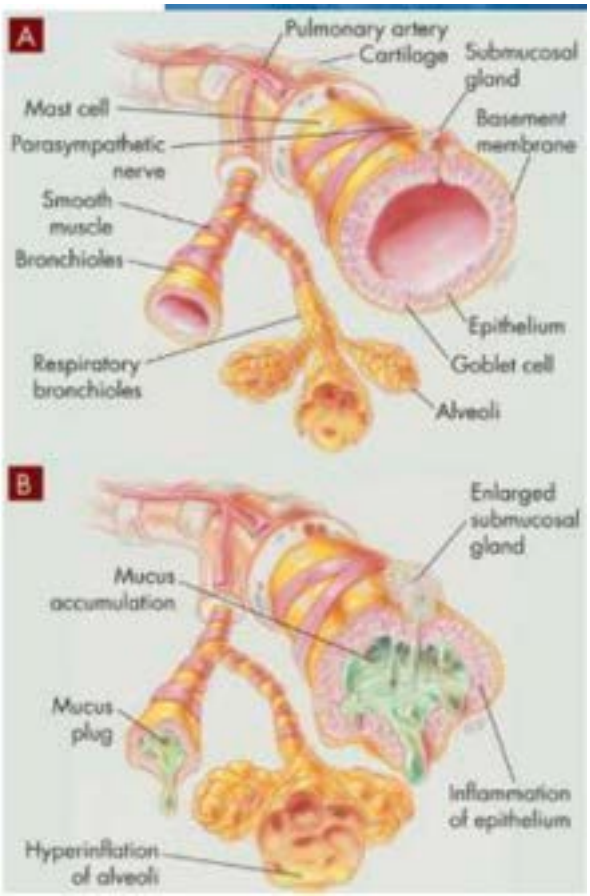
## DEFINITIONS

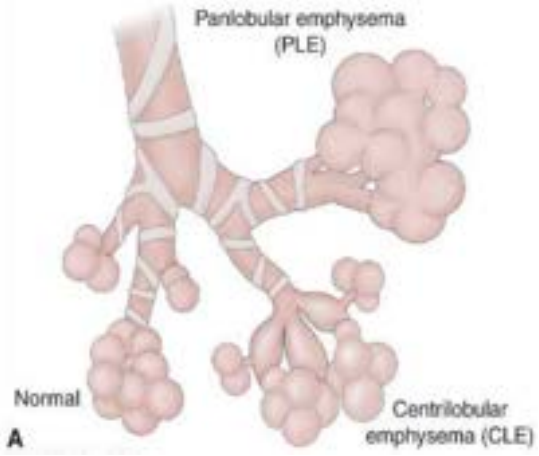
Gas Trapping = Co2 trapped in the alveoli and O2 cannot get into alveoli. Results in clinical manifestations of COPD

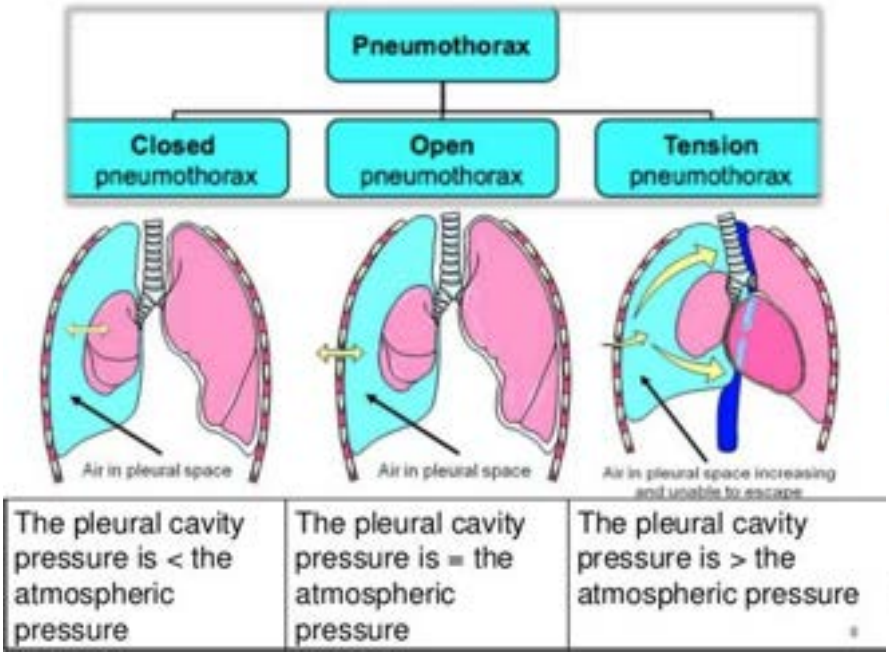
Cor Pulmonale = a condition where the right ventricle of the heart becomes enlarged and weakened due to high blood pressure in the pulmonary arteries, results in strain on the right side of the heart as it tried to pump blood through the affected lungs

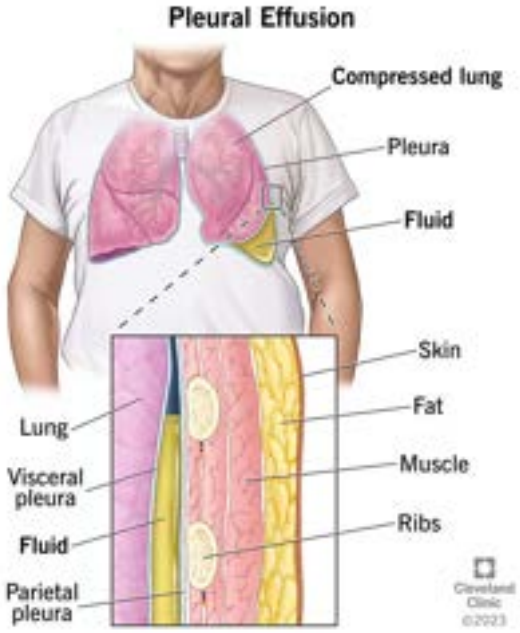
Hydrostatic Pressure = force that blood exerts on walls

## ANATOMY






# COMMON CONDITIONS

## ASTHMA

A heterogeneous disease that is characterised by chronic inflammation causing: inflammation, bronchial hyperresponsiveness and airway narrowing (bronchoconstriction)

Status Asthmaticus = acute, severe, and prolonged asthma attack lasting for 24 hours or more w/o improvement

### PATHOPHYSIOLOGY

Exposure to a trigger (pollution, cold air, exercise, resp tract infection, smoking) → release of inflammatory mediators (histamine, prostaglandins, leukotrienes, nitric oxide, interleukins) → bronchial smooth muscle contraction (bronchoconstriction), airway oedema, airway hyperresponsiveness, excess mucus in airways, airway remodelling → obstruction of the airways, v/q mismatch, air trapping, respiratory acidosis, hypoxemia

### SIGNS AND SYMPTOMS

| | | |
|---|---|---|
| **Wheeze (usually on expiration) can be audible or only on auscultation** | **Breathlessness** | **Chest tightness** |
| **Coughing** | **Decreased ability to speak** | **Increased WOB** |
| **Tracheal Tug** | **Cyanosis** | **Diaphoresis** |

| | | |
|---|---|---|
| **Altered Level of Consciousness** | **Silent Chest on Auscultation (very severe asthma)** | **Hypotension** |
| **Bradycardia** | | |

RISK FACTORS AND CAUSES

| | | |
|---|---|---|
| **Smoking** | **Pollutants** | **Genetic Predisposition** |
| **Allergens** | **Irritants** | **Urban areas** |
| **Viral Respiratory Infections** | **Obesity** | |

DIAGNOSIS

| | | |
|---|---|---|
| **Spirometry** | **FeNO test** | **Bronchial Provocation Test** |
| **Physical Exam** | Medical History | |

NURSING CONSIDERATIONS & INTERVENTIONS

1) Acute asthma management
- (i) Correct significant hypoxaemia
    - Hudson Mask (6 litres or more)
    - For more severe exacerbations: non-rebreather, venturi
    - Reposition to high fowlers
- (ii) Reverse airflow obstruction
    - Pharmacological management
        - Reliever medications
        - Preventer medications
- (iii) Plan to prevent further events
    - Education
    - Management of triggers
    - Proper inhaler technique use

## PNEUMONIA

Infection in the lungs caused by a virus, bacteria, or fungus. Causes inflammation and infection of the pulmonary parenchyma. Classified by the type of infection, where the lung is affected and where it is acquired.

Type of infection = bacteria, virus, fungus, etc.

Where in the lung = lobar pneumonia, interstitial pneumonia, or bronchopneumonia

Classification based on location acquired = community acquired pneumonia, or hospital acquired pneumonia

## PATHOPHYSIOLOGY

Pathogen enters the respiratory tract → pathogen colonises the bronchioles or alveoli and causes irritation → immune response is triggered and T-cell activation occurs → inflammation spreads and permeability is increased to facilitate movement of leukocytes to the infection → chemotaxis occurs in response to proinflammatory mediators → recruitment of neutrophils to infection site occurs → mucus begins to build up due to epithelial cells trapping debris and pathogens → increased capillary permeability causes oedema in the region → Consolidation occurs (build-up of fluid, fibrinous exudate, and cell debris) as well as mucus build up at the site of gas exchange→ impairs gas exchange and obstructs the air ways → ventilation-perfusion mismatch and hypoxaemia ensues as well as acidosis.

Note aspiration pneumonia is also a type of pneumonia which occurs due to aspirated food or fluids, or also from gastric contents. → occurs when the natural defences against aspiration have been compromised, e.g. due to stroke → right lung is more often affected due to the structure and angle of the right main bronchus.

## SIGNS AND SYMPTOMS

| | | |
|---|---|---|
| **Chest pain with breathing and coughing** | **Confusion or GCS changes** (esp adults >65) | **Productive or nonproductive Cough** |
| **Fatigue** | **Fever** | **Diaphoresis** |
| **Chills** | **Nausea, Vomiting, diarrhoea,** | **SOB** |
| **Dyspnoea** | **Tachycardia** | **Tachypnoea** |
| **Crackles on auscultation** | **Cyanosis** | **Low O2 Sats** |
| **Low blood pressure (Hypotension)** | | |

## RISK FACTORS AND CAUSES

| | | |
|---|---|---|
| **Stroke Hx** | **Dysphagia** | **Being hospitalised** |
| **Smoking** | **Chronic Disease** | **Immune system suppression** |

## DIAGNOSIS

| | | |
|---|---|---|
| **Chest X-Ray** | **Sputum Testing** | **Blood Cultures** |

**CT Scan**

## NURSING CONSIDERATIONS & INTERVENTIONS

1) Check White Blood Cell Counts
- Check the bloods to ensure the infection is resolving
2) General nursing care
- Respiratory assessment
- Obs
3) Oxygen management if indicated
4) Initiate/Encourage Deep Breathing and Coughing
5) Get a sputum sample
6) Administer antibiotics in a timely manner since they are time sensitive
7) Encourage regular vaccinations

# CHRONIC OBSTRUCTIVE PULMONARY DISORDER (COPD) - CHRONIC BRONCHITIS AND EMPHYSEMA

Progressive chronic disease characterised by irreversible obstruction of the airway that may cause chronic bronchitis, emphysema, or a combination of both. Forced expiratory volume and Forced vital capacity are reduced BUT total lung capacity is often higher due to air trapping

Chronic Bronchitis: airway inflammation, increased mucus, remodelling

Emphysema: destruction of alveolar tissue, alveolar hyperinflation, and decrease in lung elasticity

## PATHOPHYSIOLOGY

Regardless of the aetiology the end results are: airway obstruction, air trapping, dyspnoea, frequent infections, abnormal v/q ratio, hypoxaemia, hypoventilation, cor pulmonale

Chronic Bronchitis (CB)

- Irritation of the airways (e.g. cigarette smoke) → inflammation and hypersecretion of mucous → increase in mucus gland (hypertrophy and hyperplasia of goblet cells and bronchial mucinous glands) size and thickness of secretions occur → inability to fully clear out the air ways → ciliary function is reduced, bronchial walls thicken, bronchial airways narrow → mucus plugs the airways → alveoli become damaged, scarred, alveolar macrophage function diminished, gas trapping occurs

Emphysema (EM) – mainly affects the acinus

- Pollutant or smoke → causes irritation and inflammation of airway epithelium → infiltration of inflammatory cells and release of cytokines (neutrophils, macrophages, lymphocytes, leukotrienes, interleukins) → Proteases cause breakdown in elastic tissue → results in destruction of alveolar septa and loss of elastic recoil of bronchial walls →

causes collapsed airways during exhalation → decrease alveolar surface area causes an increase in 'dead space', alveolar hyperinflation, and impaired oxygen diffusion. Loss of elastic recoil decrease amount of air that can be expired passively → increases/causes gas trapping, v/q mismatch, hypoxaemia. Decreased forced vital capacity, decreased forced expiratory volume, BUT increased total lung capacity (due to gas trapping).

- NOTE if someone's emphysema is due to a lack of alpha-1 antitrypsin production, their emphysema is due to a lack of these alpha 1 antitrypsin because it is a protease inhibitor. SO without it, excessive damage due to unchecked proteases occurs.

| SIGNS AND SYMPTOMS | | |
|---|---|---|
| **Cough and Sputum Production for > 3 months for 2 yrs+ (CB)** | **Frequent resp infections** | **Barrel Chest (esp emphysema)** |
| **Muscle Weakness** | **Bloating and oedema (chronic bronchitis)** | **Hypoxaemia** |
| **Hypercapnia** | **Cor Pulmonale (**caused by hypoxic vasoconstriction and pulmonary hypertension**)** | **Bronchospasm** |
| **Purse lip breathing (**b/c it increases air pressure in the lungs to help prevent airways from collapsing when breathing**)** | **Dyspnoea** | **Weight Loss (emphysema)** |
| **Wheeze and Crackles on auscultation (CB)** | **Cyanosis (CB)** | |

| RISK FACTORS AND CAUSES | | |
|---|---|---|
| **!!Smoking!!** | **Air Pollution** | **Occupational Exposures** |
| **Alpha-1 Antitrypsin Deficiency** | **Recurring Resp Infections** | **Hx asthma** |
| **Underdeveloped lungs** | **>40 age** | **Genetic** |

| DIAGNOSIS | | |
|---|---|---|
| **Spirometry** (pulmonary function tests) | **Chest X-Ray** | **CT Scan** |

**Lab Tests:** arterial blood gas, AAT deficiency

**Reid Index** (only postmortem though)

## Nursing Considerations & Interventions

1) Oxygen Management (sats 88-92%)
- Aim for lower O2 Sats than you normally would as excessive oxygenation can cause hypercapnic respiratory failure in COPD patients → increases V/Q mismatch and the Haldane Effect
2) Respiratory assessment
3) Lifestyle Modifications
- Cease smoking

# Pneumothorax

The presence of air or gas in the pleural space caused by rupture in visceral pleural or the parietal pleura and chest wall.

Types:

1) Spontaneous: pneumothorax acquired in the absence of trauma
2) Primary: found in patients without underlying pulmonary pathology
3) Secondary: found in patients with underlying lung disease and damage to the alveolar-pleural barrier (COPD or Asthma)
4) Traumatic: from any kind of trauma to the chest wall, inc. surgical complications
5) Tension: complication, can develop when pressure in pleural space pushes against collapsed lung and causes compression atelectasis. Can eventually push on heart and cause a decrease or no cardiac output.
6) Closed: air leaks into pleural space due to disruption of visceral pleura without trauma
7) Open: commonly due to acute external injury to chest (e.g. gunshot, stabbing, motor vehicle accident), causes air to enter pleural space due to injury.

## Pathophysiology

Disruption of the parietal or visceral pleural or the tracheobronchial tree occurs → air enters the pleural space (due to negative pleural pressure) and is trapped → air between visceral and parietal pleura separates the two areas → pressure increases in the ipsilateral hemithorax, creating positive pressure in the lungs → Increased pressure causes the ipsilateral lung to partially or fully collapse → lung is unable to expand during inspiration due to the change from negative to positive pressure in the lungs. Decrease in vital capacity → no air entry into the effected lung → causes decreased chest wall expansion on affected side, decreased breath sounds, dyspnoea, hypoxia, decreased SpO2, tachypnoea, impaired gas exchange at the alveoli, V/Q mismatch and blood shunt.

Progression to Tension Pneumothorax: increased pressure in the ipsilateral hemithorax → mediastinal shift away from affected side and compression of the superior vena cava and/or inferior vena cava → decreased blood return to the right atrium → blood backs up into venous system → decreased cardiac output, increased jugular venous pressure, tachycardia, hypotension

Notable aetiological features

- Increased sound resonance through air compared to lung tissue → tympany/hyper-resonance on percussion
- Sound vibrations unable to travel from larynx through lung tissue to chest wall → decreased vocal fremitus

## SIGNS AND SYMPTOMS

| | | |
|---|---|---|
| **Chest Pain** (unilateral, pain with inhale esp.) | **SOB** | **Cyanosis** |
| **Tachypnoea** | **Tachycardia** | **Fatigue** |
| **Dyspnoea** | **No breath sounds on Auscultation in affected area** | **Tracheal deviation** |
| **Decrease in Tidal Volume** | **Poor Tissue perfusion** | **Anxiety, Stress, Agitation** |
| **Decreased air entry on Auscultation** | **Decreased chest movement on affected side** | **Hypotension** |
| **Decreased O2 Sats** | **Increased Jugular Venous Pressure** | |

## RISK FACTORS AND CAUSES

| | | |
|---|---|---|
| **Chest Injury/Blunt Force Trauma** | **Lung Disease** | **Ruptured Air Blisters** |
| **Mechanical Ventilation** | **Smoking** | **Genetics** |
| **Hx of pneumothorax** | **Hx Asthma** | **Hx Pneumonia** |
| **Hx COPD** | **Cystic fibrosis** | **Lung cancer** |
| **Acute respiratory distress syndrome** | **Gunshot/Stab Wound** | **Medical Procedures** |
| **Pregnancy** | **Endometriosis** | **Scuba Diving** |

## DIAGNOSIS

| CT Scan | Chest X-Ray | Lung Ultrasound |
|---|---|---|
| **Lab Testing** (arterial blood gas) | | |

## NURSING CONSIDERATIONS & INTERVENTIONS

1) Treatment
- Thoracentesis: needle inserted between ribs to evacuate air in chest
- Chest tube drainage/Underwater-Seal Drainage System (UWSD)
- Intercostal Catheter (ICC)

2) Monitor Pain
3) Respiratory assessment and full set of obs

# PLEURAL EFFUSION

Abnormal fluid collection which occurs at the base of the lung, in the pleural space, either because the body produces too much pleural fluid (transudative or exudative) or the lymphatic's cant effectively drain the fluid. Usually secondary to another disease process (e.g. heart failure, TB, pneumonia). Not considered a disease itself. Excess fluid can either be protein-poor (transudative) or protein-rich (exudative)

## PATHOPHYSIOLOGY

**Transudate effusion** (change in oncotic or hydrostatic pressure): Too much fluid leaks out of the capillaries into the pleural space → due to increase in hydrostatic pressure (heart failure) and/or decrease in oncotic pressure (cirrhosis, nephrotic syndrome) in blood vessels → increased pressure in the vessels forces fluid out of the capillaries and into pleural space or a change in oncotic pressure causes fluid to move from the low to high solute concentration area. Fluid is low in protein and LDH

- E.g. a heart failure patient where the heart cannot effectively pump blood so it backs up into the pulmonary vessels and causes increased pressure in the vessels.

Increased hydrostatic pressure and/or plasma oncotic pressure

**Exudative effusion** (Increased capillary permeability) inflammation of the pulmonary capillaries occurs due to cytokine release → larger spaces between endothelial cells → allows leaking of fluid, immune cels, and large proteins to leak into the pleural space. Fluid is high in protein and LDH

**Lymphatic Pleural Effusion** (a chylothorax): thoracic duct disrupted and lymphatic fluid accumulates in pleural space due to an impaired ability to drain pleural fluid (e,g, damage during surgery, tumours)

| SIGNS AND SYMPTOMS | | |
|---|---|---|
| **Slow develop compared to pneumothorax** | **Sharp pain, gen on inhale** | **Dyspnoea** |
| **Tachypnoea** | **Anxiety, stress, agitation** | **Tachycardia** |
| **Decreased air entry at the case of the affected side** | **Orthopnoea** | **Crackles on auscultation** |
| **Dry Cough** | **SOB worse when lying down** | **Decreased tactile fremitus** |
| **Dullness on percussion** | **Decreased breath sounds on auscultation** | **Tracheal deviation** |
| RISK FACTORS AND CAUSES | | |
| **Heart Failure (transudative)** | **Nephrotic Syndrome (transudative)** | **Pneumonia (Exudative)** |
| **TB** | **Lugn Cancer (exudative)** | **Cirrhosis (transudative)** |
| **Infections** | **Pulmonary Embolism (exudative or transudative)** | **Kidney Disease (exudative)** |
| **Inflammatory Disease (exudative)** | **Open-heart surgery complication (exudative)** | **Chest trauma** |
| **Use of tobacco products** | **Exposure to asbestos** | |
| DIAGNOSIS | | |
| **Chest X-Ray** | **CT scan** | **Ultrasound** |
| **Pleural Fluid Analysis** | **Thoracentesis or Biopsy** | |
| NURSING CONSIDERATIONS & INTERVENTIONS | | |

1) Chest Drainage
- ICC, Pleural Catheter, or thoracentesis

2) General Nursing Care
- Full Vitals
- Resp Assessment
- Pain assessments

3) Treat underlying condition causing pleural effusion

## PHARMACOLOGY

| Drug Class | Indication | MOA | Contra/Comps |
|---|---|---|---|
| <u>Antihistamines</u> | | | |
| *H1-Antihistamines*<br>*-ine, -ate* | Seasonal Allergies<br>Adjunct: Allergic Reactions<br>Pruritus<br>Conjunctivitis<br>Allergic Rhinitis | Inverse agonist → binds to H1 histamine receptor sites to prevent histamine from binding → prevents inflammatory response → can help open airways<br>Antimuscarinic in some cases | **Comps**: Sedation, Dry moutn, Somnolence, Fatique, Pharyngitis, GI upset, headache, Nausea, Blurred vision<br>**Contra**: other depressants, alcohol, bronchial asthma, renal impairment |
| | **Example**s: Azelastine HCL, Bilastine, Cetirizine, | | |
| <u>Nasal Decongestants</u> | | | |
| *Intranasal Corticosteroids*<br>*-sone, -lone, -ide* | Cold and Flu<br>Allergic Rhinitis<br>Inflammatory Disorders | Reduces inflammatory response → decreases the secretion of inflammatory mediators, reduces tissue oedema and vasoconstriction | **Comps**: burning sensation on administration, nasal irritation, dryness, congestive heart failure, weight gain, menstrual irregularities, muscle weakness, fluid retention; Cushing's Triad |
| | **Examples:** Beclomethasone, Budesonide, Ciclesonide, Flucasone, Mometasone, Triamcinolone | | |
| *Sympathomimetics* | Cold and Flu<br>Allergic Rhinitis<br>Inflammatory Skin Disorders<br>COPD | Constrict dilated blood vessels in nasal mucosa by stimulating alpha (a1)-adrenergic receptors in smooth muscle → reduces blood flow in oedematous area, slows formation of | **Comps**: Rebound nasal congestion, Local ischemia and irritation, Insomnia, Agitation, Restlessness; aggravate: hypertension, cardiac arrhythmias, ischaemic heart disease, diabetes, hyperthyroidism |

| | | | |
|---|---|---|---|
| | | mucus, permits better drainage, relieves discomfort | **Contra**: Hypertension, heart disease, diabetes, hyperthyroidism |
| | **Examples**: Oxymetazoline HCL, Phenylephrine HCL, Pseudoephedrine HCL, Tramazoline, Xylometazoline | | |
| *Antimuscarinics* | Cold and Flu<br>Allergic Rhinitis | Blocks nasal muscarinic receptors → decreases rhinorrhea (runny nose) | **Comps**: nasal irritation and dryness |
| <u>Antitussives</u> | | | |
| *Antitussives / Suppressants* | Non-productive Cough | Suppresses the cough centre in the medulla to stop coughing from occurring | **Comps**: CNS depression, constipation, bronchial constriction, dizziness, nausea, sweating, hypersensitivity, restlessness, GI upset<br>**Contra**: pre-existing pulmonary distress, older adults, children, CNS Depressants |
| | **Examples**: largely narcotics: Codeine, Pholcodine, Dextromethorphan | | |
| <u>Medications Affecting Respiratory Secretions</u> | | | |
| *Expectorants* | Productive Cough | Decrease viscosity of sputum, stimulate and facilitate movement of sputum; hydrates dry, irritated tissue and provides soothing coating | **Comps**: stomach upset, nausea<br>**Contra**: Coughs with excess secretions |
| | **Examples**: Guaifenesin | | |
| *Mucolytics* | Product Cough<br>COPD<br>Cystic Fibrosis | Breaks disulfide bonds of glycoproteins in bronchial secretions → breaks down mucus, | **Comps**: bronchospasm, irritation, rash, dry mouth, facial flushing, constipation, tachycardia |

| | | | |
|---|---|---|---|
| | Pneumonia<br>Asthma | improves movement → removal by ciliary action and pulmonary ventilation improved | **Contra**: can react with other minerals and medications |
| | **Examples**: Acetylcysteine, Bromhexine, | | |
| *Surfactants*<br>*-actant* | Premature Birth<br>Lung Injury<br>DNA Mutations<br>Atelectasis | Mixture of lipoprotein and phospholipids that coat alveoli → changes the surface tension in the air sacs to ensure lungs do not collapse | **Comps**:<br>**Contra**: |
| | **Examples**: Beractant, Poractant Alfa | | |
| <u>Bronchodilators</u> | | | |
| *Sympathomimetics / Beta Receptor Agonists* (long acting LABA, short acting SABA)<br>-terol, -amol<br><br>***** | Relief of Bronchospasm (SABAs)<br>Control of Asthma and COPD Symptoms (LABAs)<br>Reduction of Oedema | Stimulates B2 receptors to increase the formation of cyclic adenosine monophosphate (cAMP) → Smooth muscle relaxation and bronchodilation | **Comps**: changes in heart function and BP, GI disturbances, CNS stimulation, insomnia, nervousness, anxiety, tremor<br>**Contra**: monoamine oxidase (MOA) inhibitors |
| | **Examples**: Salbutamol, Salmeterol, Formoterol, Indacaterol, Olodaterol, Seretide (combo salmeterol and flucasone (corticosteroid)) | | |
| *Antimuscarinics (muscarinic antagonists) – Anticholinergic Subclass*<br>*-ium Bromide*<br><br>***** | Bronchospasm<br>COPD<br>Asthma | Antagonise the action of acetylcholine at M3-muscarinic receptor and blocks parasympathetic nervous system response by decreasing intrinsic vagal tone in the airway → creates bronchodilation | **Comps**: urinary retention, constipation, ocular symptoms, dry mouth, pharyngitis, abdominal pain, upper respiratory infections<br>**Contra**: Glaucoma |
| | **Examples**: Ipratropium Bromide, tiotropium bromide, | | |

| | | | |
|---|---|---|---|
| *Methylxanthine Bronchodilators* | Reversible Bronchospasm<br>Chronic Bronchitis<br>Asthma<br>Emphysema<br>Paroxysmal Dyspnoea | Interferes with the action of Phosphodiesterase → causes increase in intracellular cyclic adenosine monophosphate (cAMP) → bronchodilation promoted | **Comps**: tachycardia, dysrhythmias, diuresis, CNS stimulation, insomnia, hyperexcitability, seizures, toxicity |
| | **Examples**: caffeine, Theophylline | | |
| <u>Prophylactics</u> | | | |
| *Corticosteroids*<br>*-sone,* | Prevent symptoms of COPD and Asthma | Decreases the number and activity of inflammatory cells, stops the rupture of mast cells, reduces the synthesis of inflammatory mediators, stops production of new antibodies → decreases mucus production, oedema and bronchoconstriction | **Comps**: increased susceptibility to infections, fluid and electrolyte disturbances, oral candidiasis; Cushing's Triad |
| | **Example**: ***Seretide MDI*** (combo of Fluticasone and Salmeterol) used for Asthma and COPD, Beclomethasone, | | |
| *Leukotriene Receptor Antagonists* | Prevent symptoms of COPD and Asthma | Blocks Leukotriene receptors in the lungs to stop leukotrienes from binding → prevents inflammation, bronchoconstriction, and lessens mucus production | **Comps**: GI upset |
| | **Example**: Montelukast | | |

| | | | |
|---|---|---|---|
| *Mast Cell Stabilisers* | Prevent symptoms of COPD and Asthma<br>Mild-Moderate Asthma Maintenance<br>Mastocytosis | Stabilise sensitised mast cells → prevent the release of mediators histamine and leukotriene → reduce airway inflammation and bronchospasms | **Comps**: cough and throat irritation |
| | **Examples:** sodium cromoglycate. Nedocromil sodium | | |
| *Monoclonal Antibody Therapy (Biologics)* | Add on therapy for Severe Asthma | Omalizumab: binds with immunoglobulin (Ig)E → stops it from binding to IgE receptors on mast cells → prevents allergic response<br>Mepolizumab: binds to and inhibits Interleukin-5 → leads to reduction and survival of eosinophils → reduces inflammation of airway | **Comps**: respiratory infection, headaches, sinusitis, sore throat |
| | **Examples**: Omalizumab, Mepolizumab | | |

# CARDIOVASCULAR

## DEFINITIONS

Zone of ischemia = the outer layer of ischaemic tissue around the zone of injury

Zone of Infarction = A central area of dead, necrotic tissue in an MI

Zone of injury = a layer of injured myocardial tissue around the zone of infarction

Non-Transmural Infarction = zone of infarction involves only a section of the myocardium, such as the endocardial layer