Al-Noor University College

Dept. of Anesthesiology

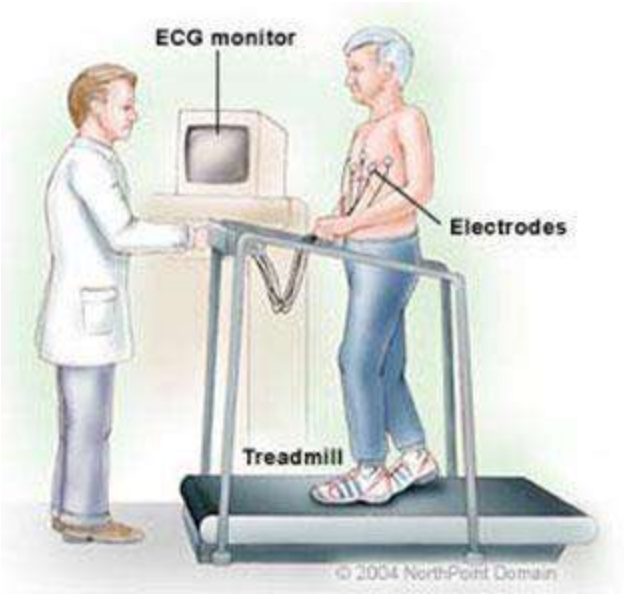
Subject: Practical Applied Physiology

Second Year

Lecturer: Asmaa J. Al-lella

Lab.6: Exercise Electrocardiography

Stress test: An exercise electrocardiogram (ECG) records heart's response to the stress of exercise. An exercise ECG measures heart's electrical activity, blood pressure and heart rate while exercise, usually by walking on a treadmill.



Why is it done?

- A stress test is usually done to pinpoint the cause of unexplained chest pain, especially if **coronary artery disease** (heart disease) is suspected.
- If have had a heart attack or heart surgery, it can help determine how much work or exercise can do safely.
- It may also be recommended if are experiencing irregular heartbeats (**arrhythmia**), very fast or slow heartbeats (tachycardia or bradycardia), palpitations (unusual throbbing or fluttering sensations in the heart), dizziness or excessive fatigue.

How do you prepare?

- Wear clothing and shoes that are comfortable for exercising.
- probably be told not to eat for at least two hours before the test.
- If a smoker, need not to smoke for at least two hours before the test.
- Talk to doctor about any medications are taking.

Other Types of Stress Tests?

Dobutamine or adenosine stress test: This is for people unable to exercise. You'll take a drug to make the heart respond as if you were exercising. This way, the doctor can still determine if there are blockages in the arteries.

Stress echocardiogram: An echocardiogram (often called "echo") is a graphic outline of the heart's movement. A stress echo can accurately visualize the motion of the heart's walls and pumping action when the heart is stressed; it may reveal a lack of blood flow that isn't always apparent on other heart tests.

Nuclear stress test: This helps figure out which parts of the heart are not working well. A small amount of radioactive substance will be injected into you. Your doctor will use a special camera to see rays emitted from the substance in your body. This will give them clear pictures of the heart tissue on a monitor. These pictures are done at rest and after exercise.

محاضرة (١): عناية مركزة

د. علاء غانم الكواز

Intensive care unit (ICU)

(Critical care unit/ Intensive therapy unit)

Definition:

Intensive care units (ICUs) are special ward in the hospital that provide management to critically ill patients, that their condition is unstable, life threatening or potentially threatening.

The ICU usually staffed with well trained nurses & medical professionals & contains special & advanced monitoring equipments & life support medications.

*** Primary function of ICU:**

One important key of success in any ICU is depending on good understanding of the primary functions of ICU services. There are 2 intervals of ICU functions:

1. During early acute period of primary pathology till the point when definitive diagnosis is reached, supportive measures will help to delay the collaps of body's vital functions.

2. During period from the point of primary Pathology diagnosis with putting the plan of specific therapeutic measures throughout the duration of management until recovery.

Difference of ICU ward from usually medical ward in the hospital

1. ICUs take care of patients with sever or life threatening illnesses or injuries, which require life support equipments, close observation & monitoring.
2. The ICUs contains highly trained nurses, Anesthetists, physicians, respiratory therapists who are specialized in dealing with critically ill patients.
3. ICUs also characterized by high staff- patient ratio (1:2 or 1:1).
4. It contents advanced medical equipments and resources that is not routinely available in other hospital ward.

Definition of critical care

It is a term used to describe the care of patients who are extremely ill/ or whose clinical condition is unstable or potentially unstable.

Aim of critical care:

1. Saving the life after primary insult by providing optimal therapy to critically ill patient, and treating the immediate life-threatening condition of the patient.
2. Using advanced therapeutic, monitoring and diagnostic technology.
3. Maintain vital organ system functioning to improve the patient's condition until the underlying injury or illness can be treated.

Primary functions of ICU

It is to provides supportive medical measures and care for critically ill patients which include:

1. Respiratory care
2. Circulatory care
3. Fluid & Electrolytes balance
4. Acid- base balance
5. Metabolic status monitoring and regulations.
6. Nutritional care, Entral Feeding, parantral feeding.
7. Physiotherapy & rehabilitation.
8. Psychotherapy.

Types of ICU

1. **General types**

a. Medical intensive care unit (MICU)
b. Surgical intensive care unit (SICU)
c. Main or mother intensive care unit (M.I.C.U) which is combined medical & surgical & Trauma ICU.

2. **Specialized type**

a. Post-anesthetic care unit (PACU) usually inside operating theater, patient can be held for 6hr or more.
b. Neonatal intensive care unit (N.I.C.U)
c. Pediatric intensive care unit (P. I. C. U)
d. Coronary care unit (CCU)
e. Respiratory care unit (RCU)
f. Neurosurgery intensive care unit (NSICU)
g. Trauma intensive care unit (T. I. C. U)
h. Burn intensive care unit (B. I. C. U)

High dependency unit

Usually provide a level of care intermediate between intensive care unit & general ward care. It may be located in or near speciality word, it provides invasive monitoring & support for patients with or at risk of developing acute single organ failure, equipments should be available to manage short term emergencies.

Classification of critical care units

1. **Level (1):**

- Usually in small hospital.
- Provides observation, monitoring and short term ventilation.
- The medical director should be a certified intensive care specialist, the nurse- patient ration is 1:3.

2. **Level (2):**

- Located in large hospitals.
- Should capable of providing a high standard of general intensive care including multiple system life support, provides observation, monitoring and long term ventilation.
- The medical director and at least one other specialists should be certified intensive care specialist.
- Nurse- patient ratio is 1:2, and medical staff are available in the unit all the time.

3. **Level (3)**

- Located in major referral hospital.
- It should provide all aspect of intensive care management for indefinite period.
- Should be staffed by intensive care specialist, critical care nurses and all health professionals.
- Complex investigation & imaging is available at all times.
- Nurse-patient ratio is 1:1 & should be present all the times.

محاضرة (٢) عناية مركزة

د. علاء غانم الكواز

Structure & designing of I.C.U

Design of ICU

1. Designs should produce improvement of physical and psychological state of the patient and staff.
2. In general ICU beds is 2-10% of total hospital beds.
3. It should meet the need of the hospital, sometimes one ICU is not enough; we should prepare 2 or more ICU.
4. The design should allow all patients to be easily observed.
5. It can be linear, U-shaped or circular placement of beds with central nursing station.
6. It should contain open bed area in addition to isolated rooms for infectious cases.
7. The no. of beds should not less than 4 & not more than 20.
8. The optimal no. is 14, if more than 14 is needed, then 2 ICUs can be opened.

Size of ICU:

The size depends on the type of services provided.

1. In general hospital 2% of hospital beds.
2. In sub-specialty hospital 10% of total beds.

Location of ICU:

1. Should be centrally located with easy access to operating theater, emergency, laboratory, radiology and other departments.
2. Easily to be reached.
3. Away from main hospital traffic.
4. Restricted area & restricted entry.

Structure of ICU:

1. More than one source of oxygen supply (both central and cylinder) is needed for each bed, 4 oxygen inlet, 3 air outlets, and 3 suction inlets.
2. More than one source of power supply (at least 16-20 power points near every bed).
3. 4 data outlet.
4. Adequate artificial light
5. Large windows with good access of sun light.

Entrance to ICU

1. Broad corridor allow easy transport of beds and equipments.
2. Double door entrance, over size elevator and doors.
3. Changing room for shoes, Gowns & masks.
4. Separated toilet & kitchen facilities.
5. Areas for supportive and storage services.
6. Emergency Exit

Bed space

Sufficient space is required around the beds for free movement of ventilator, monitors & other equipments

1. At least 20 m^2 for each bed space
2. Total patient care area is 2.5-3 times of total bed space area
3. Minimum of 1.2 m head- wall space
4. Minimum of 1.8 m leg space
5. Space between two bed is 2 m
6. In circular design minimum of 3.5 m space between center & beds.
7. The isolated room is minimum of 25 m^2 space to accommodate patient, staff and equipments with at least 2.5m traffic area beyond the bed.

Supportive and storage space

1. Storage area is at least 10m^2 per bed.
2. It should be away from patient's area but not more than 30m , it contains intravenous fluids, giving sets, sheets and dressing, gowns.. etc.
3. Supportive spaces of dirty (25m^2) and clean (15m^2) rooms are necessary.
4. Doctor on call rooms (15m^2) with wash room and other room with food and drinks facilities.

ICU environment

1. Large windows with plenty of sunlight.
   Patient exposing to sun light have been shown to decrease stress and improve sleep quality, lack of natural light and outside view increase incidence of disorientation, clock and calendar for each bed space are useful for patients orientation.
2. Air conditioning with temperature maintained at 25-27 ℃ and humidity of 40- 50%
3. Positive pressure air flow from patient to outside
4. Easily cleaned walls and floor (eg. Mosaic floor).
5. Noise free, with noise absorbable materials, silence or soft music, and light colors.

Nurse station

1. Should be central and allow all patients to be observed
2. Central monitoring system
3. Complete visibility of all patients
4. Special places for patients files & records and reference books.
5. Pharmacy and refrigerator for emergency and essential drugs
6. Easy communication, Telephone, intercom system and computers with internet connection.

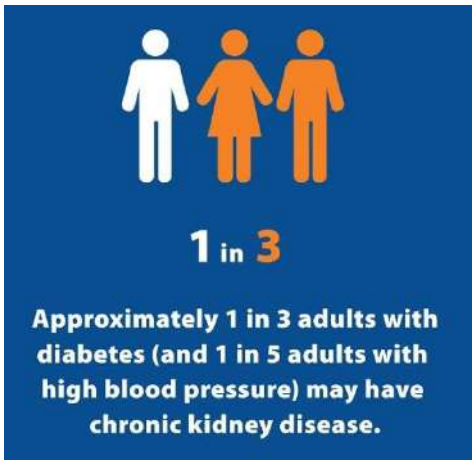
7. **Staffing**

1. Doctor, anesthetist, physician, and sometimes surgeon.
2. Nursing staff: 1:1 or 1:2 patient ratio
3. others:

- Radiographer
- Physiotherapist
- Pharmacist
- Dietitians
- Psychotherapist
- Laboratory staff if available.

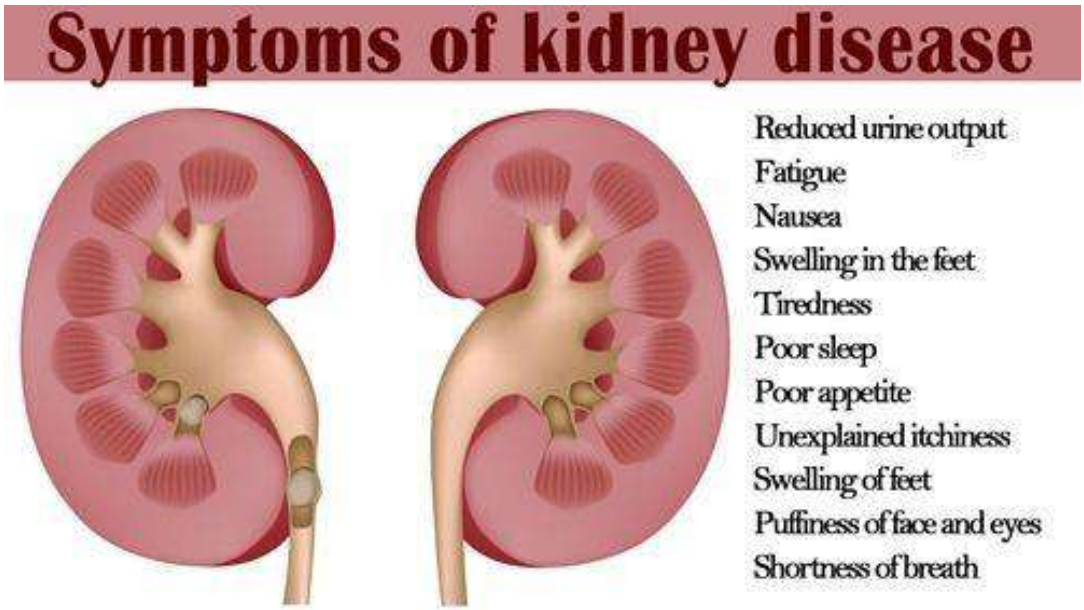
Renal failure: acute renal failure, chronic renal failure: clinical features, investigations and treatment.

Definition/description: Renal failure is the loss of renal function leading to uraemia. Two forms exist: acute and chronic RF. chronic RF is characterised by a gradual and permanent loss of renal function.



Cause: These include diabetes mellitus, Glomerulonephritis (GN), pyelonephritis, hypertension, renal stones, bladder outlet obstruction, and connective tissue disease.

Symptoms and signs:

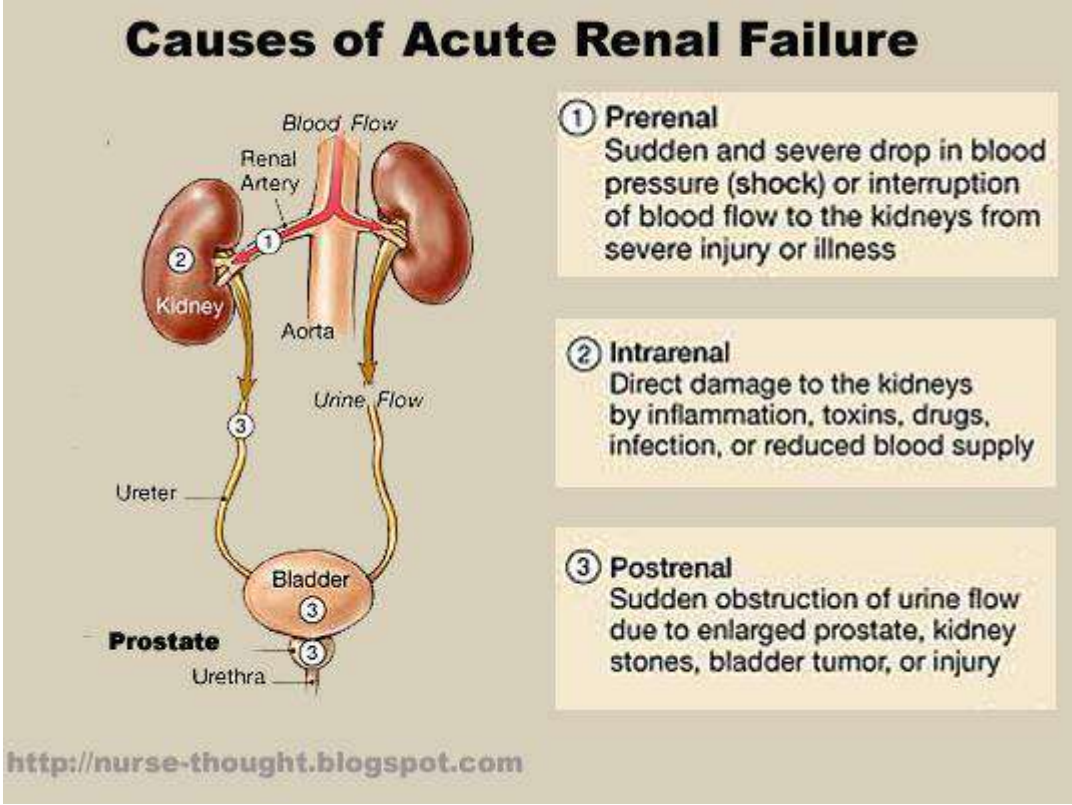


Acute renal failure (ARF) is also called acute kidney injury (AKI)

This is characterised by a rapidly progressive loss of renal function resulting in oliguria, and fluid and electrolyte imbalances. Acute RF involves rapid deterioration of renal function within hours or days. Causes include injuries (due to accidents), complications of surgeries (such as bypass surgery on the heart restricting blood flow to the kidneys for extended periods of time), chemicals and accidental overdoses of drugs.

When kidneys fail to filter properly, waste accumulates in the blood and the body. This condition is called azotemia. This stage may be asymptomatic in some patients.

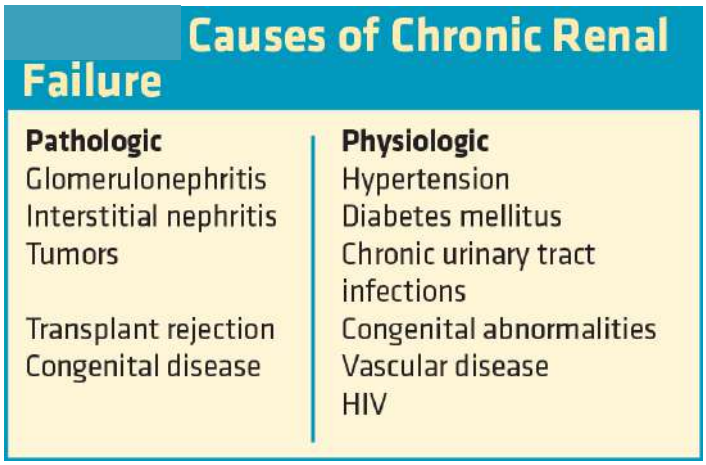
Renal failure accompanied by noticeable symptoms is termed uraemia which is characterised by high levels of urea in the blood. The term uraemia is used for the illness accompanying kidney failure.



In chronic renal failure CRF any of the following symptoms and signs may be encountered:

Apathy, confusion, drowsiness (due to accumulation of nitrogenous end products), ammoniacal breath odour, brown-coated tongue, metabolic acidosis leading to over-breathing, anorexia, nausea, vomiting, bleeding or bruising tendencies, anaemia,

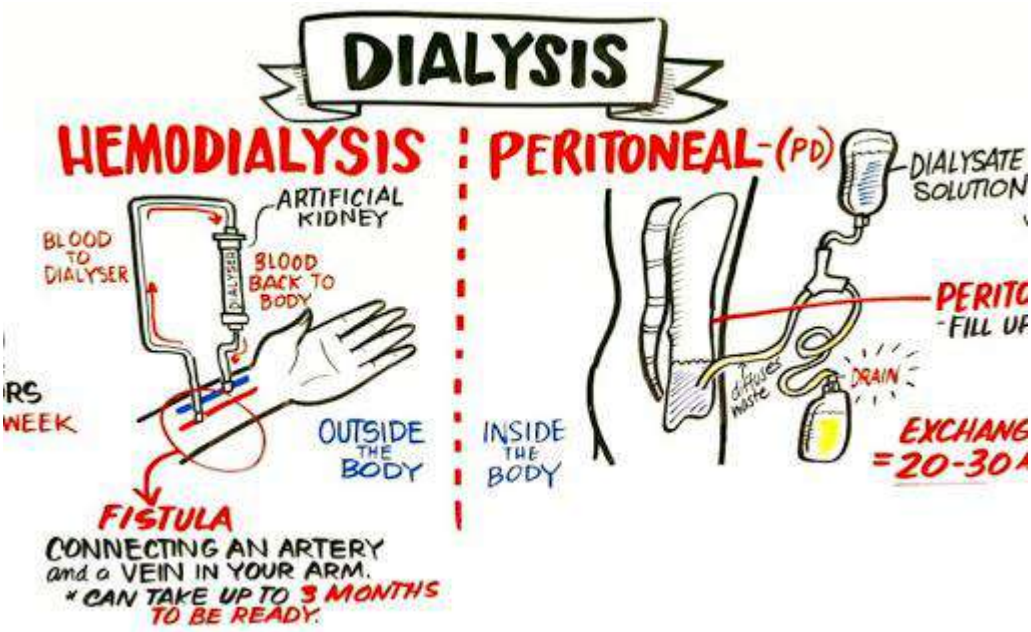
polyuria, peripheral oedema, increased pigmentation, ascites, pleural effusion and pericarditis.

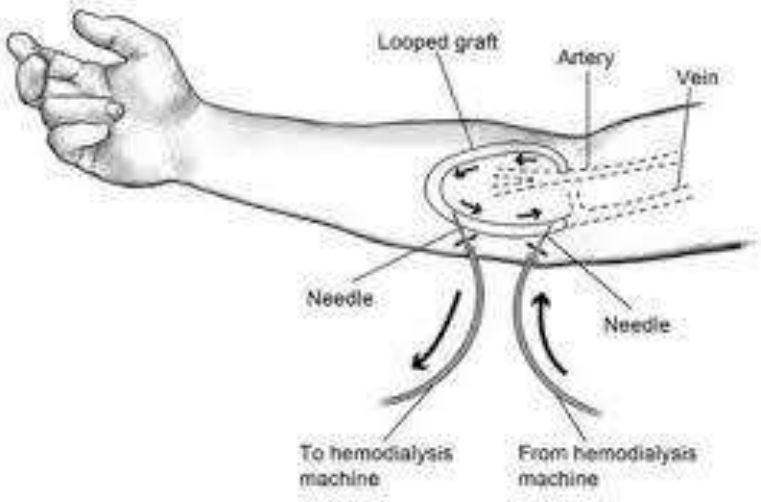


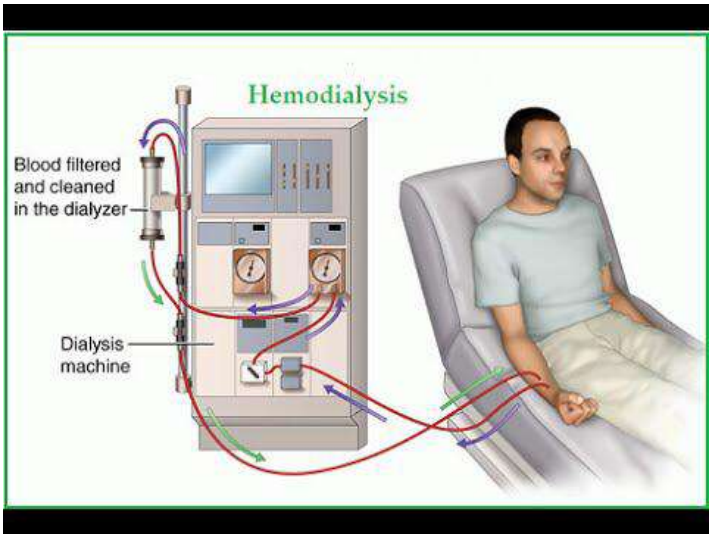
Investigations: Biochemical: increased urea and creatinine, hyperkalaemia (abnormally high levels of potassium in the circulating blood), hypocalcaemia (abnormally low concentrations of calcium in the circulating blood), hyperphosphataemia (abnormally high levels of phosphates in the circulating blood), and hyponatraemia (abnormally low concentrations of sodium ions in the circulating blood). Urine examination includes microscopy for casts, protein analysis, specific gravity and creatinine clearance. Radiology and biopsy includes plain x-ray, retrograde pyelography and renal biopsy.

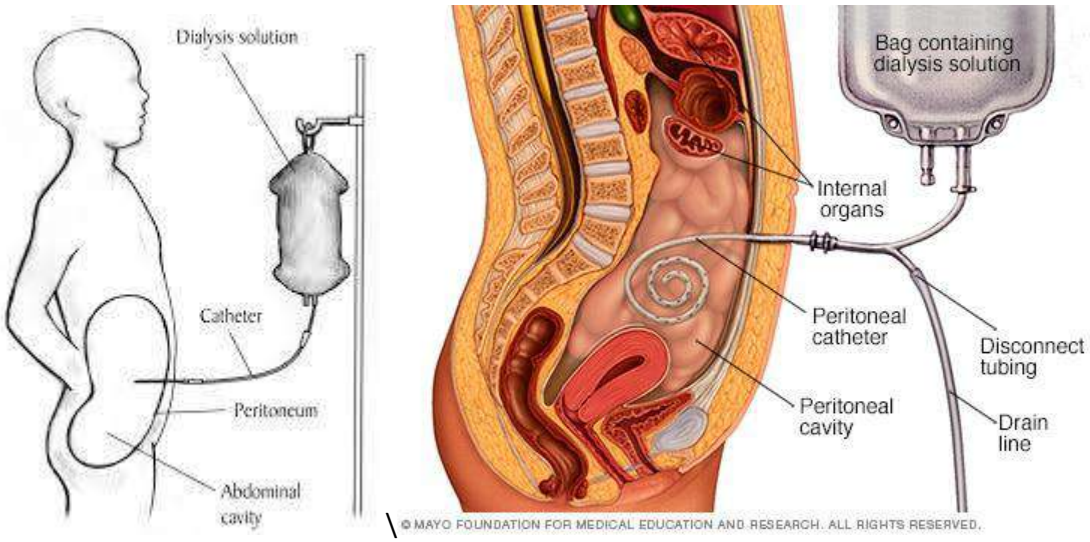
Management:

Management of BP is an essential part of the treatment. BP should be maintained at less than 130/80 mmHg in chronic renal failure (CRF), and for those CRF patients with diabetes, BP should be maintained at less than 120/70mmHg. Vitamin D, avoidance of nephrotoxic drugs such as tetracyclines, a diet low in protein and salt, and renal dialysis (haemodialysis or chronic ambulatory peritoneal dialysis) are the recommended management strategies for CRF.


DIALYSIS
HEMODIALYSIS
PERITONEAL-(PD)
ARTIFICIAL KIDNEY
BLOOD TO DIALYSER
BLOOD BACK TO BODY
DIALYSER
OUTSIDE THE BODY
INSIDE THE BODY
DIALYSATE SOLUTION
DRAIN
FISTULA
CONNECTING AN ARTERY and a VEIN IN YOUR ARM.
* CAN TAKE UP TO 3 MONTHS TO BE READY.


Looped graft
Artery
Vein
Needle
Needle
To hemodialysis machine
From hemodialysis machine


Hemodialysis
Blood filtered and cleaned in the dialyzer
Dialysis machine


Dialysis solution
Catheter
Peritoneum
Abdominal cavity
Bag containing dialysis solution
Internal organs
Peritoneal catheter
Peritoneal cavity
Disconnect tubing
Drain line
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Ischemic heart diseases: clinical features, diagnosis, treatment.

DEFINITION

Ischemic heart disease (IHD) describes the condition in which atherosclerotic plaque is present in the coronary arteries, giving way to coronary artery disease (CAD).

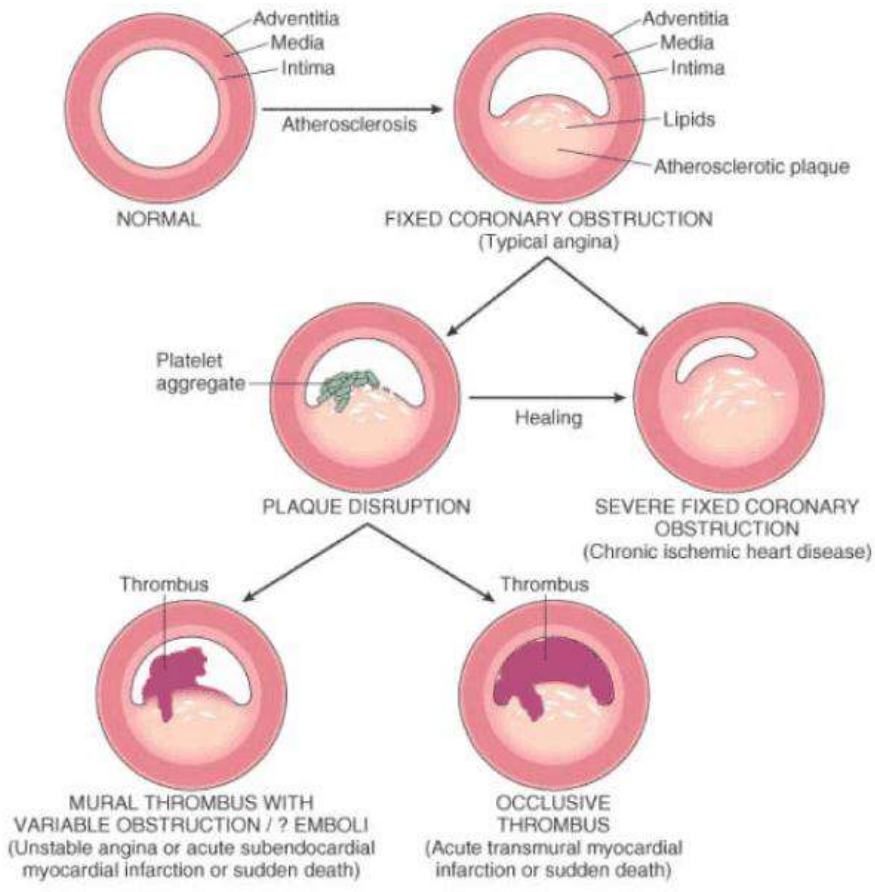
Ischemic Heart Disease

Ischemic Heart Disease

- **Hypoxemia (diminished transport of oxygen by the blood) less deleterious than ischemia**
- **Also called coronary artery disease (CAD) or coronary heart disease**
- **IHD =Syndromes**
  - **late manifestations of coronary atherosclerosis**
- **Cause => 90% of cases, coronary atherosclerotic arterial obstruction**

Ischemic Heart Disease

- **Classification = mainly 4 types**
  - **Myocardial infarction (MI)**
  - **Sudden cardiac death**
  - **Angina pectoris**
  - **Chronic IHD with heart failure**



Ischemic heart disease risk factors

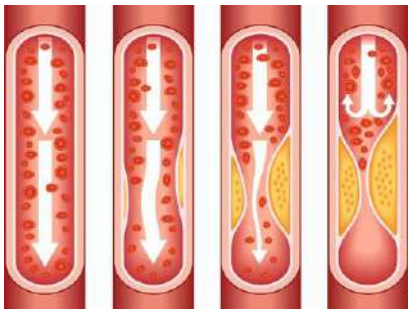
- Hypertension.
- Smoking.
- Diabetes mellitus.
- Family history (1st-degree relative <60yrs old with IHD).
- Hyperlipidemia.
- Male gender

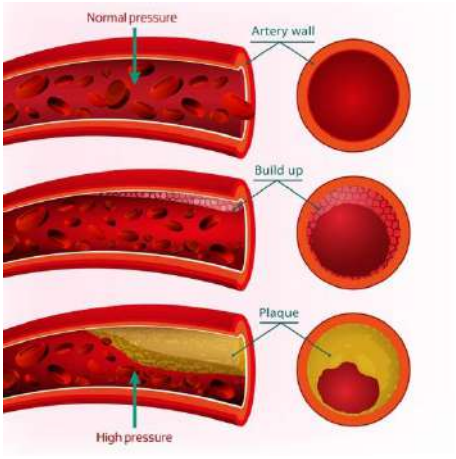
- ***Angina (Angina pectoris)***

Definition/Description: Angina, also called angina pectoris, is a common symptom characterized by pain of cardiac origin as a result of Ischemic heart disease (IHD). IHD causes an imbalance between the myocardial oxygen supply and demand.

Causes: A reduction of the coronary arterial luminal diameter (by 70-90%) due to Ischemic heart disease (IHD) causes angina.

Non-IHD diseases that can cause angina include coronary artery spasm (Prinzmetal's angina), aortic stenosis, and cardiomyopathy.





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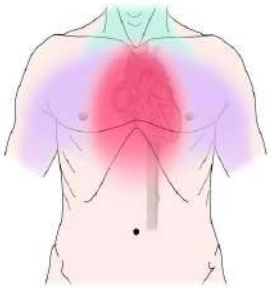
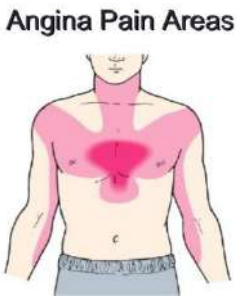
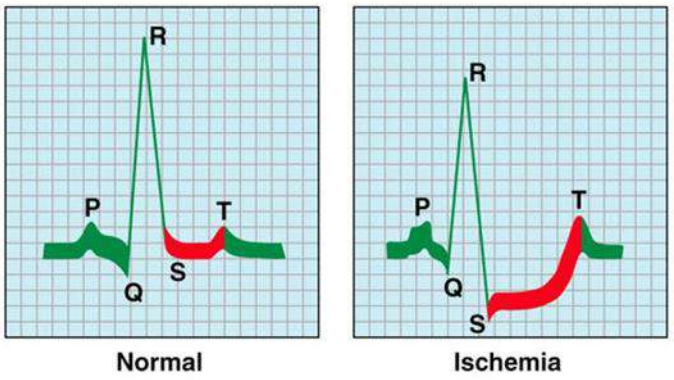
Symptoms and signs: These include severe central chest pain (gripping, constricting, crushing or tightness) often with shortness of breath, faintness and pain radiating to the left (and sometimes right) arm, and into the neck and jaw. Pain is typically induced by exercise, emotion, a heavy meal and cold weather, and is relieved by rest and nitrates. This is called **stable angina**.

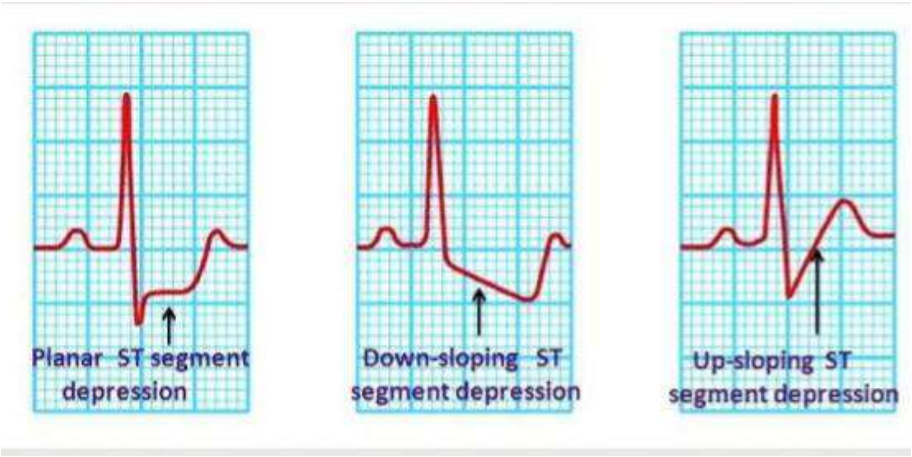




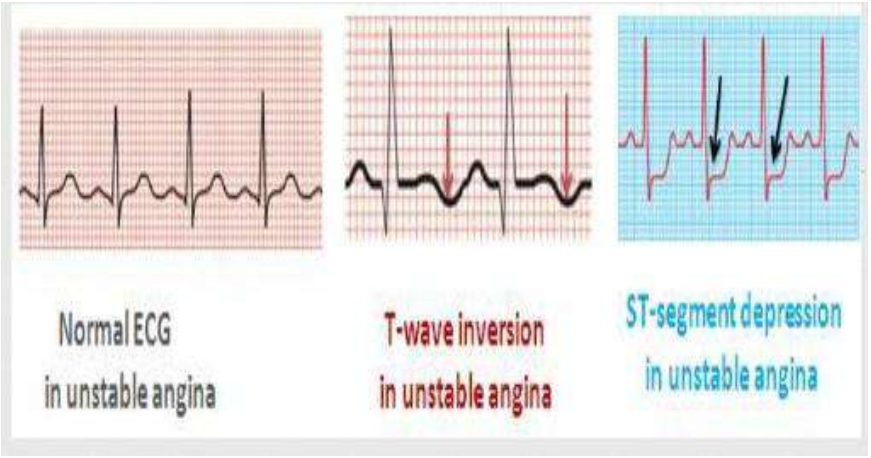
If angina is of recent onset, severe and rapidly worsening on minimal or no exertion, and lasts longer than a few minutes, it is considered as **unstable angina**. This is usually a forerunner of myocardial infarction (MI). The pain of myocardial infarction is of similar character and site to stable angina, but is more prolonged, more severe, and accompanied by nausea, vomiting, breathlessness, sweating, and abnormal heart rate and rhythm. This pain is not relieved by nitrates. Signs of angina are sometimes absent. Risk factors such as hypertension may be identified in a majority of cases.

Investigations: Electrocardiogram (ECG). This reading is abnormal during an attack, and often normal at rest. If the ECG has been recorded as normal during rest (in a patient with suspected angina), a treadmill exercise ECG or radionuclide scanning is usually considered.



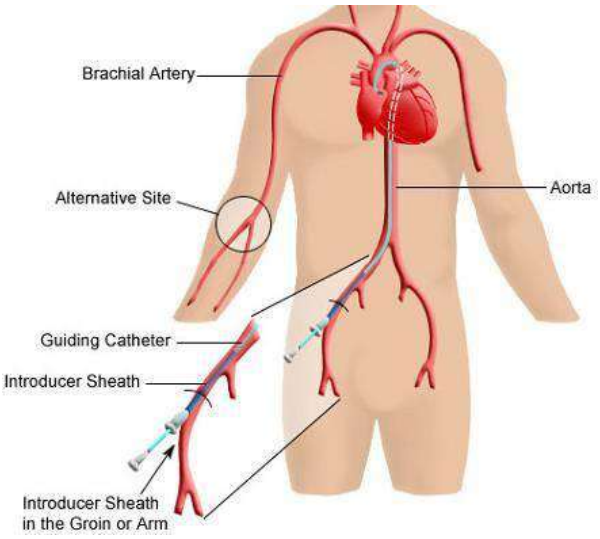


Stable Angina



Unstable Angina

ECG and BP are monitored during the recovery period after exercise. Coronary angiography is considered for those who may require angioplasty or bypass surgery.



A full blood count (FBC) and erythrocyte sedimentation rate (ESR) are required to exclude non-atheromatous causes of angina.

Management: Recognition and correction of risk factors such as hypertension, smoking, obesity, diabetes and Hyperlipidemia are the key factors in the management of angina.

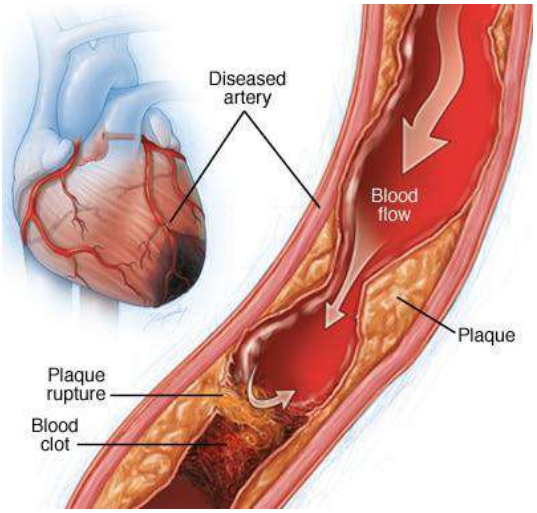
Drug therapy for angina includes aspirin (75 mg daily), nitrates (isosorbide mononitrate 20 mg bd), β-blockers (atenolol 50 mg daily), and calcium channel antagonists (nifedipine 10 mg tid or amlodipine 5-20 mg/day, or ATP-sensitive potassium channel activators (nicorandil 10-20 mg bd).

Surgery includes coronary angioplasty revascularisation (with stents) for proximal arterial stenosis, and coronary artery bypass surgery for triple coronary artery disease.

- ***Myocardial infarction (MI)= heart attack***

Definition/description: Death (necrosis) of a part of the heart muscle due to total occlusion of the coronary artery results in myocardial infarction.

Cause: Embolism following rupture of atheromatous coronary artery plaque is the major cause of myocardial infarction. MI is common in the winter months, and may be precipitated by vigorous exercise, major surgery or infections.



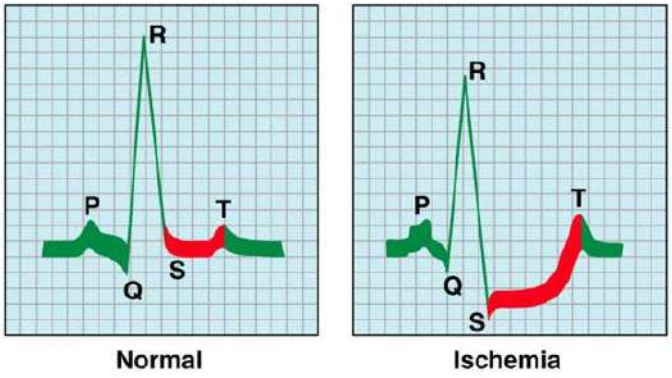
Symptoms: These include severe, crushing, central chest pain, often radiating to the neck, left arm and mandible, and not relieved by nitrates. Pain is usually associated with nausea, sweating, breathlessness and vomiting. In some cases symptoms do not occur and infarction is discovered incidentally when an ECG is performed at a later date. In this situation the infarct is called a **silent infarct.** Sudden death is due to ventricular fibrillation in 50% of heart attack patients.

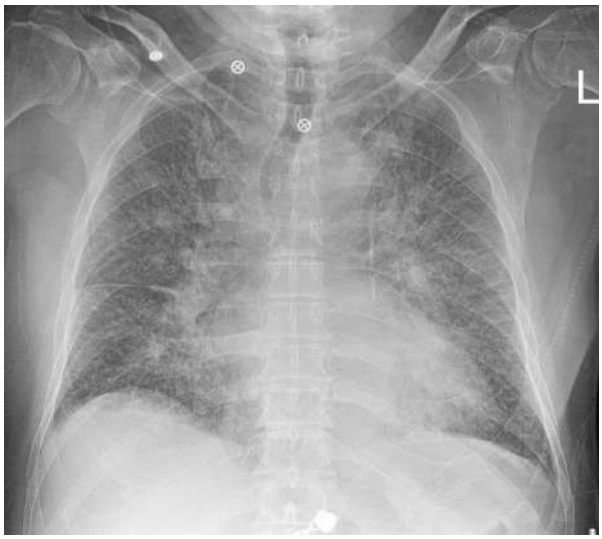
Signs: Common signs of myocardial infarction include pallor, circulatory shock, tachycardia, low blood pressure, cyanosis and gallop rhythm. Gallop rhythm usually

refers to the abnormal rhythm of the heart on auscultation. It includes three or four sounds, thus resembling the sound of a galloping horse.

Investigations: These include abnormal ECG findings and plasma cardiac enzymes such as creatine kinase, transaminases and lactate dehydrogenase (elevated). The troponin level is also elevated in MI. Chest x-rays are used to identify pulmonary oedema, and aortic dissections are required.

- Detectable by changes in S-T segment of ECG
- Myocardial infarction (MI) is a heart attack
  - Diagnosed by high levels of creatine phosphate (CPK) & lactate dehydrogenase (LDH)





Pulmonary Oedema

Management: Therapeutic management of MI includes aspirin (300 mg soluble) as soon as possible and opiates (diamorphine 2.5 mg IV with an antiemetic (metoclopramide 10 mg IV). Immediate transfer of the patient to ICU is required. ECG recording, bed rest with high flow oxygen administration, thrombolytic therapy (subcutaneous heparin 5000 IU eight hourly) and monitoring blood cardiac enzymes are other essential measures.

Arrhythmias: cardiac arrest.

Cardiac arrhythmias

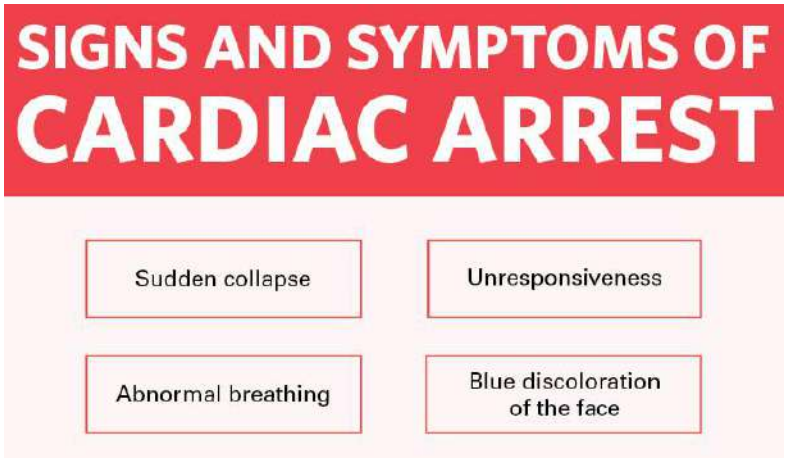
Definition/description: Loss of rhythm resulting in irregularity of the heartbeat is called arrhythmia. Cardiac arrhythmias are divided in to two categories: (1) supraventricular and ventricular arrhythmias, and (2) bradyarrhythmias and tachyarrhythmias.

Cardiac arrhythmias include isolated ectopic beats, bradycardia and tachycardia. Cardiac arrest can occur as a result of arrhythmias.

Causes: Cardiac arrhythmias are associated with a range of diseases of cardiac and non-cardiac origin. These include ischaemic heart disease, myocardial infarction, rheumatic heart disease, congestive heart failure, pneumonia, obstructive lung disease, thyrotoxicosis, systemic infections, drug-related side effects, and electrolytic imbalances.

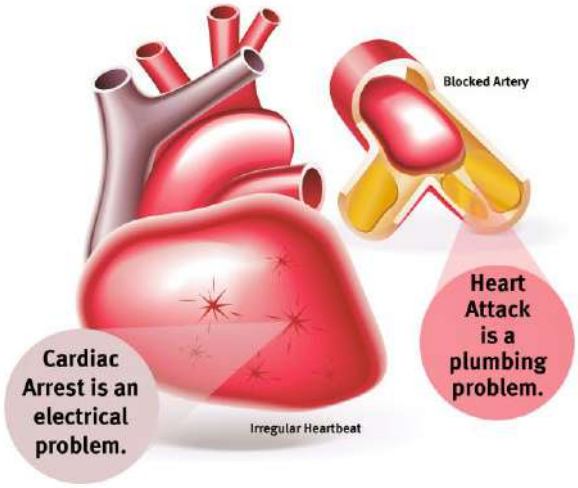
Symptoms and signs: Cardiac arrhythmias can cause palpitations and collapse.

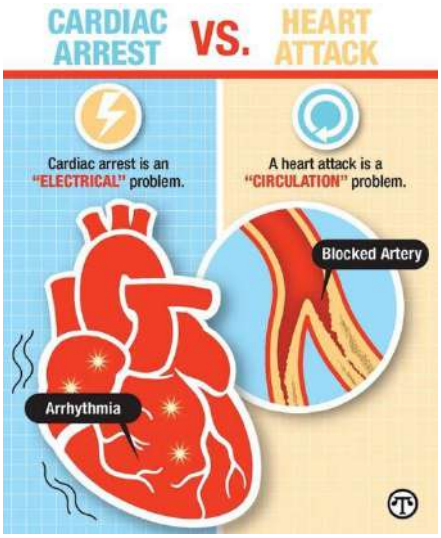
Irregularly irregular pulse and rapid atrial rhythm can be considered as signs of atrial fibrillation. The rate at the apex is faster than that at the radial artery.



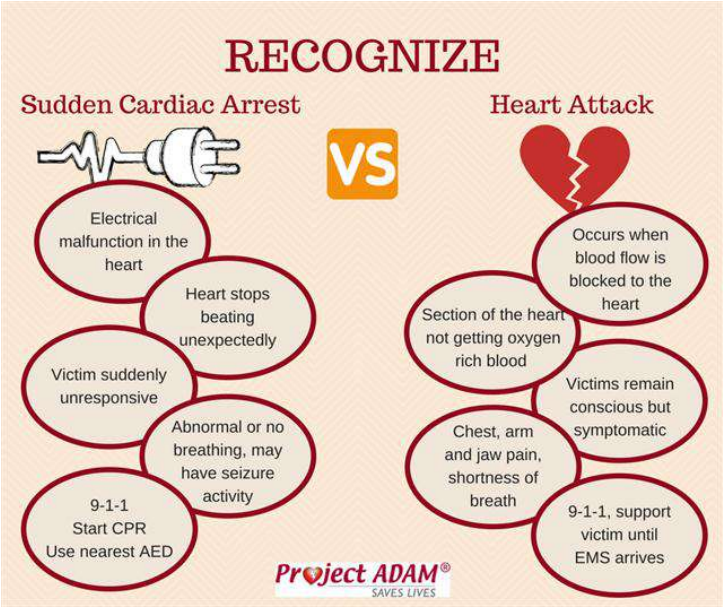
A sinus rate greater than 100 beats per minute is considered as tachycardia. A sinus rate of less than 60 bpm is considered to be bradycardia.

Sudden Cardiac Arrest





Check for obvious causes using ABCs (**A**irway patency, **B**reathing adequacy, and **C**irculatory adequacy). Sudden cardiac arrest is often a strong indication for open thoracotomy to inspect the heart for pericardial tamponade or other injuries or to perform open-chest cardiac massage; abdominal incision may also be indicated to look for other sources of bleeding; rapid blood and fluid resuscitation are continued as indicated.



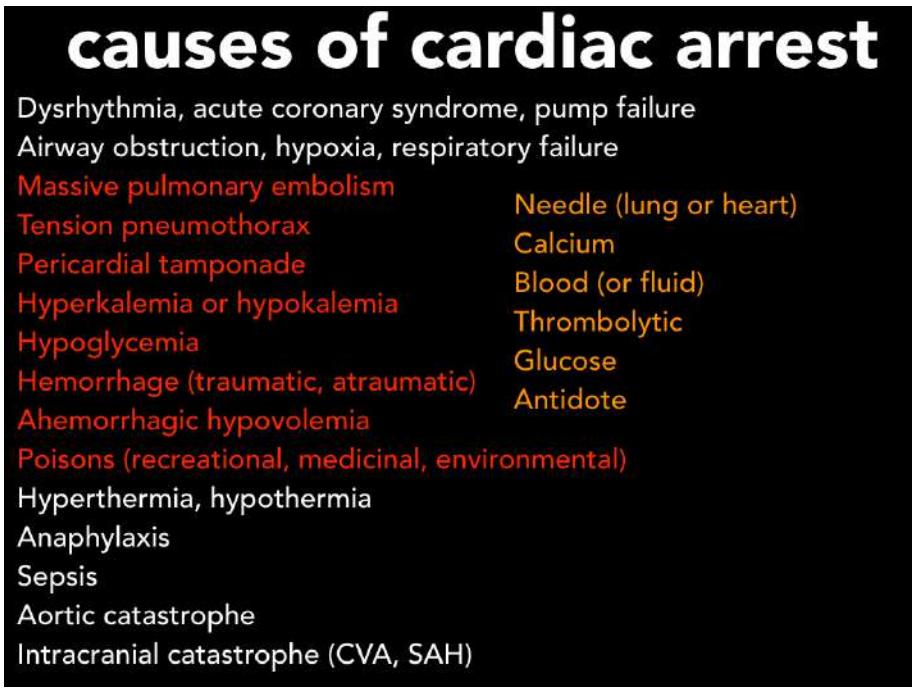
Unexpected cardiac arrest has been reported after the routine administration of succinylcholine, with less than 40% of patients successfully resuscitated.

Cardiorespiratory arrest

Confirm absence of pulse, blood pressure, and/or respirations.

Causes Myocardial infarction (MI), pulmonary embolism (PE), trauma, tension pneumothorax, electrocution, shock (septic, cardiogenic, neurogenic, hypovolemic,

and anaphylactic), hypoxia, hypercapnia, hypothermia, electrolyte/acid/base imbalance, drugs (e.g., digoxin).



Basic life support Request help immediately. Check for responsiveness. Check breathing. Ask someone to call the arrest team and bring the automatic external defibrillator. Note the time. Check the pulse for no longer than 10 seconds. Place patient in a supine position.

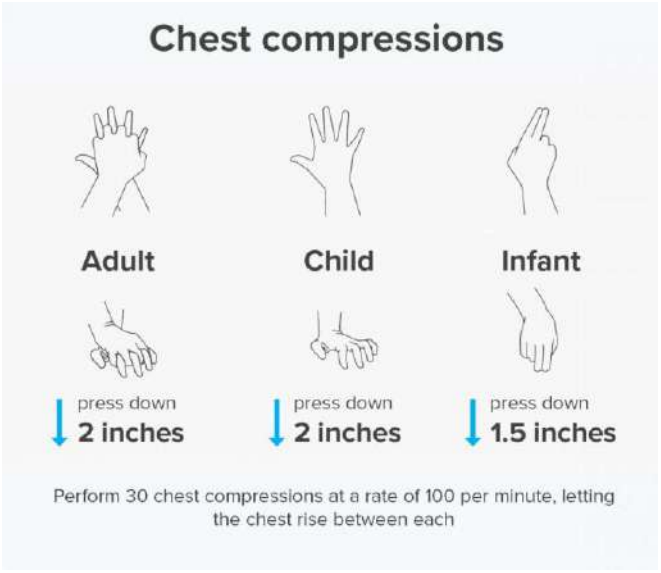
Begin cardiopulmonary resuscitation (CPR) as follows:

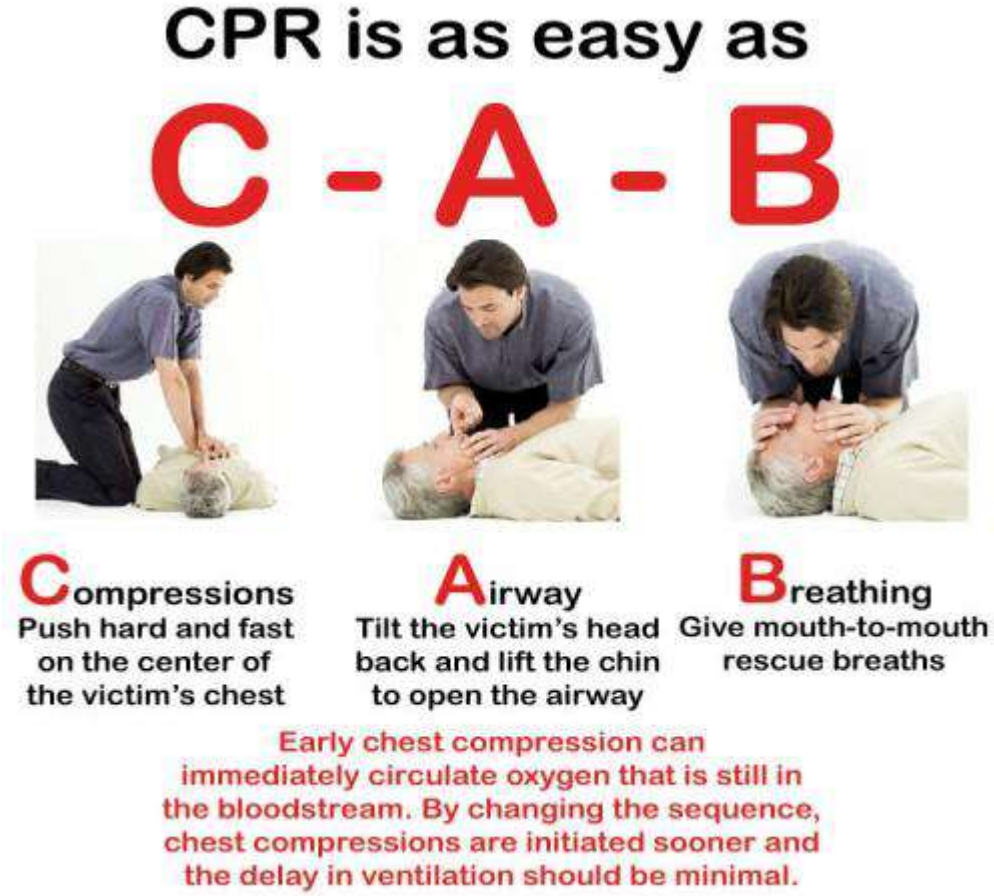
Chest compressions: Give 30 compressions to 2 breaths (30:2)in adults. CPR should not be interrupted except to give shocks. Use the heel of hand with straight elbows. Center over the lower half of the sternum. Aim for 4–5 cm compression at 100/min. Allow the chest to return to normal position. Push hard and push fast.

Airway: Open airway. If no contraindications, use head tilt, chin lift. Clear the mouth.

Breathing: Assess breathing, if inadequate, give two breaths, each inflation ~1 sec long. Use specialized bag-and-mask system (e.g., Ambo R system) if available and two resuscitators are present. Otherwise, mouth-to-mouth breathing.


Step-by-Step CPR Guide
1. Shake and shout
2. Call 911
3. Check for breathing
4. Place your hands at the center of their chest
5. Push hard and fast—about twice per second
6. If you've had training, repeat cycles of 30 chest pushes and 2 rescue breaths
well


Chest compressions
Adult
Child
Infant
press down
2 inches
press down
2 inches
press down
1.5 inches
Perform 30 chest compressions at a rate of 100 per minute, letting the chest rise between each



AED = automated external defibrillator

Heart failure: definition, classification, causes, precipitating factors, investigations, treatment.

Definition/description: Heart failure is a clinical syndrome characterized by a change in the pumping function of the heart accompanied by typical symptoms such as shortness of breath or weakness.

Classification:

LVF and *RVF* may occur independently, or together as *congestive heart failure (CHF)* . *Low-output cardiac failure:* The heart output is inadequate (e.g., ejection fraction <0.35) or is only adequate with high filling pressures.

Cause:

These include ischaemic heart disease, hypertension, valvular heart disease, arrhythmias, pulmonary embolism, anaemia, thyrotoxicosis, myocarditis, cardiomyopathy, infective endocarditis, and thiamine deficiency, or idiopathic.

Precipitating factors

Advise patients to avoid exposure to cardiotoxic substances such as alcohol, tobacco, and illicit drugs, particularly cocaine.

Symptoms:

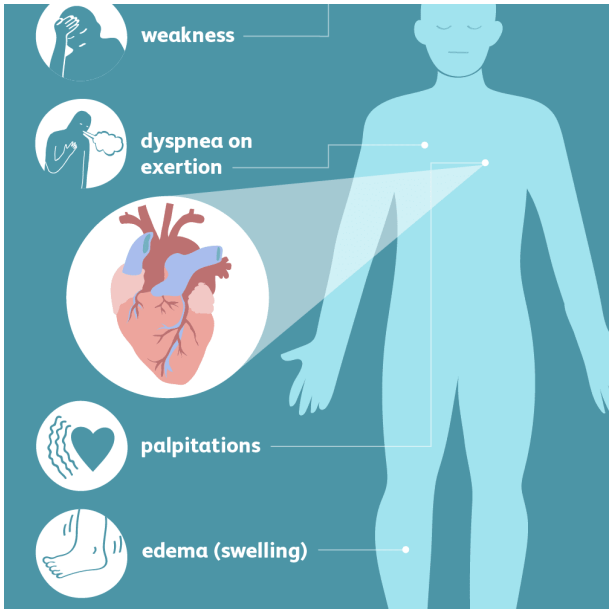
depend on which ventricle is more affected.

LVF :

Dyspnea, poor exercise tolerance, fatigue, orthopnea, paroxysmal nocturnal dyspnea (PND), nocturnal cough (pink frothy sputum), wheeze (cardiac asthma), nocturia, cool peripheries, weight loss, muscle wasting.

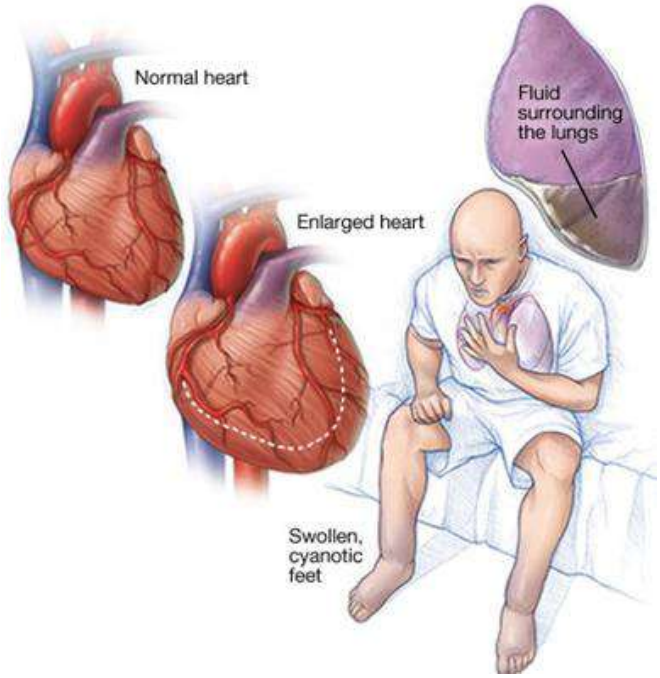
RVF :

Peripheral edema (up to thighs, sacrum, abdominal wall), abdominal distension (ascites), nausea, anorexia, facial engorgement, pulsation in neck and face (tricuspid regurgitation), epistaxis. In addition, patients may be depressed or complain of drug-related side effects.



Signs:

The patient may look ill and exhausted, with cool peripheries and peripheral cyanosis.



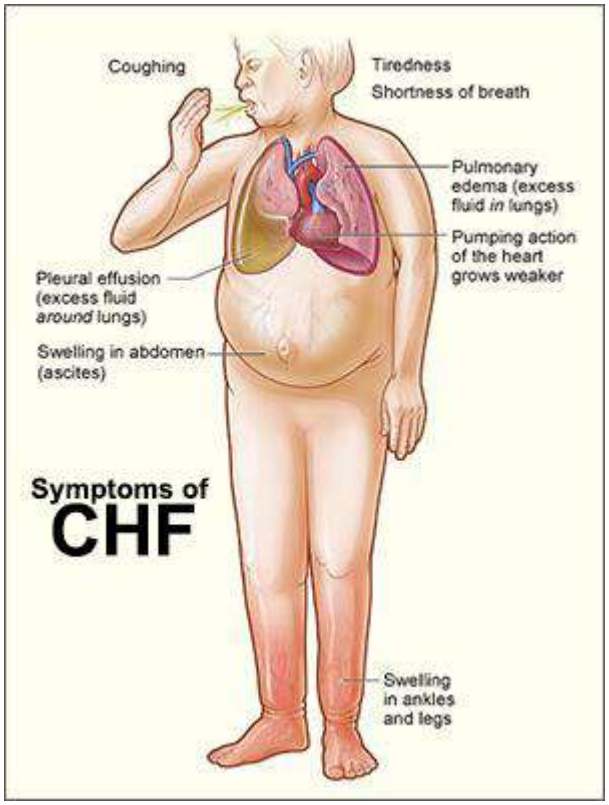
Pulse : Resting tachycardia, pulses alternant. Systolic BP ↓ , narrow pulse pressure, JVP ↑ .

Precordium : Displaced apex (LV dilatation), RV heave (pulmonary hypertension).

Auscultation : S_3 gallop, murmurs of mitral or aortic valve disease.

Chest : Tachypnea, bibasal end-inspiratory crackles, wheeze ("cardiac asthma"), pleural effusions.

Abdomen : Hepatomegaly (pulsatile in tricuspid regurgitation), ascites, peripheral edema.

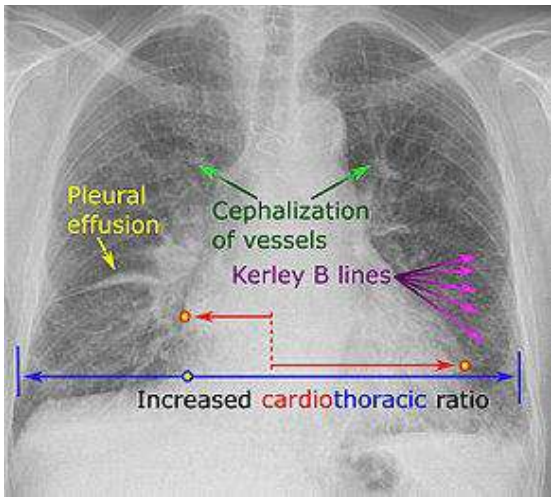


Investigations

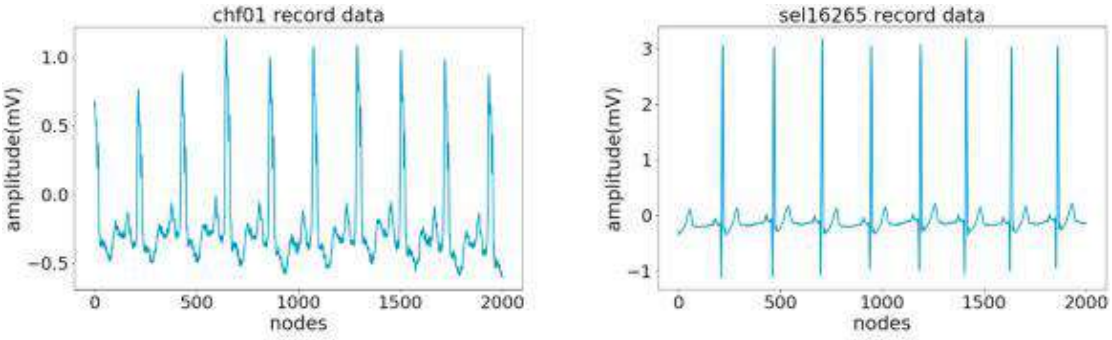
Chest x-ray, ECG, echocardiography, and radionuclide imaging are used in the diagnosis of heart failure. Blood tests such as a complete blood count, blood creatinine, blood glucose, albumin, liver function tests, and thyroid function tests are also necessary.

Blood tests: CBC, electrolytes, TSH, iron studies.

***CXR* :** Cardiomegaly (cardiothoracic ratio >50%), prominent upper lobe veins (upper lobe diversion).

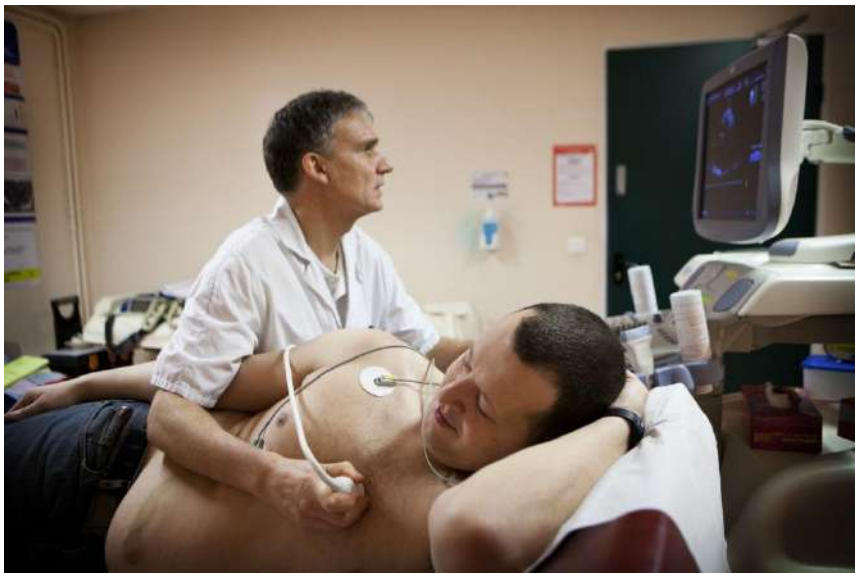


ECG: may indicate cause (look for evidence of ischemia, MI, or ventricular hypertrophy). It is rare to get a completely normal ECG in chronic heart failure.



(a) Congestive Heart Failure (b) Normal Sinus Rhythm

Echocardiography is the key investigation. It may indicate the cause (MI, valvular heart disease) and can confirm the presence or absence of LV dysfunction.



Endomyocardial biopsy Is rarely needed but is occasionally helpful to diagnose the cause of restrictive cardiomyopathies.

Management: Management includes dietary salt restriction, diuretics, digitalis preparations (digoxin), vasodilators, β-blockers, and Angiotensin-converting enzyme (ACE) inhibitors. If thyrotoxicosis is the cause, treatment is directed to correct it.

Acute heart failure is a medical emergency.

Chronic heart failure Treat the cause (e.g., if arrhythmias; valve disease).

- Treat exacerbating factors (anemia, thyroid disease, infection, BP↑) .
- Avoid exacerbating factors, such as NSAIDs.
- Stop smoking. Eat less salt. Maintain optimal weight and nutrition.
- Drugs: The following are used:

1 *Diuretics:* Loop diuretics routinely used to relieve symptoms (e.g., furosemide; increase dose and frequency as necessary).

2 *ACE-inhibitor:* Consider in all patients with left ventricular systolic dysfunction, (e.g., Lisinopril, Enalapril, Captopril [Capoten]).

3 *angiotensin II receptor antagonists:* **(**Valsartan 40-160 mg)

4 *B-blockers* (e.g., carvedilol, metoprolol).

5 *Aldosterone antagonists:* Spironolactone is K^+ -sparing, and hyperkalemia should be monitored closely in the elderly or those with renal insufficiency.

6 *Digoxin* improves symptoms.

7 *Vasodilators:* Long-acting nitrates reduce preload by causing venodilatation (e.g., isosorbide mononitrate 60 mg/24 h PO). Second-line agents include arterial vasodilators, which reduce afterload (e.g., hydralazine).

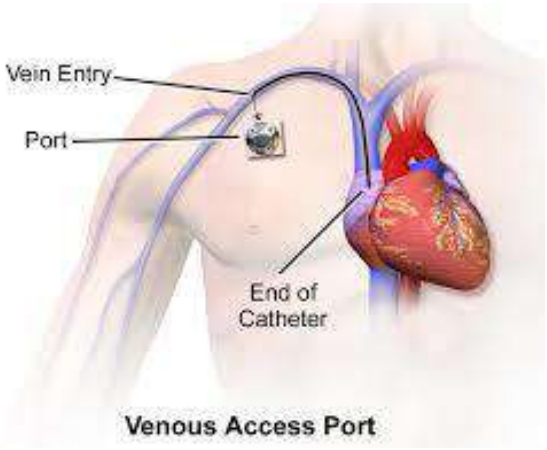
محاضرة 7

المحاضرة (7) عناية عملي

د. علاء غانم الكواز

Central Venous Line

Central venous line or catheter is a special IV. Line that is inserted into a large vein in the body.



Several veins are used for central venous catheter including those located in the shoulder (Subclavian vein), neck (Jugular vein) and groin (Femoral vein).

The most common method is seldingers technique (catheter over guide wire).

Indications

1- Monitoring of CVP (Central Venous Pressure), (normal CVP is 8-12 CmH_2O).

2- Patient does not have adequate veins in the arm.

3- Need for special medications (e.g. Chemotherapy).

4- Parenteral nutrition.

5- CVP serve as guide for fluid administration in critically ill patient.

6- Frequent blood samples for blood tests.

7- Determine the function of right side of the heart.

Complications

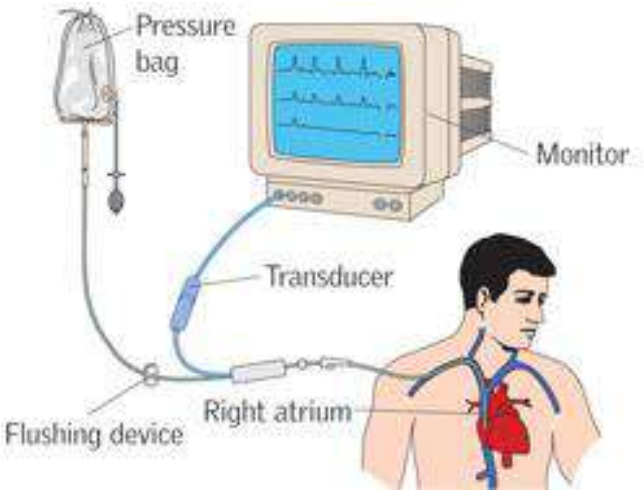
1- Bleeding and hematoma.

2- Air embolism.

3- Infection.

4- Pneumothorax (air in the chest) and heamothorax (blood in the chest), which need chest tube to re-expand the lung.

5- Cardiac arrythmia.



You should always assess for patency of the CVP line and sterile dressing should be done to prevent infection.

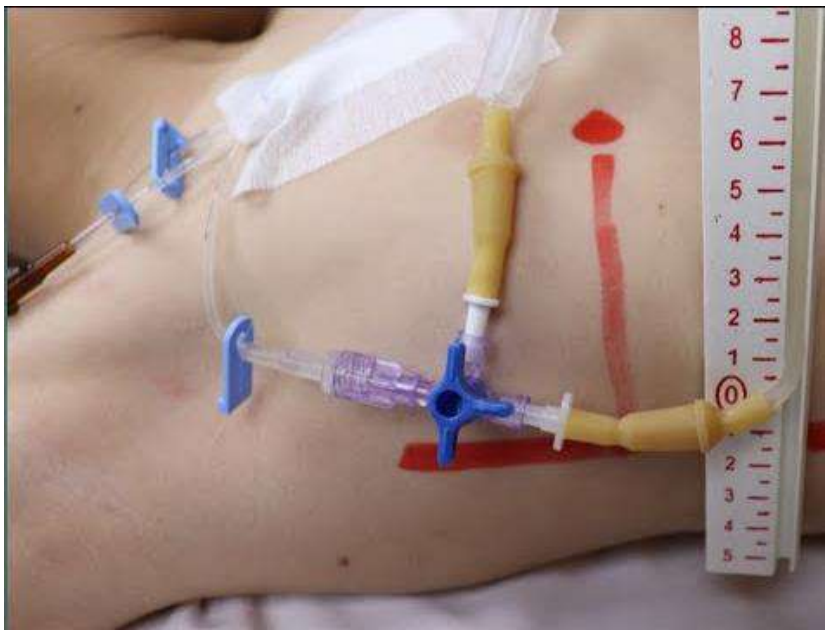
Contraindications

1- Uncooperative patient.

2- Bleeding tendency.

3- Local skin infection.

4- Pneumothorax or heamothorax on the other side.



محاضرة ٨ عناية عملي

د. علاء غانم الكواز

Cardiopulmonary resuscitation

Definition of Cardiopulmonary Resuscitation (CRP)

Technique of basic life support to oxygenate heart, lungs and brain until medical treatment arrived and restore normal cardiopulmonary function.

Cardiac arrest: Loss of cardiac function, breathing and loss of consciousness.

Aim of CPR

1- To prevent death.
2- To prevent irreversible brain damage.
3- To restore normal cardiac and pulmonary function.

Reversible Causes of cardiac arrest (5Ts and 5Hs).

5Ts:

- Tamponed (cardiac tamponed)
- Tension pneumothorax
- Thrombosis (pulmonary thrombosis)
- Thrombosis (coronary).
- Toxicity (drug poisoning and over dosage)

5Hs:

- Hypoxia.
- Hypotension (hypovolemia & shock).
- Hypothermia.

- Hypoglycemia.
- Hypo and hyperkalemia

Other causes of cardiac arrest

- Electrical shock
- Congenital heart disease
- Asphyxia
- Co. poisoning
- Aortic aneurysm rupture
- Pulmonary edema
- Brain injury and massive CVA

Causes of Respiratory arrest

- Foreign body in throat
- Suffocation
- Drowning
- Smoke inhalation
- Stroke and coma
- Accidents

Diagnosis of cardiac arrest

Warning signs:

1- Early :

A loss of consciousness and convulsion.

2- Late :

- No pulsation in carotid and femoral arteries & absence of heart sounds.
- No breathing
- Dilated pupils

3- Other:

- Cyanosis
- Hypothermia

Phases of cardio pulmonary resuscitation

1-phase I	BLS (basic life support)	C= Compression A= Airway B=Breathing
2- phase II	ALS (Advanced life support)	C- A- B and E= ECG D= Defibrillation D= Drugs
3- Phase III	Prolonged support	Post resuscitation

Basic life support (BLS)

It is life support technique without using of special equipments.

While advanced life support (ALS) is life support technique with use of special equipment (oropharyngeal airways ,endotracheal tube, Ambu bag , defibrillator and drugs).

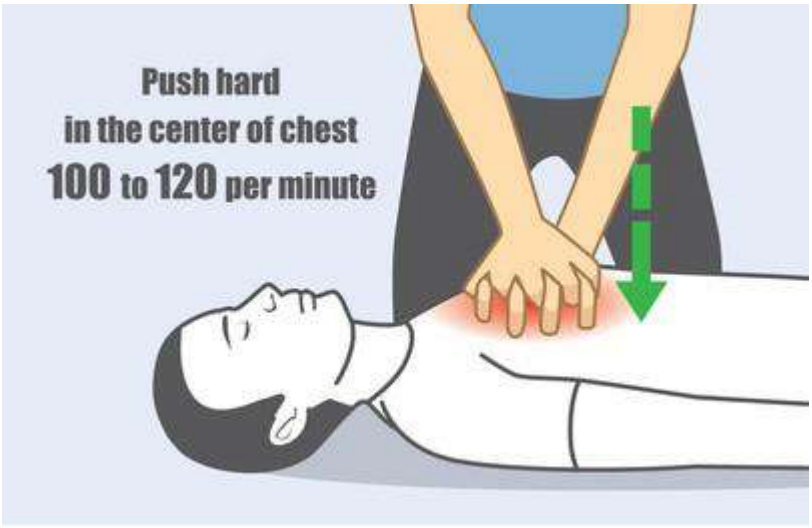
Basic life support consists of:

1- Early recognition of cardiac arrest
2- Call for help
3- Cardiopulmonary resuscitation
4- Relief airway obstruction
5- Defibrillation (automated external defibrillator AED)

- The early recognition of cardiac arrest is done by assessment of victim condition by (Look, Hear, and Listen).
  - 
- Check for response by shaking shoulder and ask (are you all right)
- If there is no breathing or only gasping breathing, no pulse is felt on carotid and femoral arteries, start CPR immediately as C-A-B (compression- airway- Breathing) sequence.
- If chest compression started within 3-5 minuet and defibrillation within 10 minutes, there is a survive chance of 50% for cardiac arrest victims.
- Assessment of patient condition should take not more then 5-10 sec.

Chest compression

- Place heal of one hand in center of the chest at the lower half of sternum.
- The other hand is places over the first hand with interlock fingers.
- The elbows are straight, locked and arms extended.
- Push down at least 4-5 cm in adult (2-4 cm in children 1-2 cm in infants)
- Push at rate of 100-120 per minuet
- Ratio of compression to ventilation is 30:2 if two rescues, chest compression must be not interrupted by ventilation
- When possible change operator every 2 minuet (because of exhaustion)
- In pediatric victims (less than 8 years old) chest compression is done by two fingers with rate of 100-120 per minute, the compression ventilation ration is 15:2.



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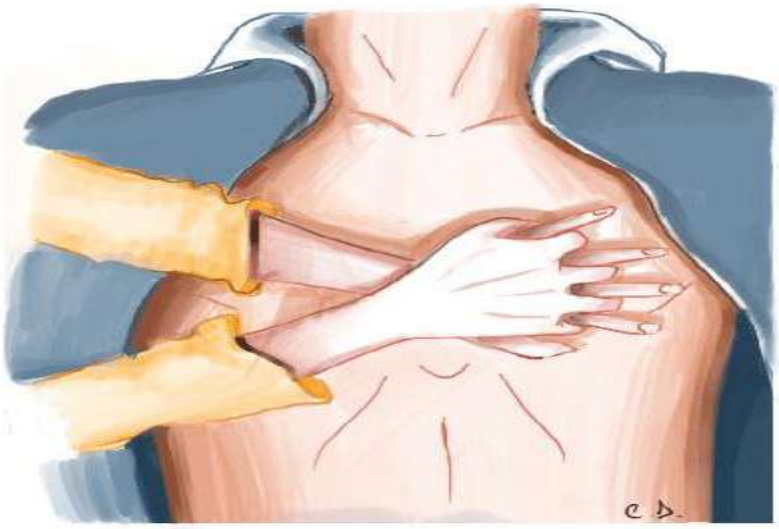
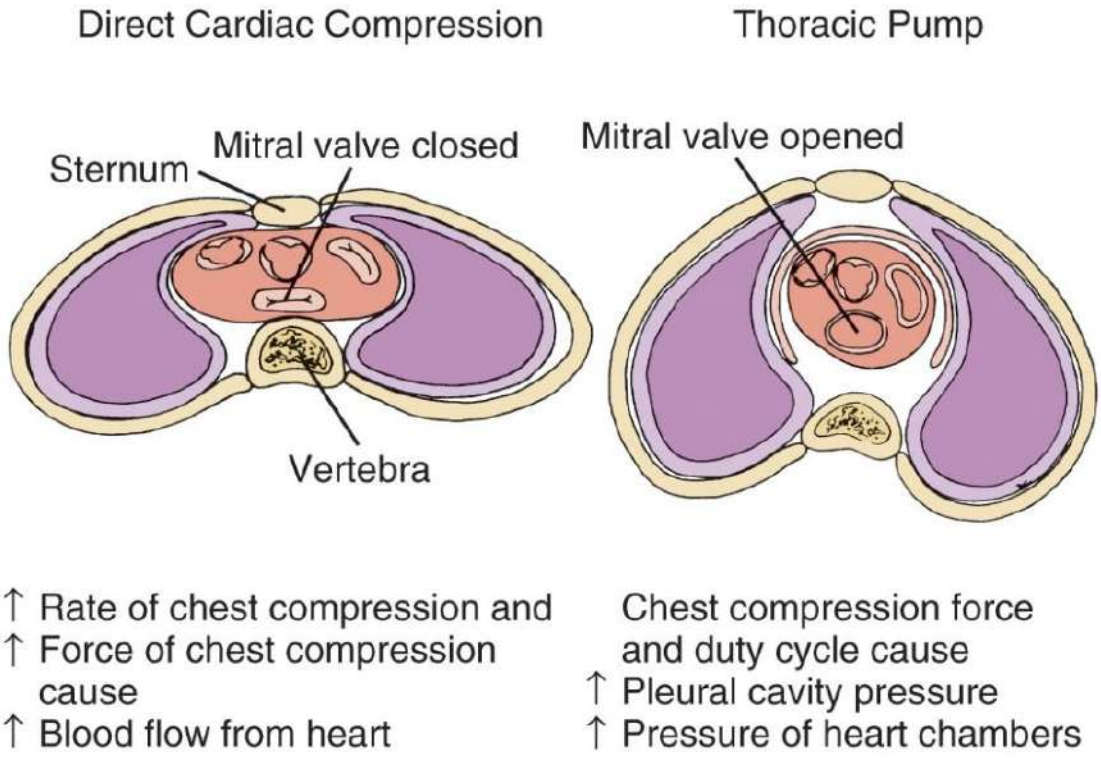


The mechanism by which chest compression force the blood to flow is

1. Direct compressing on the heart (cardiac pump).
2. Increase of intratharcic pressure (thoracic pump).



Each 5 cycles of compression (30:2) take about 2 minutes, chest compression must be continued for 5 cycles (2 minutes) before assessment of cardiac rhythm, if it is shockable (VF-VT) or not (asystole/PEA).

Causes of failure of chest compression.

1- Inadequate effort

2- Over effort : lead to injury to ribs, cardiac muscle, lungs and liver

3- Wrong hand position

- Too low lead to gastric compression, vomiting and aspiration
- Too high lead to the heart is not compressed

4- Failure to release between compression which prevent filling of the heart lead to inefficient CPR

Complications of CPR

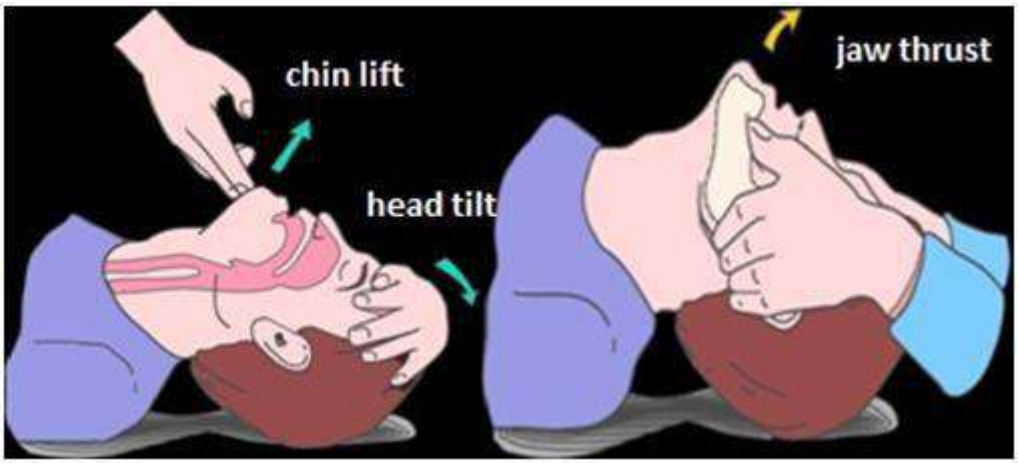
- Fractured sternum
- Laceration of the chest
- Injury to diaphragm
- Burn
- Injury to lungs and liver
- Heamothorax and pneumothorax
- Pneumopericardium
- Injury to coronary vessels
- Interference with ventilation
- Failure to return heart rhythm to normal

محاضرة ٩ عناية عملي

د. علاء غانم الكواز

Airway relief

- Clear the airway
- Do head tilt (tilt forehead back ward and chin upward)
- Do jaw thrust if you suspect injury to the neck (cervical vertebrae)
- These techniques should relief airway obstruction due to loss of airway protecting reflexes and loss of muscle tone in addition to falling of the tongue backward



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In case of suffocation due to foreign body inhalation, do not remove foreign body by figure, instead do (Heimlich maneuverer) which include sub diaphragmatic abdominal thrust that elevating the diaphragm and expelling air from lungs that displace the foreign body and take it out.

Breathing:

- Pinch the nose.
- Take a deep breath.
- Do mouth to mouth ventilation
- Blow and ensure chest rise with each breath
- Allow chest to collapse completely.
- Repeat

In CPR this can be done 2 times every 30 compressions, you can do mouth to nose ventilation when mouth is seriously injured or cannot be opened, mouth to (mouth and nose) ventilation can be done in infants and small children .

When to terminate basic life support

1- Emergency medical service was arrived.
2- Physical fatigue (when rescue was exhausted).
3- Injuries not compatible with life (eg. multiple amputation with massive bleeding, blast injuries).
4- ROSC (Return of spontaneous circulation)

Sings of ROSC:

- Patient moving.
- Coughing & swallowing.
- Returned breathing.
- Improve color of skin.
- Contraction of pupils.
- Palpable pulse and measurable BP.

Advanced cardiac life support ACLS

It is similar to BLS but using special equipment:

1- Airway equipment.
2- O_2 source.
3- Suction apparatus & nasogastric tube.
4- Oximeter.
5- IV cannula.
6- Cardiac monitor
7- Emergency drugs
8- Defibrillator

ACLS consist of:

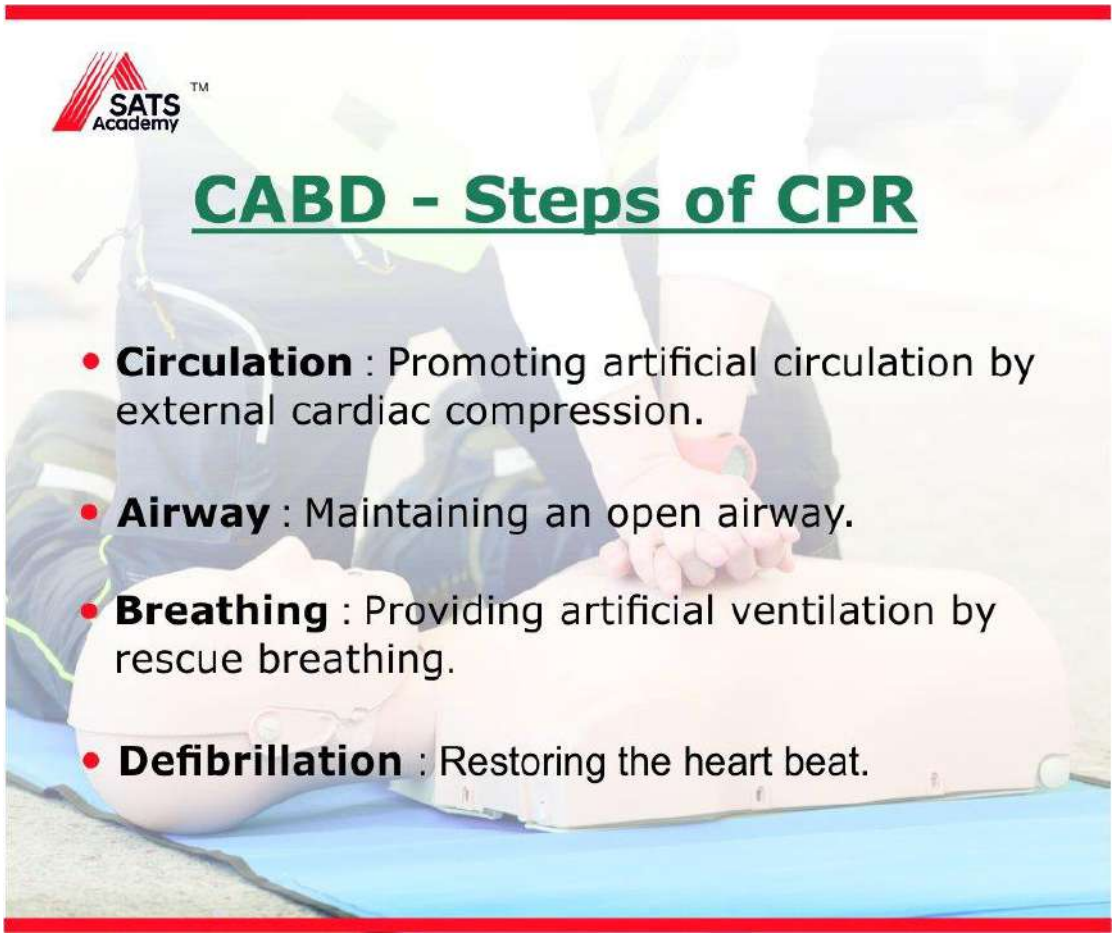
1- Chest compression.

2- Airway management by special equipments.

3- Breathing by advanced techniques.

4- Defibrillation.

5- Drugs.



1. Chest compression.

Chest compression: is same as in BLS.

Position Hands Over Sternum

2. Airway management by following.

- Orpharyngeal airway.
- Face masks.
- LMA (laryngeal mask airway).
- Endotracheal tube ETT.
- Ambu bag.
- O_2 source.
- Airway equipment.
- Suction apparatus.
- Nasogastric tube.
- Tracheostomy and cricothyrtomy.

3. Breathing:

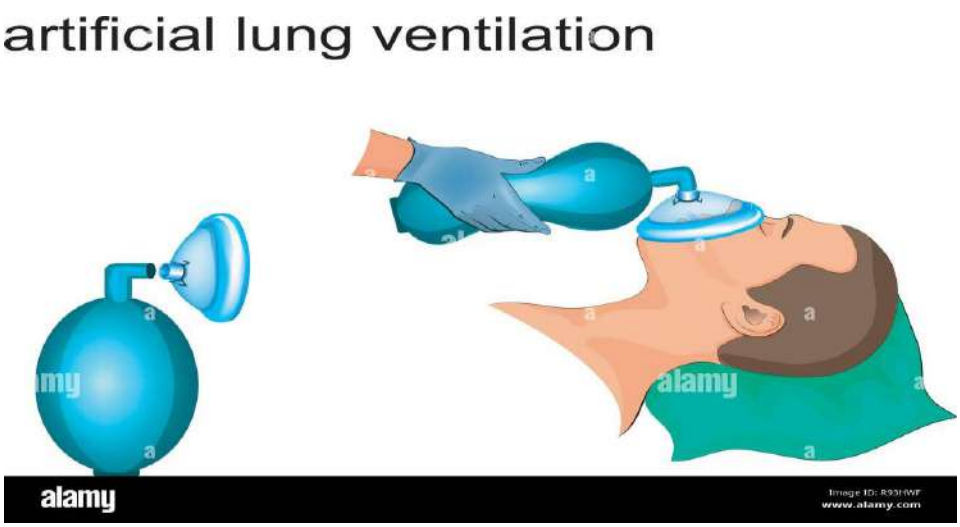
Give 100% oxygen it possible.

The expired air (mouth to mouth) give 16% oxygen while room air by Ambu bag Gives 21% O_2 , if Ambu bag is attached to O_2 source it will give, 45% O_2.

If Ambu bag with O_2 reservoir bag, this will give 85% O_2.

Advanced breathing techniques include:

1- Ambu bag.
2- Oxygen source.
3- Mechanical ventilator.
4- In the presence & artificial airway (eg ETT), the ventilation rate should be 10-12 breath/min and should not interrupt the chest compression.

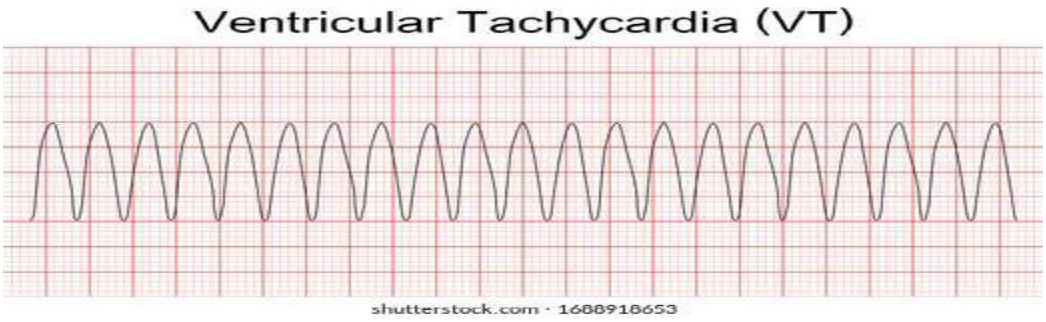


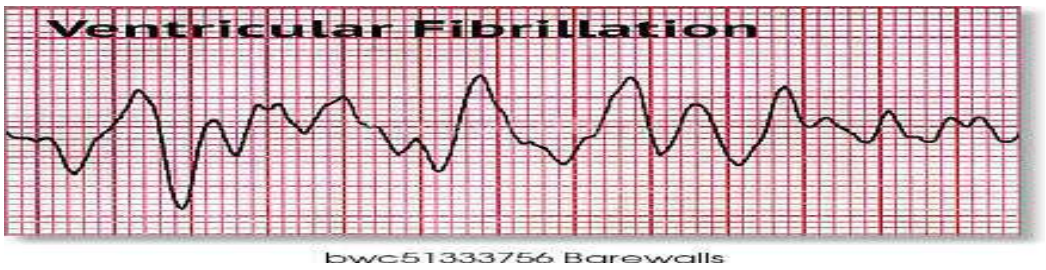
4. Defibrillation.

Defibrillator: is a device that delivers direct electrical current (DC Shock) across the myocardium to produce spontaneous depolarization of cardiac muscles.

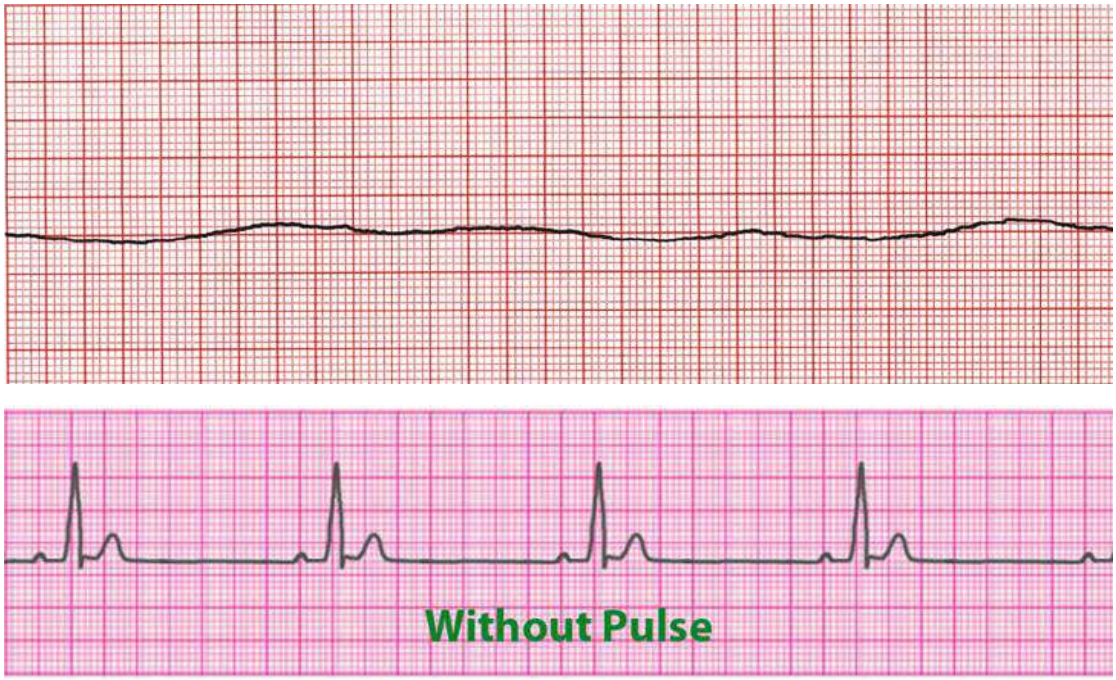
- In cardiac arrest, cardiac rhythms can be classified into two types:

1-Shockable rhythm (VT/VF): ventricular tachycardia and ventricular fibrillation.





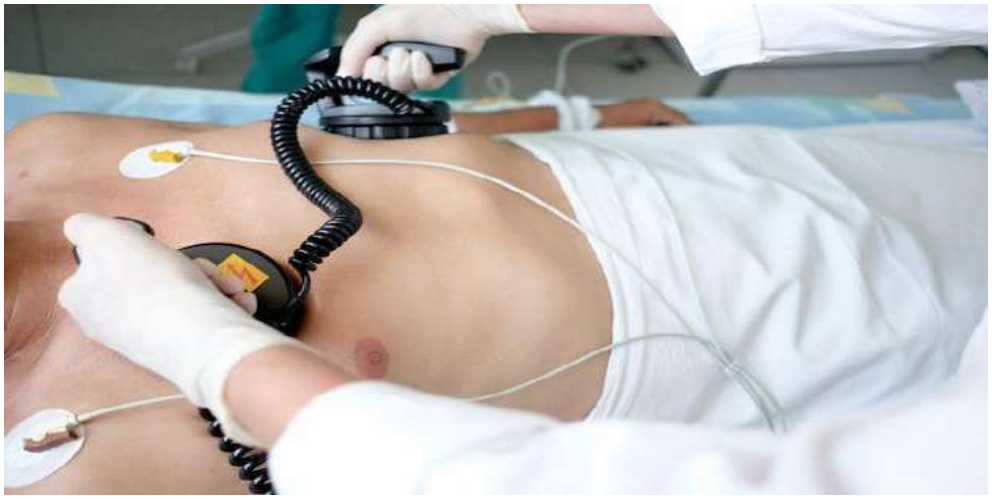
2-Non-shockable rhythm: asystole and pulseless electrical activity (PEA).



The difference between these two types of arrhythmia is that VF and VT need defibrillation.

Procedure of defibrillation.

- The first paddle is placed in right infraclavicular region.
- The other paddle is placed at the apex of the heart (left 5^{th} intercostal space at anterior axillary line).
- Voltage delivered is between 150-360 Joule.In pediatric 2-4 Joule/K



5. Drugs used in CPR.

1- Adrenalin l mg every 3-5 min.

2- Vasopressin 40 unit as alterative to adrenalin used once only.

3- Amidarone initially 300 mg then 150 mg used for VF/VT.

4- Lidocaine 1-2 mg/kg as alternative to amidarone or when it is not available.

5- Atropine 1mg i.v, full atropinisartion done with dose of 3 mg used for sever bradycardi.

6- Calcium chloride 10%.

Is given in case of hypocalcaemia or hyperkalemia

7- Sodium bicarbonate: is given only in sever acidosis with hyperkalemia pH.(7.1)

- If there is no I.V line, interosseous line can be used.
- Drugs can be also given through ETT (except sodium bicarbonate) in a dose of 2-2.5 the I.V dose.
- Intravenous fluid give N.S or Ringer lactate.
- GW given only in documented hypoglycemia

Post cardiac arrest management.

Aim:

1- To minimize the effect of loss of circulation on vital organs and improve neurological function.
2- To recognize, prevent and treat recurrent cardiac arrest.
3- To ensure hemodynamic monitoring.
4- To identify and treatment of precipitating factors.

Post arrest management includes.

1- Transfer patient to ICU.
2- Monitor closely and continuously.
3- Monitor vital signs every hour.
4- Watch for convulsion.
5- Intubate if necessary.
6- Catheterize the patient, monitor results and record the procedure.

Respiratory system management.

1- Intubation and mechanical ventilation until patient become stable.
2- Obtain chest X-Ray.
3- Administer drugs.
4- Avoid hyperventilation.

CVS management

1- Obtain expert consultation.
2- Monitor and laboratory analysis.
3- Intra-arterial BP monitoring.
4- Drugs.

محاضرة ١١ عناية نضري

د. علاء الكواز

Cardiopulmonary resuscitation

Definition of Cardiopulmonary Resuscitation (CRP)

Technique of basic life support to oxygenate heart, lungs and brain until medical treatment arrived and restore normal cardiopulmonary function.

Cardiac arrest: Loss of cardiac function, breathing and loss of consciousness.

Aim of CPR

1- To prevent death.
2- To prevent irreversible brain damage.
3- To restore normal cardiac and pulmonary function.

Reversible Causes of cardiac arrest (5Ts and 5Hs).

5Ts:

- Tamponed (cardiac tamponed)
- Tension pneumothorax
- Thrombosis (pulmonary thrombosis)
- Thrombosis (coronary).
- Toxicity (drug poisoning and over dosage)

5Hs:

- Hypoxia.
- Hypotension (hypovolemia & shock).
- Hypothermia.

- Hypoglycemia.
- Hypo and hyperkalemia

Other causes of cardiac arrest

- Electrical shock
- Congenital heart disease
- Asphyxia
- Co. poisoning
- Aortic aneurysm rupture
- Pulmonary edema
- Brain injury and massive CVA

Causes of Respiratory arrest

- Foreign body in throat
- Suffocation
- Drowning
- Smoke inhalation
- Stroke and coma
- Accidents

Diagnosis of cardiac arrest

Warning signs:

1- Early :

A loss of consciousness and convulsion.

2- Late :

- No pulsation in carotid and femoral arteries & absence of heart sounds.
- No breathing
- Dilated pupils

3- Other:

- Cyanosis
- Hypothermia

Phases of cardio pulmonary resuscitation

1-phase I	BLS (basic life support)	C= Compression A= Airway B=Breathing
2- phase II	ALS (Advanced life support)	C- A- B and E= ECG D= Defibrillation D= Drugs
3- Phase III	Prolonged support	Post resuscitation

Basic life support (BLS)

It is life support technique without using of special equipments.

While advanced life support (ALS) is life support technique with use of special equipment (oropharyngeal airways endotracheal tube, Ambu bag , defibrillator and drugs).

Basic life support consists of:

1- Early recognition of cardiac arrest
2- Call for help
3- Cardiopulmonary resuscitation
4- Relief airway obstruction
5- Defibrillation (automated external defibrillator AED)

- The early recognition of cardiac arrest is done by assessment of victim condition by (Look, Hear, and Listen).
- Check for response by shaking shoulder and ask (are you all right)
- If there is no breathing or only gasping breathing, no pulse is felt on carotid and femoral arteries, start CPR immediately as C-A-B (compression- airway- Breathing) sequence.
- If chest compression started within 3-5 minuet and defibrillation within 10 minutes, there is a survive chance of 50% for cardiac arrest victims.

Assessment of patient condition should take not more then 5-10 sec.

Chest compression

- Place heal of one hand in center of the chest at the lower half of sternum.
- The other hand is places over the first hand with interlock fingers.
- The elbows are straight, locked and arms extended.
- Push down at least 4-5 cm in adult (2-4 cm in children 1-2 cm in infants)
- Push at rate of 100-120 per minuet
- Ratio of compression to ventilation is 30:2 if two rescues, chest compression must be not interrupted by ventilation
- When possible change operator every 2 minuet (because of exhaustion)
- In pediatric victims (less than 8 years old) chest compression is done by two fingers with rate of 100-120 per minute, the compression ventilation ration is 15:2.

The mechanism by which chest compression force the blood to flow is

1. Direct compressing on the heart (cardiac pump).
2. Increase of intratharcic pressure (thoracic pump).

Each 5 cycles of compression (30:2) take about 2 minutes, chest compression must be continued for 5 cycles (2 minutes) before assessment of cardiac rhythm, if it is shockable (VF-VT) or not (asystole/PEA).

Causes of failure of chest compression.

1- Inadequate effort

2- Over effort : lead to injury to ribs, cardiac muscle, lungs and liver

3- Wrong hand position

- Too low lead to gastric compression, vomiting and aspiration
- Too high lead to the heart is not compressed

4- Failure to release between compression which prevent filling of the heart lead to inefficient CPR

Complications of CPR

- Fractured sternum
- Laceration of the chest
- Injury to diaphragm
- Burn
- Injury to lungs and liver
- Heamothorax and pneumothorax
- Pneumopericardium
- Injury to coronary vessels
- Interference with ventilation
- Failure to return heart rhythm to normal

Airway relief

- Clear the airway
- Do head tilt (tilt forehead back ward and chin upward)
- Do jaw thrust if you suspect injury to the neck (cervical vertebrae)
- These techniques should relief airway obstruction due to loss of airway protecting reflexes and loss of muscle tone in addition to falling of the tongue backward.

In case of suffocation due to foreign body inhalation, do not remove foreign body by figure, instead do (Heimlich maneuverer) which include sub diaphragmatic abdominal thrust that elevating the diaphragm and expelling air from lungs that displace the foreign body and take it out.

Breathing:

- Pinch the nose.
- Take a deep breath.
- Do mouth to mouth ventilation
- Blow and ensure chest rise with each breath
- Allow chest to collapse completely.
- Repeat

In CPR this can be done 2 times every 30 compressions, you can do mouth to nose ventilation when mouth is seriously injured or cannot be opened, mouth to (mouth and nose) ventilation can be done in infants and small children .

When to terminate basic life support

1- Emergency medical service was arrived.
2- Physical fatigue (when rescue was exhausted).
3- Injuries not compatible with life (eg. multiple amputation with massive bleeding, blast injuries).
4- ROSC (Return of spontaneous circulation)

Sings of ROSC:

- Patient moving.
- Coughing & swallowing.
- Returned breathing.
- Improve color of skin.
- Contraction of pupils.
- Palpable pulse and measurable BP.

Advanced cardiac life support ACLS

It is similar to BLS but using special equipment:

1- Airway equipment.
2- O_2 source.
3- Suction apparatus & nasogastric tube.
4- Oximeter.
5- IV cannula.
6- Cardiac monitor
7- Emergency drugs
8- Defibrillator

ACLS consist of:

1- Chest compression.

2- Airway management by special equipments.

3- Breathing by advanced techniques.

4- Defibrillation.

5- Drugs.

1. Chest compression.

Chost compression: is same as in BLS.

2. Airway management by following.

- Orpharyngeal airway.
- Face masks.
- LMA (laryngeal mask airway).
- Endotracheal tube ETT.
- Ambu bag.
- O_2 source.
- Airway equipment.
- Suction apparatus.
- Nasogastric tube.
- Tracheostomy and cricothyrtomy.

3. Breathing:

Give 100% oxygen it possible.

The expired air (mouth to mouth) give 16% oxygen while room air by Ambu bag Gives 21% O_2 , if Ambu bag is attached to O_2 source it will give, 45% O_2.

If Ambu bag with O_2 reservoir bag, this will give 85% O_2.

Advanced breathing techniques include:

1- Ambu bag.
2- Oxygen source.
3- Mechanical ventilator.
4- In the presence & artificial airway (eg ETT), the ventilation rate should be 10-12 breath/min and should not interrupt the chest compression.

4. Defibrillation.

Defibrillator: is a device that delivers direct electrical current (DC Shock) across the myocardium to produce spontaneous depolarization of cardiac muscles.

- In cardiac arrest, cardiac rhythms can be classified into two types:

1- Shockable rhythm (VT/VF): ventricular tachycardia and ventricular fibrillation.
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The difference between these two types of arrhythmia is that VF and VT need defibrillation.

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Hypoxia and Oxygen therapy

محاضرة (١٣)

عناية مركزة نظري

د. علاء غانم الكواز

Definition of hypoxia:

It is the condition in which the tissue has low level of oxygen, when the oxygen is entirely absent it is called anoxia.

- Hypoxia: low level of oxygen at tissue level.
- Anoxia: no oxygen at tissue level.
- Hypoxemia: low level of oxygen in blood.

Cyanosis

It means blueness of the skin which caused by excessive amount of deoxygenated Hb in the skin blood vessels, especially in the capillaries.

The deoxygenated blood has an intense dark (blue-purple) color that is transmitted through the skin.

- Clinically, definite cyanosis appears only when the arterial blood contains more than 5 gm/dL of deoxygenated Hb, so in severe anemia (Less than 5 gm/dL) the cyanosis does not appear.

Types of Hypoxia

1- Hypoxic hypoxia

2- Anemic hypoxia

3- Stagnant hypoxia

4- Histotoxic hypoxia

Hypoxic hypoxia:

In which the oxygen in the blood going to the tissue is too low to saturate the Hb.

Causes

1. Decrease FiO_2 (decrease O_2 in inspired air) as in high altitude or breathing low concentration O_2 mixture in closed area.
2. Decrease pulmonary ventilation: as in respiratory muscles paralysis or disease, Respiratory Centre depression, sleep apnea, airway obstruction or suffocation.
3. Defect in gas exchange and O_2 transfer as in pulmonary fibrosis, pulmonary edema and pneumothorax.

Anemic hypoxia:

In which the amount of functional Hb is too small, so the capacity of blood to carry O_2 is too small.

Causes

1. Severe anemia
2. Hemorrhage and shock
3. Abnormal Hb: eg. met Hb (iron in ferric instead of ferrous form), sickle Hb (Hbs) and carboxy Hb (CoHb)…etc.

Stagnant hypoxia

In which the blood is normal but the flow of blood to the tissue is reduced or stopped.

Cause

1. Congestive heart failure CHF
2. Shock
3. Venous or arterial obstruction

4. Circulatory failure and poor tissue perfusion of organs (eg: pulmonary thrombosis and embolism

Histotoxic hypoxia:

In which the cells of tissue are poisoned and are unable to use the oxygen.

Causes

Cyanide poisoning, in histotoxic hypoxia oxygen therapy is not useful.

Oxygen therapy

Definition: is the administration of oxygen at a concentration greater than that present in the environmental atmosphere.

The air in the atmosphere is containing about 21% oxygen.

Aim of O_2 therapy

O_2 therapy is used to increase oxygen saturation in tissue where the saturation level is too low due to illness or disease.

There are three clinical goals to O_2 therapy:

1- Treatment of hypoxemia.

2- Decrease work of breathing.

3- Decrease myocardial work.

Signs and symptoms of hypoxia

1- Dyspnea (shortness of breath)

2- Tachypnea (rapid respiratory rate)

3- Restlessness and irritability

4- High blood pressure and pulse rate

5- Headache

6- Chest pain

7- Confusion and loss of consciousness

8- Convulsion

Indications of O_2 therapy

1. Documented hypoxemia arterial tension is < 60 mmHg or O_2 saturation is < 90% on room air.
2. Acute respiratory failure and sever respiration disease.
3. Acute myocardial infarction and cardiac failure.
4. Shock.
5. Increase metabolic demand by burns, sepsis and sever trauma.
6. Short term therapy during anesthesia and recovery.
7. Carbon monoxide poisoning (CO).
8. Severe anemia.
9. During CPR.

Oxygen therapy devices

Classified into two types:

A- Low Flow Oxygen Delivery Devices (Variable performance):

Do not provide constant FiO_2 and it is patient dependent, that is the oxygen in the inspired air (FiO_2) well depend on patient's minuet ventilation, and part of the tidal volume will provide by room air.

1- Nasal canula (prongs)

2- Simple face mask

3- Partial rebreathing mask

4- Non rebreathing mask

B- High Flow Oxygen Delivery Devices (Fixed performance devices):

It is patient independent and provides specific and constant percent of oxygen regardless of patient minuet ventilation, and they deliver a gas mixture at high flow that exceed the patient demand.

1- Venturi Mask
2- Oxygen hood and Oxygen tent
3- AMBU bag
4- Tracheostomy collar
5- T-tube
6- Oxygen concentrator

O_2 therapy rules

O_2 prescription should include:

1- Delivery device
2- Flow rate
3- Duration
4- Instruction for monitoring

Nasal canula (prongs)

It is disposable, plastic device which contains two prongs for insertion into the nostrils and connected to oxygen source.

It can give (24 - 44%) oxygen concentration at flow rate of (1 – 6 L/min) respectively.

Advantages

1- Inexpensive and easily to use.
2- Patient able to talk and eat easily with oxygen in place.
3- Safe and simple.
4- Well tolerated and comfortable
5- Deliver low concentration, so can be used for long term period therapy (eg: in COPD).

Disadvantages

1- Unable to use with nasal obstruction or epistaxis, not good for mouth breathers.

2- Need correct placement and can be dislodged from naris easily.

3- Dryness of mucous membrane at flow rate more than 4 L/min.

4- Cause skin irritation or injury at naris or over ears.

5- 80% of O_2 gets wasted during expiration.

Face Masks

1- Simple face mask

2- Partial re-breather mask

3- Non re-breather mask

4- Venturi mask

The simple face mask

It is disposable device, made of clear plastic that can be molded to fit the face, it can cover both nose and mouth, it is held to the head by elastic bands and some have a metal clip that can be bend over the bridge of the nose for comfortable fitness.

The oxygen flow must be at sufficient rate (usually 5 L/min. or more) to prevent rebreathing.

Usually used for O_2 therapy for short period (less than 12 hr) eg. post-operative recovery room and transportation of the patient.

The simple O_2 mask delivers (35 – 60%) oxygen, at flow rate of (6 – 10) L/min., it has vents on its sides which allow room air to leak-in at many places, so diluting the delivered oxygen.

Advantages:

Light wight and can provide FiO_2 up to 60% for short term therapy.

Disadvantages:

1- Difficult to keep mask in position over nose and mouth.

2- Need to remove when speak, eat, drink or vomit.

3- Uncomfortable with possibility of fascial skin redness and trauma.

4- Dryness and irritation of eyes.

5- Lack of seal proof borders so pure O_2 gets mixed with air.

Partial re-breather mask

It is a simple mask with reservoir bag that must remain inflated during both inspiration and expiration,

The 1 liter size reservoir bag collect part of the patients expired air.

The re-breather mask can provide oxygen concentration up to 70% , but the flow rate must be maintained at 6 L/min or more to ensure that the patient does not re-breath large amount of exhaled air.

The mask can give 40 – 70% O_2 at flow rate of 6-10 L/min

Advantages:

1- Deliver high concentration of Oxygen.

2- Patient can inhale room air through mask openings if oxygen supply is briefly interrupted.

Disadvantages:

1- Require tight seal.

2- Unable of eating or talking while the mask in the place.

3- Uncomfortable and heavy with difficult to keep in place.

4- Can produce Resp. depression in COPD patient.

The non-rebreather mask

It is similar to partial rebreather mask but characterized by presence of one-way valve, when the patient exhales air, the valve closes and all the expired air is deposited into the atmosphere, not the reservoir bag.

In this way the patient is not rebreathing any of the expired gas, the bag well act as oxygen reservoir.

This mask provides the highest concentration of oxygen (up to 100%) at a flow rate of (6-15) L/min.

Advantages

1- Delivers the highest possible oxygen concentration (60-100%).

2- Exhaled O_2 from anatomic dead space is conserved.

3- Suitable for patient breathing spontaneously with sever hypoxemia.

Disadvantages

1- Insufficient flow rate may lead to rebreathing of CO_2.

2- Lack of good fascial seal system can affect O_2 concentration.

3- Drying and irritation of the eyes.

4- Impractical for long term therapy.

5- Expensive and uncomfortable to the patient.

6- Can produce respiratory depression in COPD patient who depend on hypoxic respiratory drive.

Venturin mask

It is high flow oxygen delivery device, it can deliver O_2 concentration of 28-60 % at flow rate of 4-15 L/min, the mask is constructed so that there is constant flow of air blend with fixed concentration of oxygen.

It is used for those who require constant high FiO_2 level and for patient with variable ventilation and breathing pattern also for severely dyspneic and hypoxemic patients.

It is designed with wide-bore tubing and various color-code is correspond to a precise oxygen concentration and specific air flow eg.

- Orange color gives O_2 31% at 6 L/min flow
- Yellow color gives 35% O_2 at 8 L/min flow
- Red color gives 40% O_2 at 10 L/min flow
- Green color gives 60% O_2 at 15 L/min flow

Advantages

1- Deliver most precise O_2 concentration

2- Doesn't dry mucous membrane

3- Used for severely hypoxemic patient

Disadvantages

1- Uncomfortable

2- Risk and skin irritation

3- Produce respiratory depression in COPD patients with high O_2 concentration (50% or more)

Oxygen hood and Oxygen tent

An O_2 hood is used for babies who can breathe on their own but still need oxygen, it is a plastic box with warm moist oxygen inside. The hood is placed on baby's head.

The Oxygen tent is made of see-through plastic material, placed over the head and shoulders, or over the entire body to provide oxygen at a higher level than normal

AMBU BAG

(Artificial Manual Breathing Unit) or Bag Valve Mask Ventilation (BVM). It is a hand-held device commonly used to provide positive pressure ventilation to patients who are not breathing adequately.

Tracheostomy Collar / Mask

Inserted directly into trachea

- is indicated for chronic O_2 therapy
- O_2 flow rate is 8 – 10 L/min
- Provide accurate FiO_2 with good humidity, it is comfortable and efficient.

T-piece (tube)

Used on end of endotracheal tube when start weaning from ventilator, provide accurate FiO_2 and good humidity.

Oxygen Concentrator

Device which extracts O_2 from the environmental air, Deliver up to 90-95% O_2 at the flow of 3-10 L/min.

Risks and Complications of O_2 therapy

1- O_2 toxicity

Definition: is a condition resulting from the harmful effects of breathing molecular oxygen at increased concentration, it is mostly associated with long term oxygen therapy or hyperbaric oxygen therapy (O_2 therapy at pressure more than 1 atmosphere)

Factors affecting O_2 toxicity

a. Oxygen concentration (more than 60%)

b. Pressure (normobaric or hyperbaric)

c. Time of exposure

- $FiO_2 >$ 60% longer than 24 hr
- or FiO_2 80-100% longer than 12 hr
- Long term O_2 therapy can produce pulmonary toxicity, ocular toxicity and CNS toxicity.

2- Pulmonary damage (toxicity)

High PaO_2 can damage capillary endothelium causing interstitial edema and increase alveolar-capillary membrane permeability.

3- Hypoventilation

Depressional ventilation is seen in COPD patients who have chronic CO_2 retention and hypoxic respiratory drive (depending on hypoxia instead on hypercapnia as respiratory stimulation)

4- CNS toxicity

CNS toxicity can cause seizures, brief period of rigidity followed by convulsions a loss of consciousness, more commonly occurs during long term hyperbaric O_2 therapy.

5- Absorption atelectasis

Given pure O_2 can result in collapse of alveoli as it is absorbed quickly from alveoli leading to collapse of dependent part of the lungs.

6- Retinopathy of prematurity

Ocular toxicity usually occurs in low birth weight, premature infants which cause fibro lental hyperplasia and Retinal detachment and can lead to blindness

7- Risk of fire:

Incorrect storage and use of oxygen

Symptoms of O_2 toxicity

1- Dry cough
2- Sore throat and throat irritation
3- Chest tightness and chest pain
4- Difficult breathing
5- Muscle twitching especially in face and hands
6- Blurred vision
7- Nausea
8- Convulsion, loss of consciousness
9- Death

Ministry of Higher Education and Scientific Research

College of Health and Medical Technology

Anaesthesia Techniques Department

Subject: Intensive Care Unit, 3rd stage.

2023-2024

Hypoxia and Oxygen Therapy

L-8

Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

Introduction

he administration of supplemental oxygen is one of the world's most used therapies and is a cornerstone of care in the intensive care unit (ICU). The primary rationale is to avoid hypoxemia in patients with, or at risk for, impaired pulmonary gas exchange. Oxygen is generally considered to be widely available (which may not be true in less developed countries), inexpensive, and very safe. Consequently, it is typically administered liberally with an upward titration of the fraction of inspired oxygen (Fio2) to achieve a high level of arterial oxygen saturation (e.g., >96%), with less attention on avoidance of excess use.

Introduction

However, the use of supplemental oxygen is not without risk. An elevated level of the partial pressure of arterial oxygen (Pao2), or hyperoxemia, increases the production of toxic reactive oxygen species, which can cause injury, especially in the lungs, retinae, and central nervous system. High Fio2 values in patients with alveolar–capillary units that are poorly ventilated can also lead to absorption atelectasis. Liberal oxygen use is associated with increased mortality in observational studies, but residual confounding complicates an interpretation of these studies

Previous study

- Hypoxemia should certainly be avoided, but the fact that the liberal administration of oxygen to patients in intensive care units and emergency rooms tends to increase morbidity and mortality implies the advisability of a conservative, normoxic oxygenation strategy.
- In emergency situations, oxygen therapy can be life-saving for patients with hypoxemia, but harmful effects of exposure to high oxygen concentrations (oxygen toxicity) have long been known. Within a few years of the introduction of oxygen therapy, the issue of potential oxygen toxicity was raised.

Previous study

- Today, we know that increased concentrations of oxygen free radicals cause cellular damage which can lead to apoptosis or necrosis, especially in the presence of other factors, such as, for example, infection. Cell death triggers the release of mediators, causing, in combination with oxygen free radicals, further cell damage; thereby a vicious cycle is initiated and maintained

- Grensemann J, Fuhrmann V, Kluge S. Oxygen Treatment in Intensive Care and Emergency Medicine. Dtsch Arztebl Int. 2018 Jul 9;115(27-28):455-462. doi: 10.3238/arztebl.2018.0455. PMID: 30064624; PMCID: PMC6111205.

Anoxia, hypoxia, and hypoxemia

Anoxia, hypoxia, and hypoxemia are related terms that all refer to different aspects of oxygen deficiency in the body, but they have distinct meanings and causes:

- **Anoxia:**

- Anoxia is a severe condition characterized by the complete absence of oxygen supply to body tissues and cells.
- It typically occurs when there is a total lack of oxygen, such as when someone is suffocating or drowning and cannot breathe at all.
- Anoxia can result in rapid and severe damage to tissues, including brain damage, if not promptly addressed

Anoxia, hypoxia, and hypoxemia

Hypoxia:

- Hypoxia refers to a condition where there is an insufficient supply of oxygen to body tissues and cells, but it does not necessarily mean a complete lack of oxygen.
- Hypoxia can be caused by various factors, such as reduced oxygen levels in the air (e.g., at high altitudes), decreased oxygen-carrying capacity of the blood (e.g., due to anemia), impaired lung function, or poor circulation.
- There are different degrees of hypoxia, ranging from mild to severe, depending on the extent of oxygen deprivation. It can be chronic or acute.

Anoxia, hypoxia, and hypoxemia

Hypoxemia:

- Hypoxemia specifically refers to the presence of low oxygen levels in the bloodstream. It is a measurement of the oxygen content in the blood, typically measured as partial pressure of oxygen (PaO2) in arterial blood.
- Hypoxemia can occur in various conditions, such as lung diseases (e.g., chronic obstructive pulmonary disease, pneumonia), cardiovascular problems (e.g., heart failure), or during certain medical procedures.
- Hypoxemia can lead to hypoxia if oxygen delivery to tissues is compromised, as oxygen in the blood is what carries oxygen to body cells

Basics

Anoxia

No oxygen availability in tissues

Hypoxia

Lack of oxygen availability in tissues

Hypoxemia

Lack of oxygen in the blood

Transport of Oxygen from the Lungs to the Body Tissues

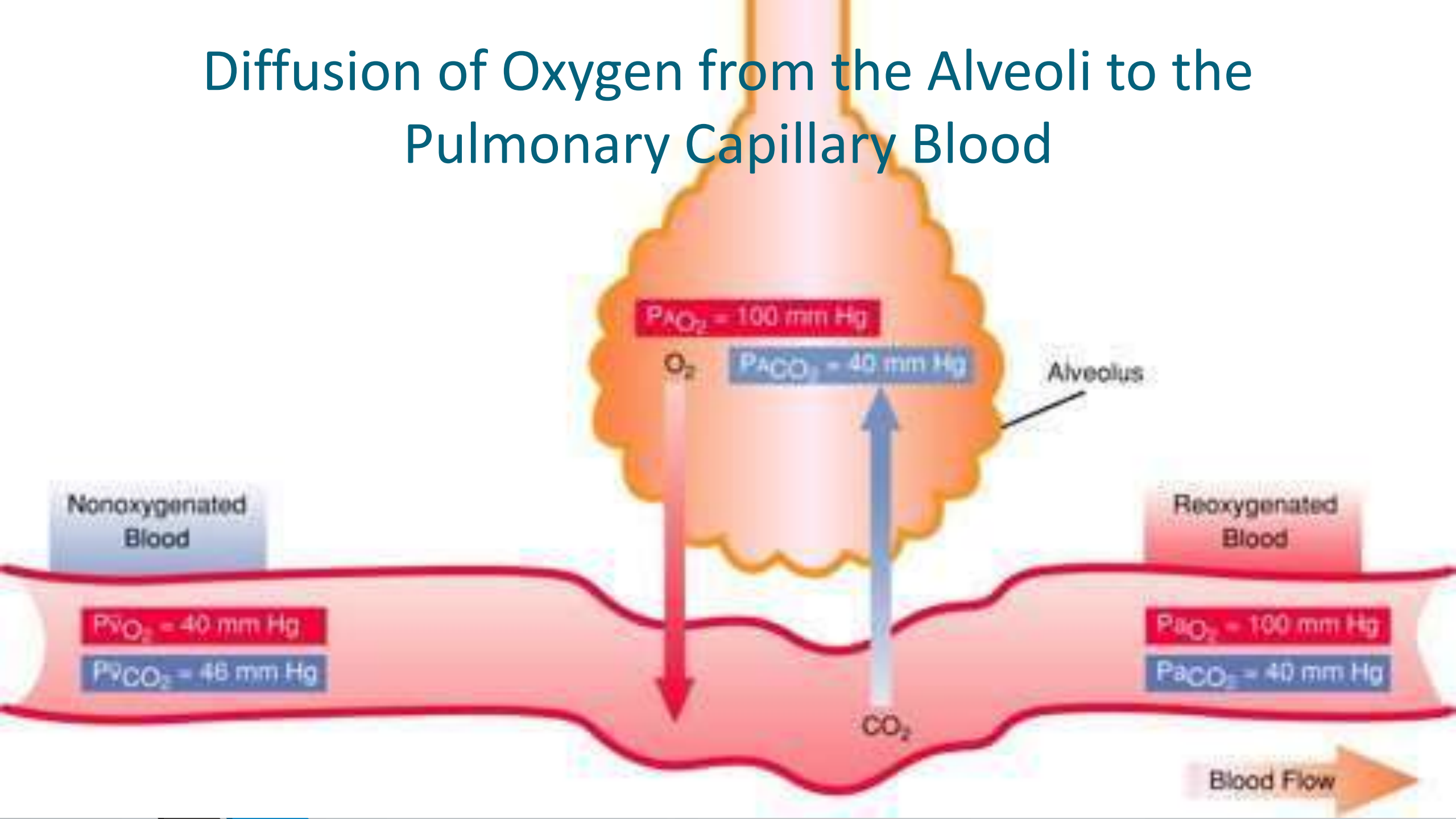
- Oxygen diffuses from the alveoli into the pulmonary capillary blood because the PO_2 in the alveoli is greater than the PO_2 in the pulmonary capillary blood.
- In the other tissues of the body, a higher PO_2 in the capillary blood than in the tissues causes oxygen to diffuse into the surrounding cells.

- when oxygen is metabolized in the cells to form CO_2, the PCO_2 rises to a high value, which causes CO_2 to diffuse into the tissue capillaries.
- After blood flows to the lungs, the CO_2 diffuses out of the blood into the alveoli, because the PCO_2 in the pulmonary capillary blood is greater than that in the alveoli.

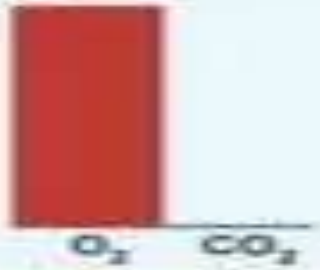
Table 40-1 Partial Pressures of Respiratory Gases (in mm Hg) as They Enter and Leave the Lungs (at Sea Level)

	Atmospheric Air	Humidified Air	Alveolar Air	Expired Air
N_2	597 (78.62)	563.4 (74.09)	569 (74.9)	566 (74.5)
O_2	159 (20.84)	149.3 (19.67)	104 (13.6)	120 (15.7)
CO_2	0.3 (0.04)	0.3 (0.04)	40 (5.3)	27 (3.6)
H_2O	3.7 (0.50)	47 (6.20)	47 (6.2)	47 (6.2)
Total	760 (100)	760 (100)	760 (100)	760 (100)

Diffusion of Oxygen from the Alveoli to the Pulmonary Capillary Blood



Inspired air:
P_{O_2} 160 mm Hg
P_{CO_2} 0.3 mm Hg



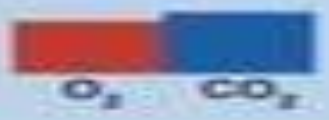
External respiration

Alveoli of lungs:
P_{O_2} 104 mm Hg
P_{CO_2} 40 mm Hg

Pulmonary arteries

Pulmonary veins (P_{O_2} 100 mm Hg)

Blood leaving tissues and entering lungs:
P_{O_2} 40 mm Hg
P_{CO_2} 45 mm Hg



Blood leaving lungs and entering tissue capillaries:
P_{O_2} 100 mm Hg
P_{CO_2} 40 mm Hg

Heart

Systemic veins

Systemic arteries

Internal respiration

Tissues:
P_{O_2} less than 40 mm Hg
P_{CO_2} greater than 45 mm Hg

Oxygen transported in the blood

O2 is carried within the circulation from the lungs to the tissues in two forms:

- Bound to Hb, accounting for 98% of O2 carried by the blood. Each gram of fully saturated Hb can bind 1.34 mL of O2 (this is called Hüfner's constant).
- Dissolved in plasma, accounting for 2% of O2 carried by the blood. The volume of O2 dissolved in blood is proportional to the partial pressure of O2 (this is Henry's law).

oxygen content equation:

O_2 content per 100 mL of blood = (1.34 ×Hb×SaO_2/ 100%) +0.023 × PO_2

where 1.34 mL/g is Hüfner's constant at 37 °C for typical adult blood, [Hb] is the Hb concentration (g/dL), SaO_2 is the percentage Hb O_2 saturation, 0.023 is the solubility coefficient for O_2 in water and PO_2 is the blood O_2 tension (kPa).

For typical arterial blood ([Hb] = 15 g/dL, SaO_2 = 97% and PO_2 = 13.0 kPa):

O_2 content per 100 mL arterial blood (CaO_2) =(1.34×15×0.97)+0.023×13 = 19.50+0.30 = 19.8 mL

whereas venous blood (Hb O_2 saturation of 75%, PO_2 = 5.3 kPa) contains

O_2 content per 100 mL venous blood (CvO_2) = $(1.34 \times 15 \times 0.75) + 0.023 \times 5.3 = 15.08 + 0.12 = 15.2$ mL

The above worked example demonstrates that Hb is a much more efficient means of O2 carriage than O2 dissolved in plasma. However, it would be wrong to think that dissolved O2 is unimportant. The O2 tension of blood is determined from the amount of O2 dissolved in plasma – the PO2 within an RBC is low because all the O2 is bound to Hb. Fick's law of diffusion states that diffusion occurs along a pressure gradient, so O2 diffuses to the tissues from the dissolved portion in the plasma, not from Hb itself. O2 then dissociates from Hb as plasma PO2 falls, replenishing the O2 dissolved in the plasma.

How do the body's oxygen stores compare with its consumption of oxygen?

Very little O2 is stored in the body, which means that periods of apnea can rapidly lead to hypoxia. In addition to O2 in the lungs (within the FRC), O2 is stored in the blood (dissolved in plasma and bound to Hb) and in the muscles (bound to myoglobin).

As described above, approximately 20 mL of O2 is carried in each 100 mL of arterial blood, and 15 mL of O2 per 100 mL of venous blood. At sea level, a 70 kg man has approximately 5 L of blood, containing approximately 850 mL of O2; a further 250 mL of O2 bound to myoglobin; 450 mL of O2 in the lungs, when breathing air.

How do the body's oxygen stores compare with its consumption of oxygen?

This gives a total of 1550 mL of O2. An adult's resting O2 consumption is approximately 250 mL per minute, which means that apnea can occur for only a few minutes before the onset of significant cellular hypoxia. Hypoxic damage occurs even more quickly when there is reduced O2-carrying capacity (for example, anemia or carbon monoxide poisoning) or an increased rate of O2 consumption (for example, in children).

The structure of red blood cells

RBCs are small, flexible biconcave discs (diameter 6–8 μm) that are able to deform enough to squeeze through the smallest of capillaries (around 3 μm in diameter). RBCs can be considered to be ‘bags of Hb’. The RBC nucleus is lost in the latter stages of maturation in the bone marrow during erythropoiesis. Reticulocytes normally make up 1% of circulating RBCs, but this proportion may be increased if erythropoiesis in the bone marrow is highly active; for example, in haemolytic anaemia or following haemorrhage.

As the RBC cytoplasm does not contain mitochondria, aerobic metabolism is not possible. RBCs are unique, as they constitute the only cell type that is entirely dependent on glucose and the glycolytic pathway to provide energy for metabolic.

What is cooperative binding?

Hb is essentially either fully saturated with O2 (oxyhaemoglobin)

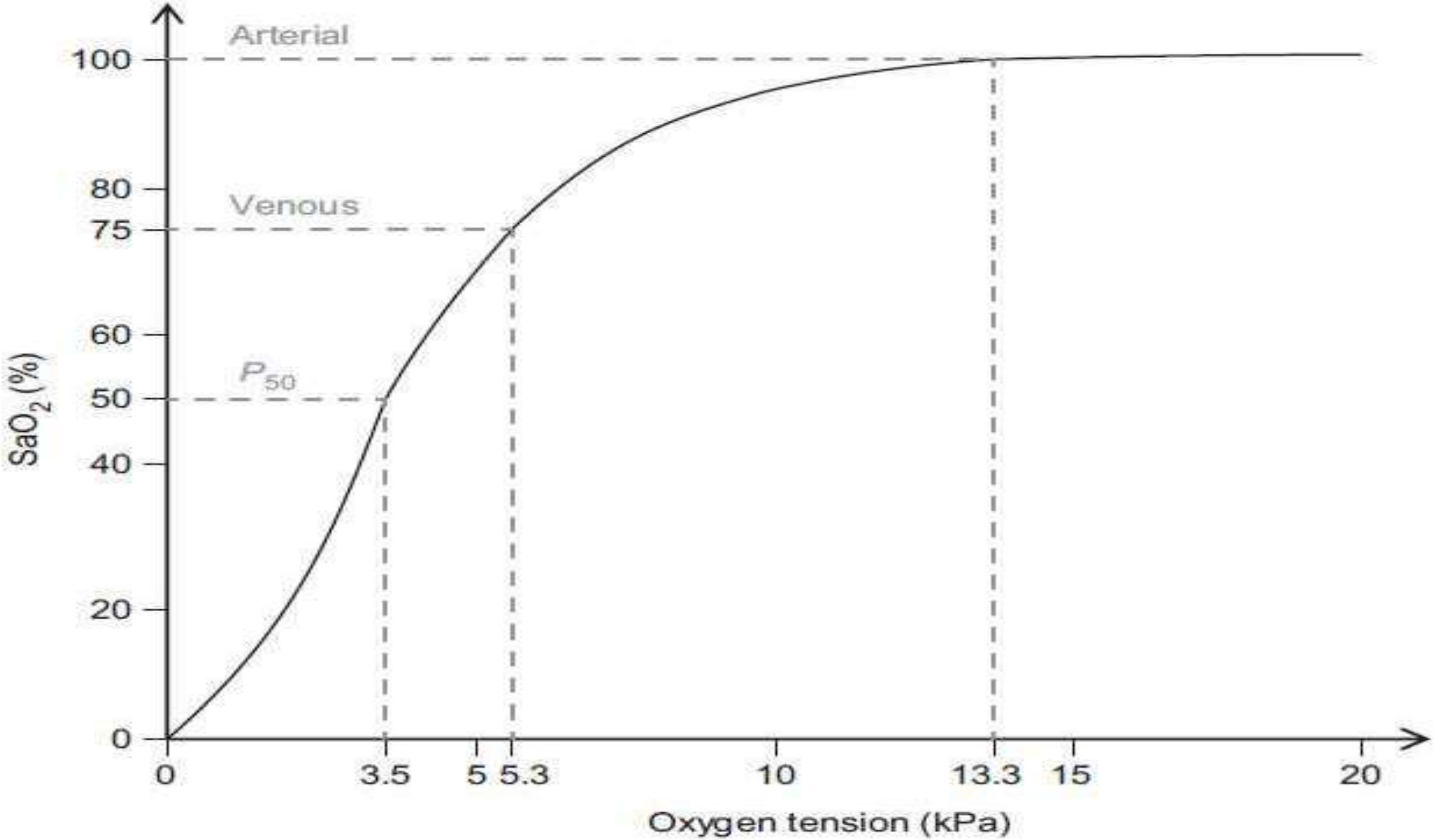
or fully desaturated (deoxyhaemoglobin) due to cooperativity.

Cooperative binding is the increase in O2 affinity of Hb with each successive O2 binding:

- The first O2 molecule is difficult to bind – strong electrostatic charges must be overcome to achieve the required conformational changes in the Hb molecule.
- Once the first O2 has bound, the conformation of Hb changes and the -chains come closer together.

What is cooperative binding?

- Once the second O2 molecule has bound, the third is easier to bind, and so on. In fact, the fourth O2 molecule binds 300 times more easily than the first.
- Once the fourth O2 has bound, Hb is said to be in the relaxed conformation. Cooperative binding is responsible for the sigmoid shape of the oxyhaemoglobin dissociation curve


Arterial
Venous
P_{50}
100
80
75
60
50
40
20
0
SaO_2 (%)
0
3.5
5
5.3
10
13.3
15
20
Oxygen tension (kPa)

THANK YOU FOR ATTENTION!

Have a nice day!!!

Hypoxia and Oxygen Therapy

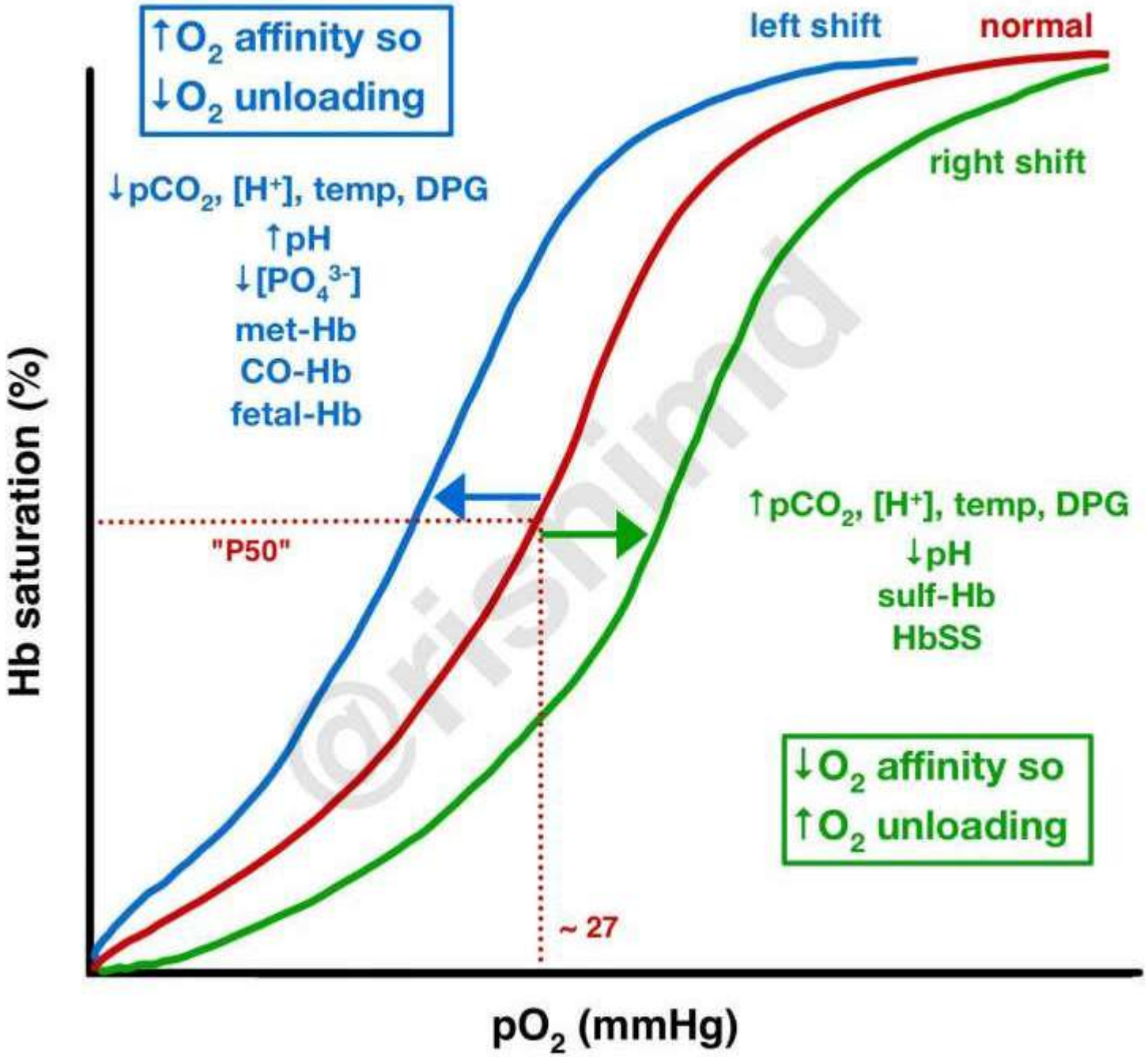
L-9

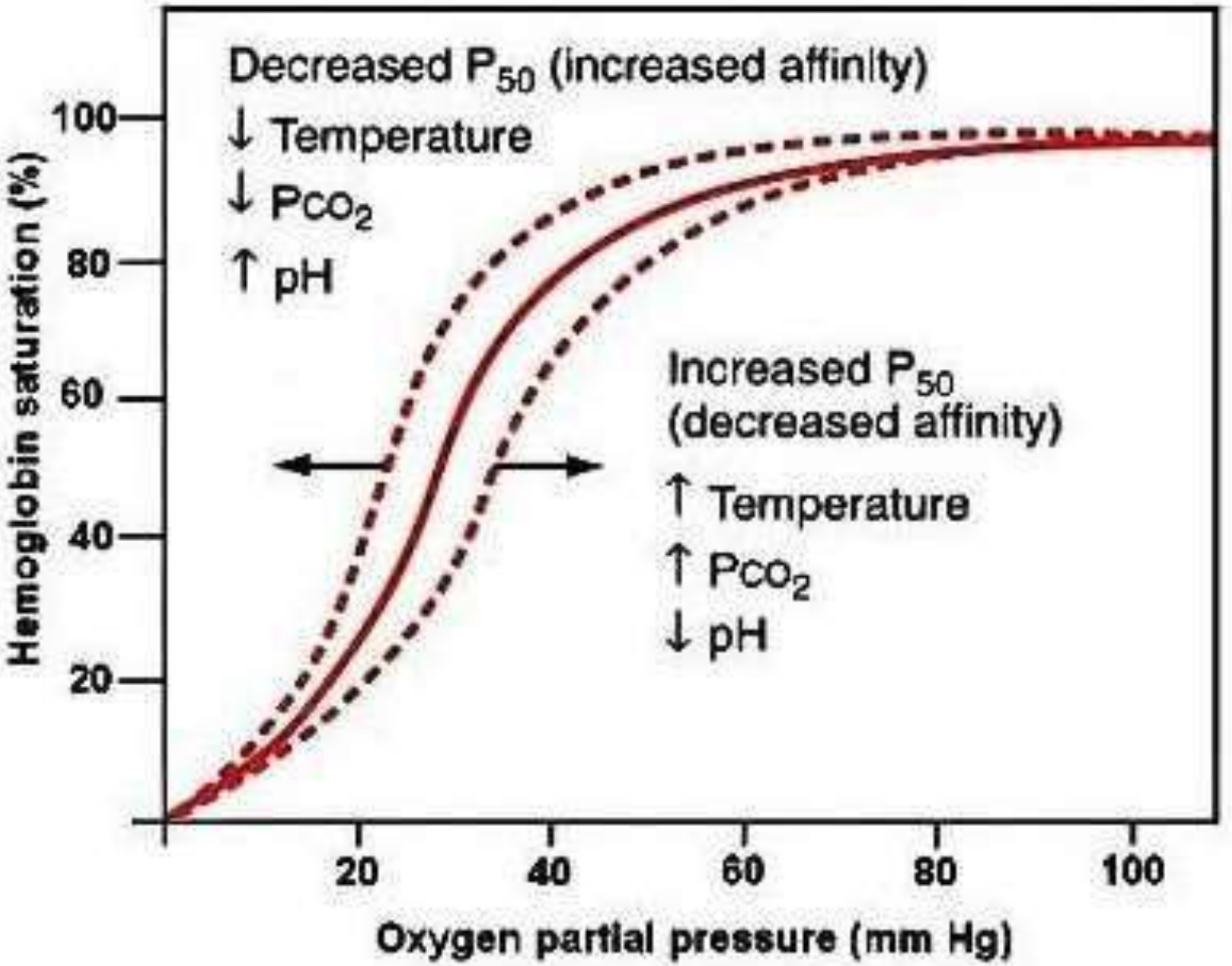
Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

What is the oxyhemoglobin dissociation curve?

The oxyhemoglobin dissociation curve describes the relationship between SaO2 and blood O2 tension. As discussed above, the cooperative binding of Hb is responsible for the curve's sigmoid shape, which has important clinical consequences:

- The upper portion of the curve is flat. At this point, even if PaO2 falls a little, SaO2 hardly changes. However, when PaO2 is already pathologically low (for example, in patients with respiratory disease) and near to the steep part of the curve, a further fall in PaO2 results in a large decrease in SaO2.
- The steep part of the curve is very important in the peripheral tissues, where PO2 is low: the steep fall in SaO2 means a large quantity of O2 is offloaded for only a small decrease in PO2.


↑O_2 affinity so
↓O_2 unloading
left shift
normal
right shift
↓pCO_2, [H^+], temp, DPG
↑pH
↓[PO_4^{3-}]
met-Hb
CO-Hb
fetal-Hb
↑pCO_2, [H^+], temp, DPG
↓pH
sulf-Hb
HbSS
↓O_2 affinity so
↑O_2 unloading
"P50"
~ 27
Hb saturation (%)
pO_2 (mmHg)



OXYGEN DISSOCIATION CURVE

The Bohr effect.

Metabolically active tissues produce CO2, heat and H+ ions. When blood arrives at these capillaries, the oxyhaemoglobin dissociation curve is shifted to the right, offloading O2 where it is most needed. This phenomenon is called the ‘Bohr effect’ or Bohr shift.

O2 loading in the lungs

When blood reaches the lungs, CO2 is excreted and the pH normalizes. The P50 of the oxyhemoglobin dissociation curve then returns to its central position. The binding affinity of O2 therefore increases: dissolved O2 binds to Hb, which in turn lowers the blood O2 tension, facilitating O2 diffusion across the alveolar–capillary barrier.

Hypoxia

In the context of an Intensive Care Unit (ICU), hypoxia is a critical concern that requires prompt recognition, assessment, and management to ensure optimal patient outcomes. Hypoxia, characterized by insufficient oxygen supply to body tissues and cells, can arise in the ICU due to a variety of underlying medical conditions and complications.

Hypoxemic hypoxia

- **Cause:** Hypoxemic hypoxia is characterized by low oxygen levels in the arterial blood, often due to respiratory problems that hinder oxygen exchange in the lungs. Causes include lung diseases (e.g., pneumonia, ARDS), pulmonary embolism, and hypoventilation.
- **Mechanism:** Impaired oxygen exchange in the lungs leads to reduced oxygen saturation in arterial blood, limiting oxygen delivery to body tissues.

Hypoxemic hypoxia

Hypoxemic hypoxia may be because of-

- Low FiO2, hypoventilation
- Ventilation perfusion mismatch,
- Diffusion defect, or
- Shunt effect

Anaemic hypoxia

- Cause: Anemic hypoxia results from a reduced oxygen-carrying capacity of the blood, typically due to a decrease in the number of red blood cells or a decrease in the hemoglobin content. It can be caused by conditions such as anemia, hemorrhage, or certain genetic disorders.
- Mechanism: In anemic hypoxia, there may be an adequate supply of oxygen in the air and normal lung function, but the blood is unable to carry sufficient oxygen to meet the body's demands. This results in tissue hypoxia.

Anaemic hypoxia

Anaemic hypoxia may be because of-

- A decreased hemoglobin level
- CO poisoning
- Excessive blood loss
- Methemoglobinemia
- Iron deficiency

Stagnant hypoxia

- **Cause:** Stagnant hypoxia occurs when there is reduced blood flow or circulation, leading to inadequate oxygen delivery to tissues. This can be caused by conditions like heart failure, shock, or circulatory problems.
- **Mechanism:** In stagnant hypoxia, the oxygen-rich blood fails to reach tissues effectively, leading to localized oxygen deficits. It can result in tissue damage and organ dysfunction.

Stagnant hypoxia

Stagnant hypoxia may be because of-

- Capillary perfusion is diminished due to different causes such as
  - Decreased heart rate
  - Decreased cardiac output
  - Shock
  - Embolism
  - Exposure to cold weather

Histotoxic hypoxia

- **Cause:** Histotoxic hypoxia arises from conditions that impair the ability of tissues to use oxygen efficiently, even when oxygen delivery is adequate. This can result from exposure to certain toxins, drugs, or metabolic disorders.
- **Mechanism:** Despite normal oxygen levels in the blood, tissues are unable to utilize oxygen effectively, leading to cellular dysfunction and hypoxia.

Histotoxic hypoxia

Histotoxic hypoxia may be because of

- The oxidative enzyme mechanism of the cell is impaired as a result of:
  - Cyanide poisoning
  - Alcohol poisoning
- Rarely accompanied by hypoxemia but is accompanied by increased venous PO2 levels.

Oxygen Therapy

Correct administration of oxygen is lifesaving, but many a times it is given without careful evaluation of its potential benefits and side effects. Oxygen is the commonest drug used in patients admitted in Intensive Care Unit. Like other drugs oxygen also carries a clear indication for treatment and appropriate methods for its delivery. Inappropriate dose and failure to monitor can have serious consequences. Vigilant monitoring is essential to detect and correct adverse effects.

Assess the Need for Oxygen Therapy

- Provide oxygen for measured hypoxemia and not only for breathlessness.
- Measure oxygen saturation and if it is below 90%, provide supplemental oxygen.
- If pulse oximetry signal not adequate, check arterial PaO2.
- Record approximate FiO2 given and oxygen saturation or PaO2/FiO2 ratio in patient's chart and monitor it at interval as per hospital protocol.

Assess the Need for Oxygen Therapy

- Set a target of oxygen saturation for each patient, low normal (88–92%) for COPD, target for not at risk of hypercapnic respiratory failure is 94–98%.
- In emergency situations (e.g., cardiorespiratory arrest, acute cardiogenic pulmonary edema, or stroke), oxygen administration may be initiated empirically, pending detailed clinical and laboratory evaluation.
- Investigate and manage the underlying cause of hypoxemia at the same time.

Assess the Need for Oxygen Therapy

- Supplemental oxygen improves oxygenation but does not treat the underlying causes of hypoxemia which must be diagnosed and treated urgently.
- Oxygen delivery devices and flow rates should be adjusted to keep the oxygen saturation in the target range.
- Prompt clinical assessment is needed if oxygen therapy needs to be initiated or increased due to a decreasing saturation level

Oxygen Administration

- Before giving oxygen, one needs to ensure patency of the airways. This might require endotracheal intubation or tracheostomy.
- It is generally customary to start with a high FiO2—100% for cardiorespiratory arrest and 50–100% for acute hypoxemic respiratory failure.
- The FiO2 can be increased or decreased after the assessment of clinical and laboratory response to the initial administration.

Oxygen Administration

- Relatively lower concentrations are used in patients with hypercapnic respiratory failure (such as COPD) with preexisting chronic hypoventilation.
- High concentration of oxygen may worsen CO2 retention and cause CO2 narcosis by abolishing the hypoxic respiratory stimulation. However, optimum FiO2 must be ensured since hypoxia is always more deleterious than hypercapnia. Various devices can be used for applying oxygen.
- Select optimum oxygen delivery device

Identifying and Treating Underlying Causes:

- Effective management of hypoxia in the ICU requires addressing the underlying medical conditions or contributing factors. Common causes of hypoxia in the ICU include acute respiratory distress syndrome (ARDS), pneumonia, pulmonary embolism, heart failure, and sepsis.
- Targeted therapies, such as antibiotics for infections or diuretics for pulmonary edema, may be administered as appropriate.

Mechanical Ventilation

- In cases of severe hypoxia or respiratory failure, mechanical ventilation is employed to support oxygenation and ventilation. Ventilators can provide precise control over oxygen levels, positive end-expiratory pressure (PEEP), and other parameters.
- Ventilatory strategies, including lung-protective ventilation, are employed to minimize ventilator-associated lung injury.

THANK YOU FOR ATTENTION!

Have a nice day!!!

Hypoxia and Oxygen Therapy

L-9

Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

Low flow oxygen systems

Low flow oxygen systems: Low flow oxygen systems or delivery devices does not meet the patients inspiratory flow demands. Therefore room air must make up the remainder of the patient's tidal volume. The percentage of oxygen delivered by low flow devices is variable depending on patient's tidal volume, respiratory rate, inspiratory time and ventilatory pattern

Low-Flow Oxygen Therapy: A Fundamental Approach to Respiratory Support

1. Purpose:

- Supplemental Oxygen: The primary goal of low-flow oxygen therapy is to increase the concentration of oxygen in the inhaled air to maintain or improve oxygen levels in the bloodstream.
- Respiratory Support: It is used to support individuals with conditions such as chronic obstructive pulmonary disease (COPD), pneumonia, heart failure, and other respiratory disorders that can lead to hypoxia (insufficient oxygen in the body).

Low-Flow Oxygen Therapy: A Fundamental Approach to Respiratory Support

2. Flow Rates:

- Low-flow oxygen therapy devices deliver oxygen at rates typically less than 15 liters per minute (LPM). The exact flow rate prescribed depends on the patient's condition and oxygen requirements.
- Flow rates are usually measured in liters per minute (LPM) and are determined by a healthcare provider based on the patient's oxygen saturation levels and clinical assessment.

Low-Flow Oxygen Therapy: A Fundamental Approach to Respiratory Support

3. Monitoring:

- Continuous monitoring of oxygen saturation (SpO2) using a pulse oximeter is essential during low-flow oxygen therapy. This helps healthcare providers adjust the oxygen flow rate to maintain target oxygen levels.
- Clinical assessment, including observing the patient's respiratory rate and overall condition, is also crucial for monitoring the effectiveness of therapy.

Low-Flow Oxygen Therapy: A Fundamental Approach to Respiratory Support

4. Considerations:

- Patients on low-flow oxygen therapy should be educated on proper device usage, care, and safety precautions.
- Patients should be advised to avoid smoking or being near open flames while using oxygen, as oxygen supports combustion.
- Humidification may be added to the oxygen source if the patient experiences dryness or irritation of the nasal passages.

Low-Flow Oxygen Therapy: A Fundamental Approach to Respiratory Support

5. Patient Populations:

- Low-flow oxygen therapy is commonly used for patients with chronic respiratory conditions, those recovering from surgery, and individuals experiencing acute respiratory distress.

7. Benefits:

- Low-flow oxygen therapy helps alleviate symptoms of hypoxia, such as shortness of breath, fatigue, and confusion.
- It can improve the overall quality of life for individuals with chronic respiratory conditions by enhancing their ability to engage in daily activities.

Low-Flow Oxygen Therapy: A Fundamental Approach to Respiratory Support

- Low-flow oxygen therapy is an essential and accessible means of respiratory support that can make a significant difference in the well-being of individuals with respiratory disorders. It is a fundamental tool in the management of various medical conditions and is often administered in outpatient settings, emergency departments, hospitals, and long-term care facilities.

Oxygen Delivery Devices

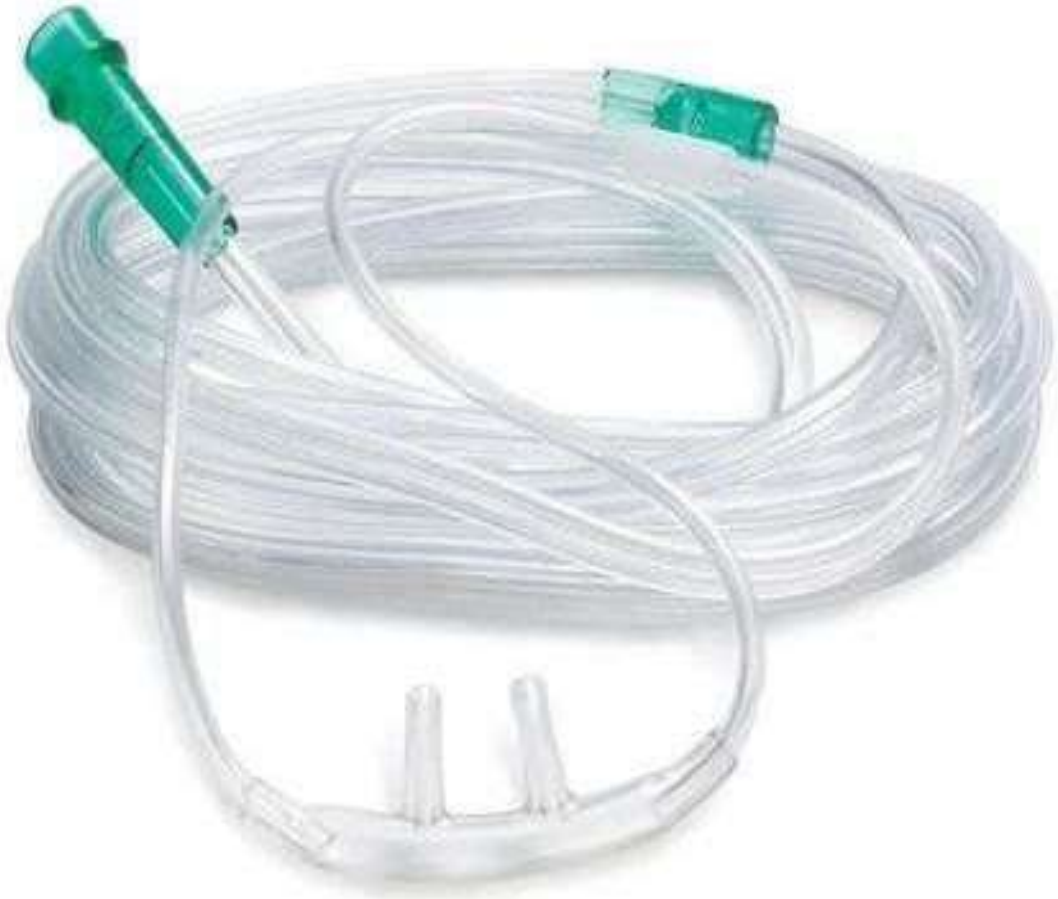
(a)**Nasal Cannula** (delivers 24–40% of oxygen at a flow rate of 1–6 L/min)

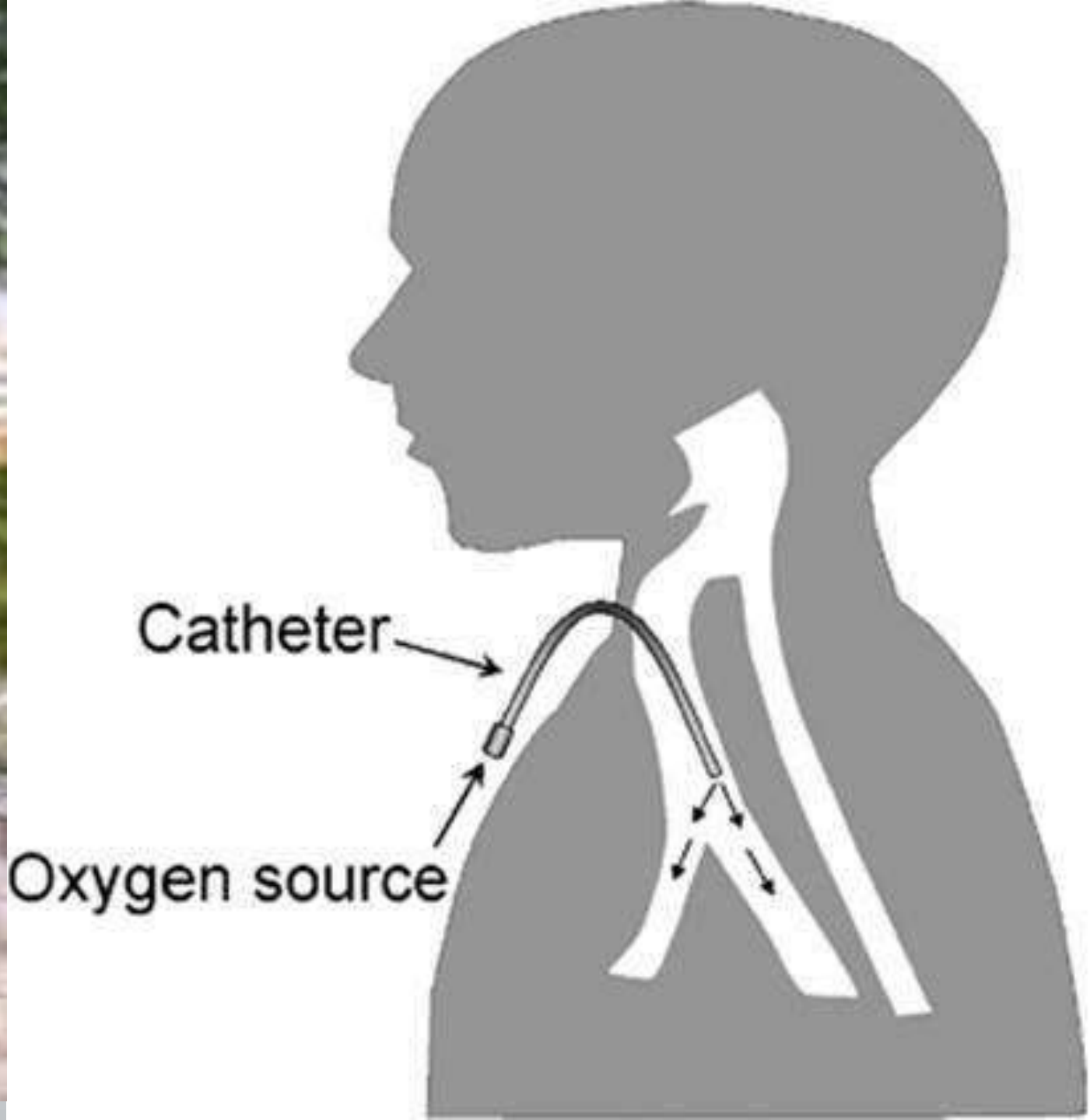
(b)**Nasal Reservoir Cannula** (Reservoir stores upto 20 mL of oxygen, delivers 22–35% of oxygen at a flow rate of 1–4 L/min)

(c) **Pendant Reservoir Cannula** (delivers 22–35% of oxygen at a flow rate of 1–4 L/min)

Oxygen Delivery Devices

(d) **Simple Oxygen Mask** (delivers 35–50% of oxygen at a flow rate of 5–10 L/min)
(e) **Partial Re-breathing Mask** (delivers 40–70% of oxygen at a flow rate of 10–15 L/min)
(f) **Non Re-breathing Mask** (delivers 60–80% of oxygen at a flow rate of 10–15 L/min)
(g) **Transtracheal Oxygen Catheter** (delivers 22–35% oxygen at a flow rate of 1–4 L/min)




Catheter
Oxygen source

Low flow oxygen devices are adequate only when patient meets the following criteria:

- Mild to Moderate Hypoxia: Low-flow oxygen therapy is generally suitable for patients with mild to moderate hypoxia, as indicated by their oxygen saturation levels (SpO2) between 88% and 92% or within the target range prescribed by their healthcare provider. Patients with severe hypoxia may require higher flow rates delivered through high-flow devices or mechanical ventilation.
- Relatively Stable Respiratory Status: Low-flow oxygen therapy is appropriate for patients with relatively stable respiratory conditions. It is commonly used for individuals with chronic respiratory conditions such as chronic obstructive pulmonary disease (COPD), bronchitis, or interstitial lung disease during their stable phases.

Low flow oxygen devices are adequate only when patient meets the following criteria:

- Adequate Respiratory Rate and Effort: Patients should have a respiratory rate within a manageable range and be able to maintain their airway and effectively breathe in room air when at rest. Low-flow oxygen therapy is often used to relieve respiratory distress in patients with increased work of breathing.
- Ability to Tolerate Nasal Cannula or Face Mask: Patients must be able to tolerate the delivery device, such as a nasal cannula or simple face mask, without significant discomfort, anxiety, or claustrophobia. Some patients may prefer certain delivery devices over others.

Low flow oxygen devices are adequate only when patient meets the following criteria:

- Compliance with Therapy: Patients should be willing and able to comply with the prescribed oxygen therapy regimen, including wearing the oxygen delivery device as recommended and maintaining appropriate flow rates.
- Appropriate Oxygen Saturation Targets: The target oxygen saturation range should be established by the healthcare provider based on the patient's specific medical condition and needs. Low-flow oxygen therapy may not be appropriate for patients requiring precise control of oxygen levels within very narrow ranges.

Low flow oxygen devices are adequate only when patient meets the following criteria:

- Oxygen Assessment and Monitoring: Regular assessment of the patient's oxygen levels, respiratory status, and clinical condition is essential to ensure that low-flow oxygen therapy remains adequate and effective. Adjustments to the therapy may be necessary over time.

THANK YOU FOR ATTENTION!

Have a nice day!!!

High Flow Oxygen Delivery Devices

ICU L11

Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

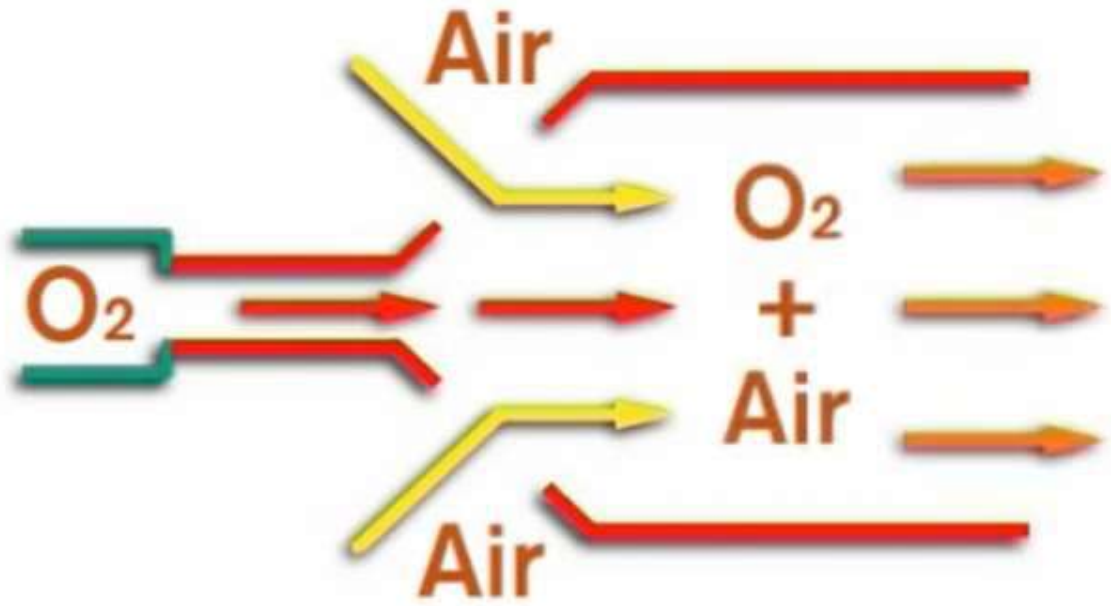
High Flow Oxygen Delivery Devices

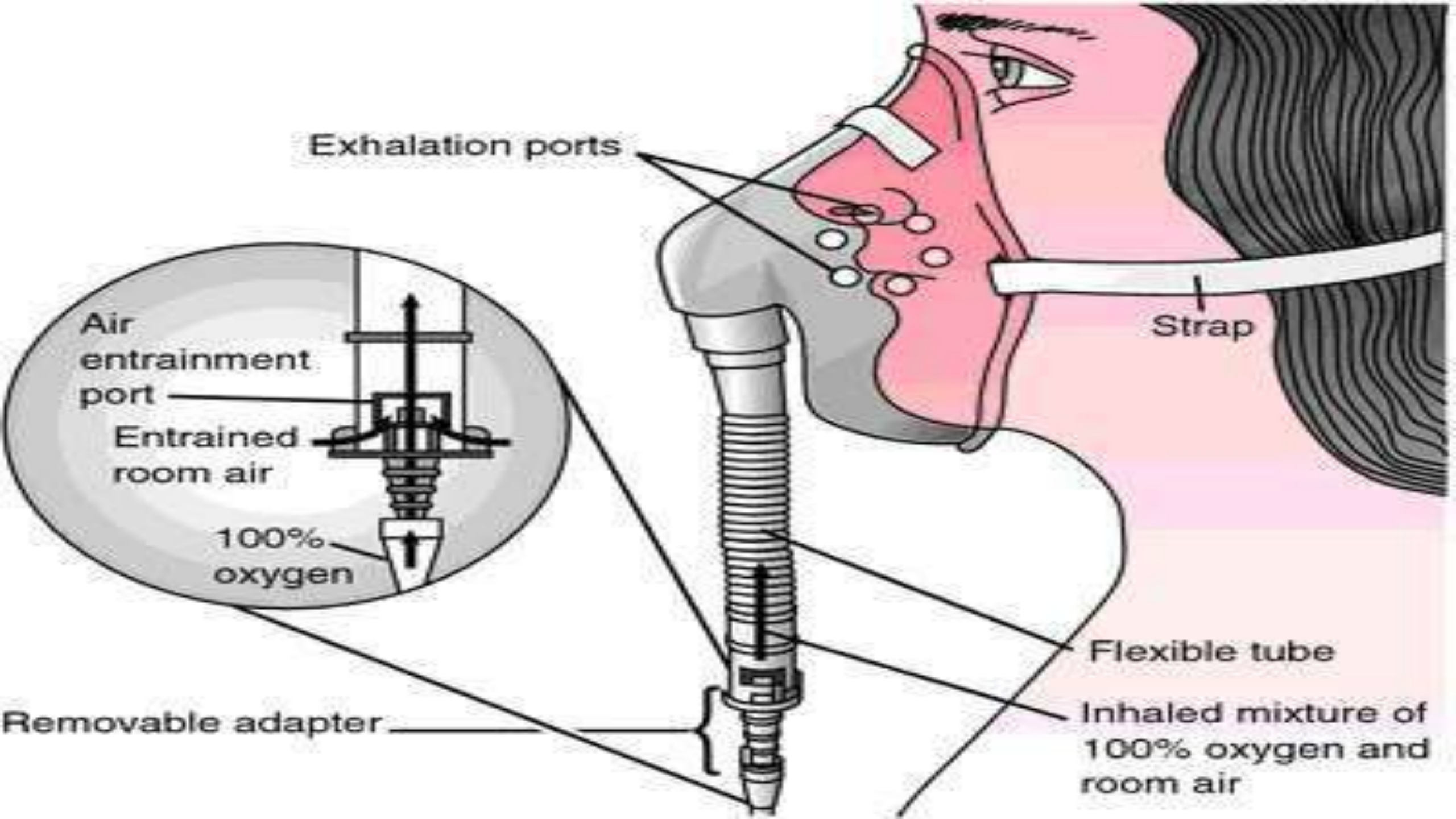
I. Air Entrainment Mask (venturi mask)

- Provides 24–50% of oxygen
- The jet size of the entrainment port determines the FiO2. The larger the jet size the less air entrained and higher the FiO2. Smaller the jet size more air entrained and lower the FiO2.
- Delivers a more accurate level of oxygen by controlling the specific amounts of oxygen delivered. The port on the corrugated tubing (base of the mask) sets the oxygen concentration. Delivers humidified oxygen for patient comfort. It does not dry mucous membranes.

O_2 percentage	Air/O_2 Entrainment ratio
24	25:1
28	10:1
30	8:1
35	5:1
40	3:1
45	2:1
50	1.7:1
60	1:1

Venturi principle




Exhalation ports
Strap
Air entrainment port
Entrained room air
100% oxygen
Flexible tube
Removable adapter
Inhaled mixture of 100% oxygen and room air


40% O2
31% O2
24% O2
28% O2
35% O2

II. Aerosol Mask

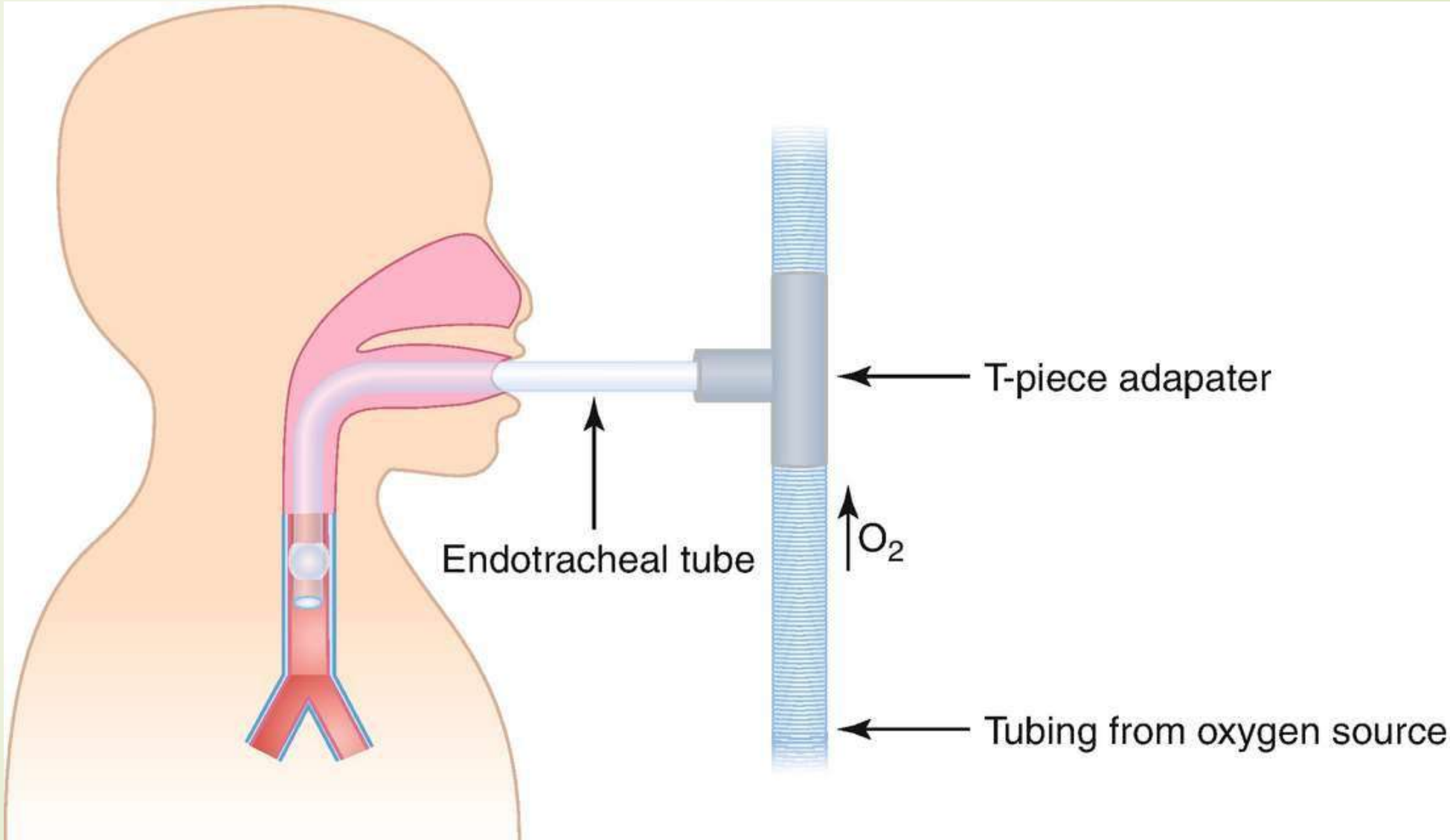
- (This mask delivers 21–100% of oxygen depending on the nebulizer setting at a flow rate of 8–15 L/min)
- The aerosol mask can be used with a nebuliser or 22mm corrugated tubing for combined oxygen therapy and humidification

III. Face tent

- (It delivers 21–40% of oxygen depending on the nebulizer setting at a flow rate of 8–15 L/min)
- Face tents are used to provide a controlled concentration of oxygen and increase moisture for patients who have facial burn or a broken nose, or who are claustrophobic.
- The mask covers the nose and mouth and does not create a seal around the nose.
- It is difficult to achieve high levels of oxygenation with this mask.

IV. T- piece

- (It delivers 21–100% of oxygen at a flow rate of 8–15 L/min)



V. Tracheostomy mask

- (It delivers 35–60% of oxygen at a flow rate of 10–15 L/min)

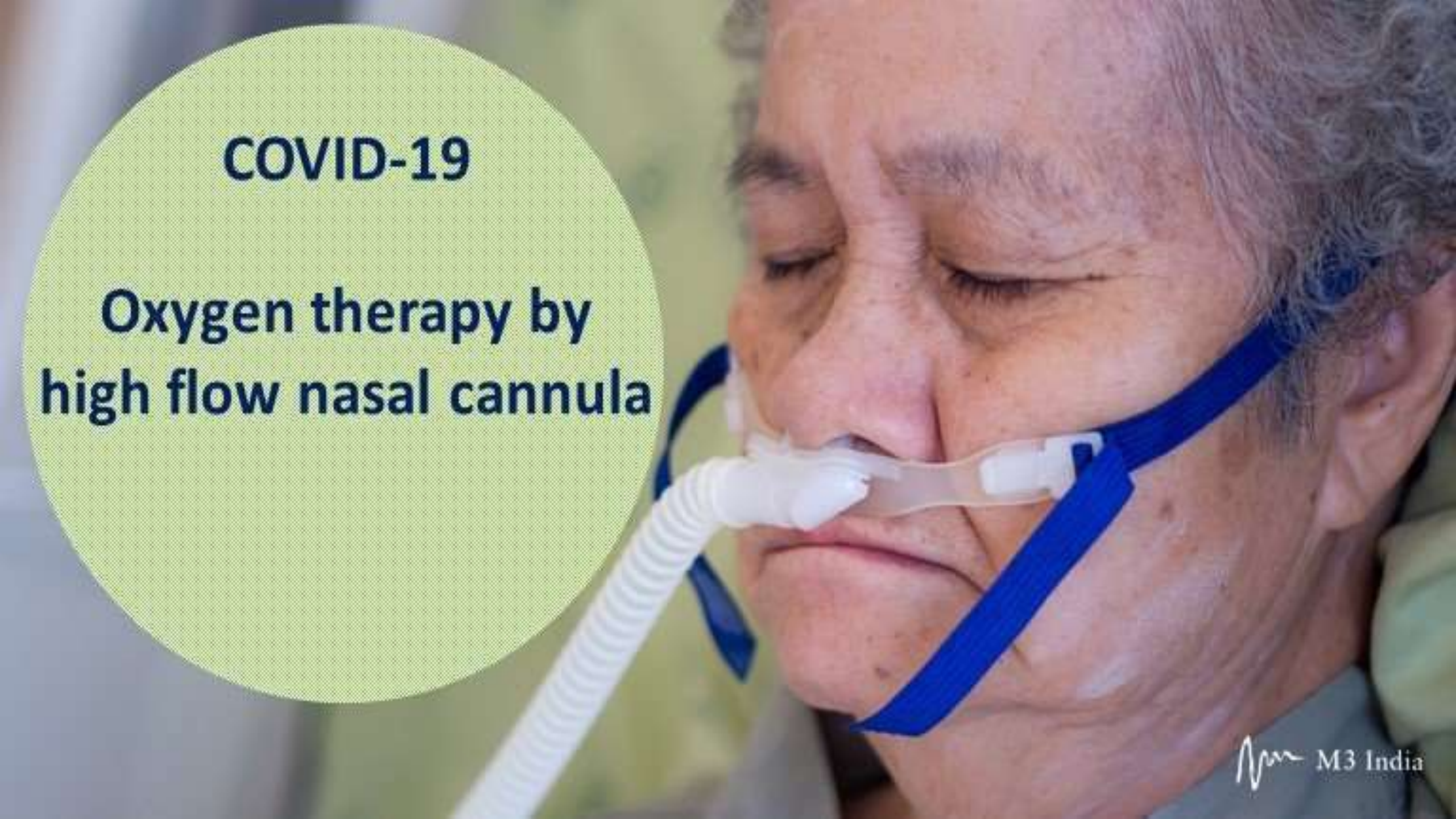
VI. High Flow nasal Cannula

- (Flows up to 8 L/min are used on infants and up to 60 L/min on adults and provide an oxygen percentage of up to 100%)
- High flow devices set on 60% or higher may deliver a total flow rate less than 25–30 L/min. Thereby not meeting the patient's inspiratory demands.
- To ensure adequate flow rate on a device along with high percentage of oxygen it is always better to use two flow meters connected together instead of one.
- To ensure adequate flow rates, set the flow meter to a rate that delivers a total flow of at least 40 L/min.
- Increasing the flow on high flow device does not increase delivered FiO2. It only increases the total flow.

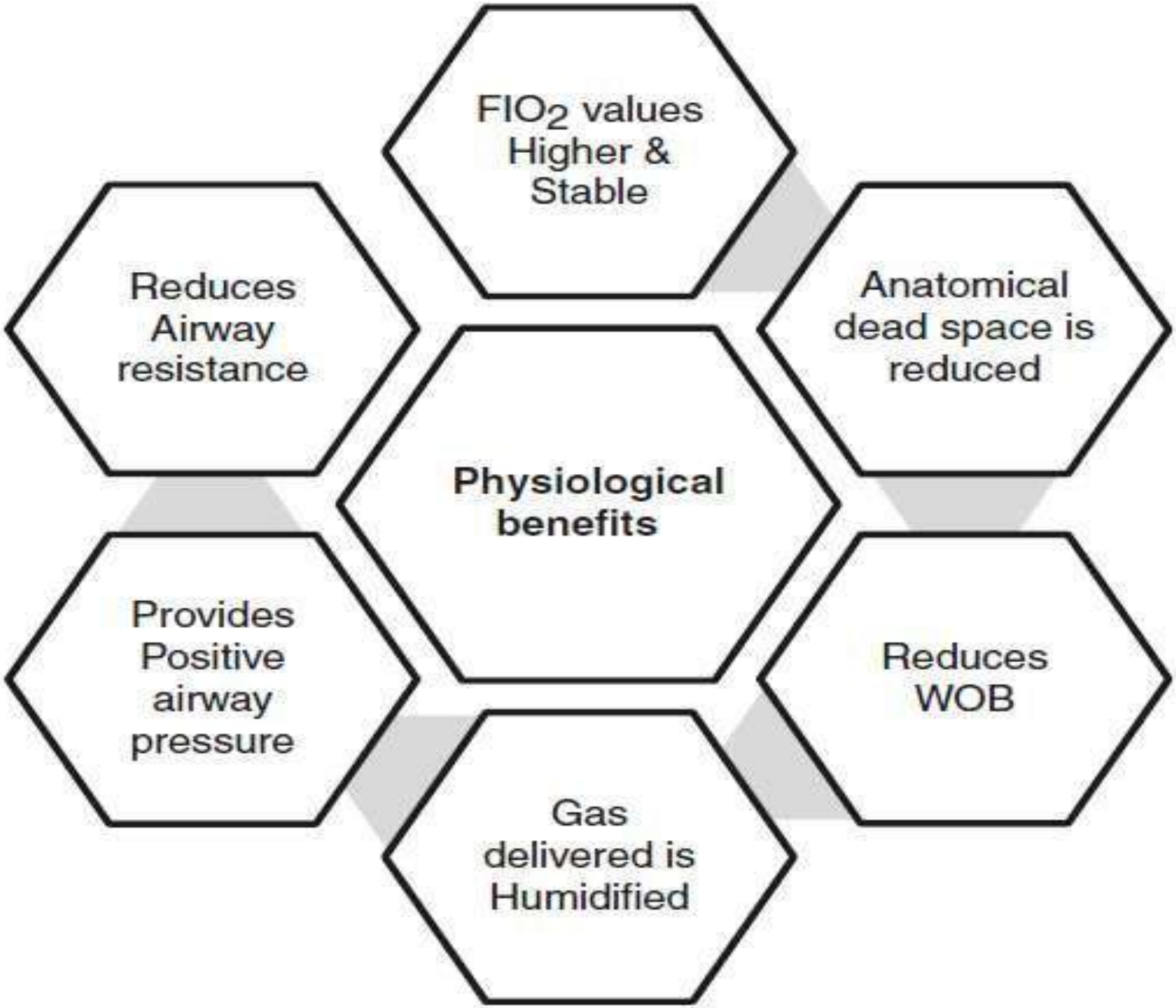
HFNC-High Flow Nasal
Cannual Therapy System

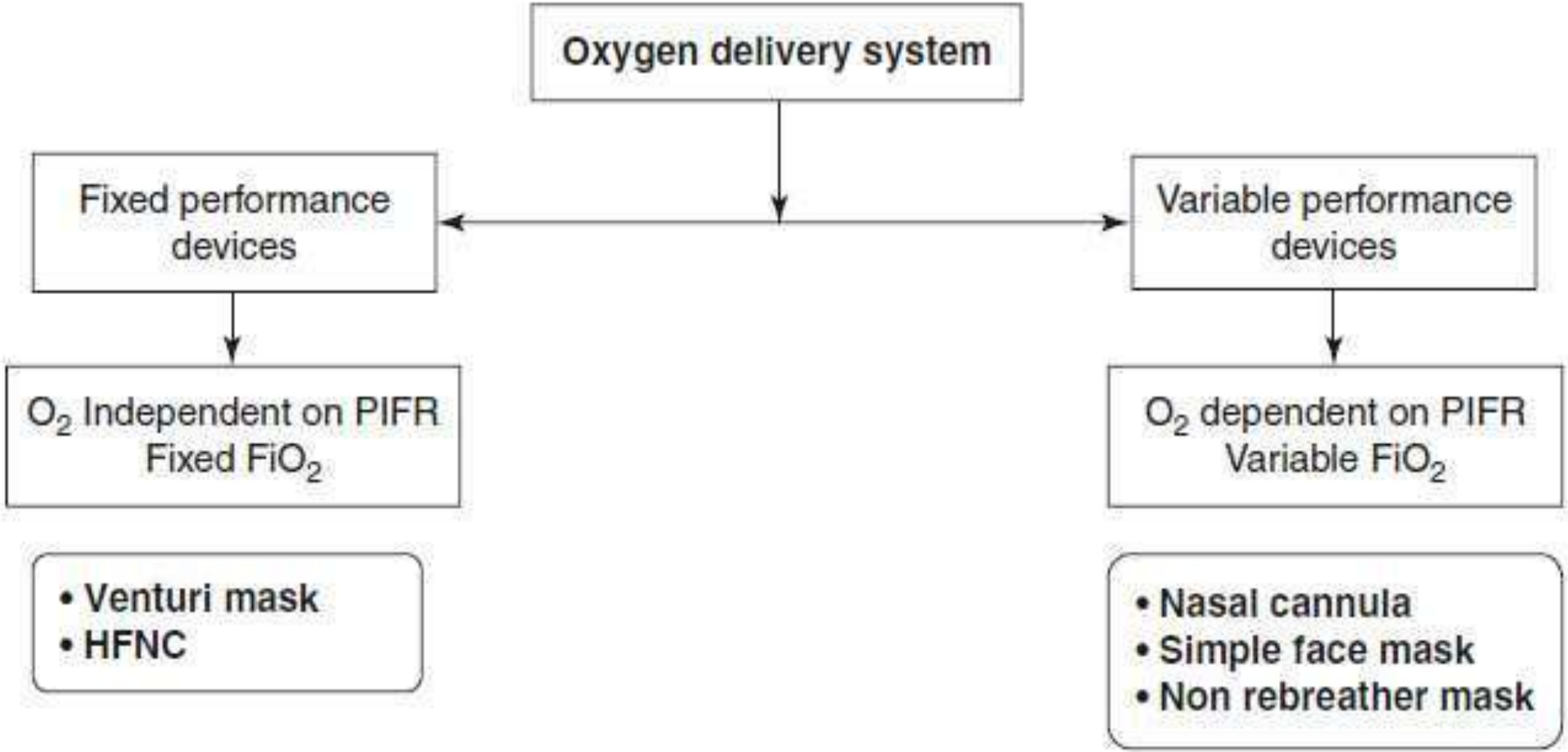
COVID-19

Oxygen therapy by high flow nasal cannula



In patient of hypoxemic respiratory failure, oxygen is applied via variable low-flow systems (e.g., nasal cannulae or face masks) or high-flow systems (e.g., Venturi masks, nonrebreathing reservoir mask). These conventional systems have lot of disadvantages. They deliver unreliable fraction of inspired oxygen and provide inadequate warming and humidification of inspired gas. In a patients with acute respiratory failure, peak inspiratory flow rate is high and often exceeds the oxygen flow delivered by these traditional oxygen devices, which results in flow starvation. High-flow nasal cannula (HFNC) oxygen therapy overcomes these limitations. HFNC is a technique of Oxygen therapy which delivers heated and humidified oxygen through a nasal cannula at high flow rates (~60 L/min)at higher oxygen concentration (FiO2 0.21–1).


FIO_2 values Higher & Stable
Reduces Airway resistance
Anatomical dead space is reduced
Physiological benefits
Provides Positive airway pressure
Reduces WOB
Gas delivered is Humidified



Oxygen delivery devices

NIV VS HFNC

1. Increased work of breathing: NIV preferable
2. Need of high FiO_2: HFNC preferable
3. Need of humidification: HFNC preferable
4. Hypercapnia: NIV preferable
5. Efficient & economical use of oxygen: NIV preferable
6. Comfort and acceptance: HFNC preferable

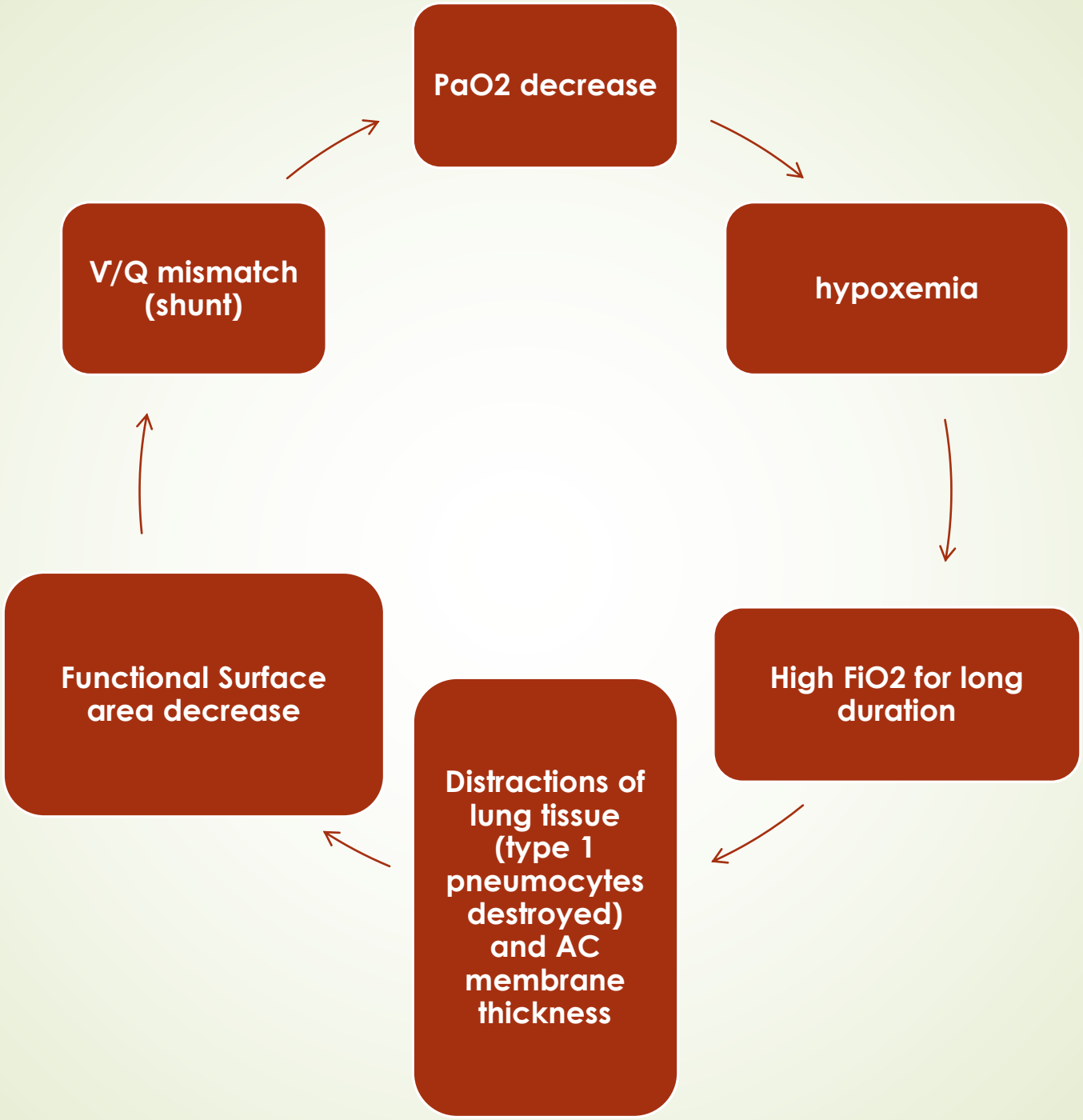
Hazards of oxygen therapy

Oxygen therapy is not without risk. In general, the potential hazards of oxygen therapy include its biologic toxic effects and its potential for physical hazards, e.g. fires. Excessive oxygen administration has been associated with adverse effects involving several organ systems.

Hazards of oxygen therapy

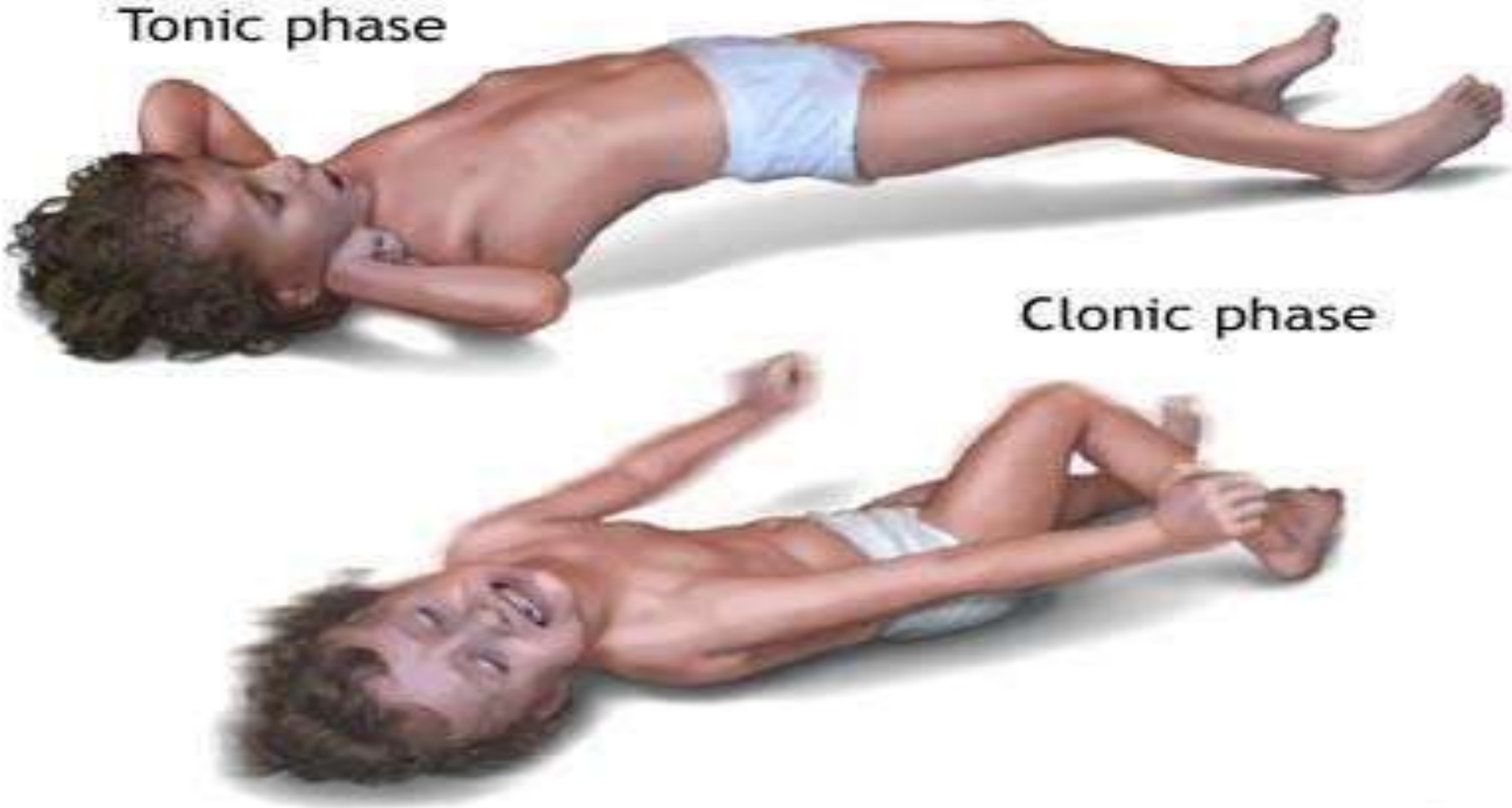
- Oxygen is a drug when given in excess it causes toxicity.
- The duration of exposure is also an important criteria for development of oxygen toxicity.
- The primary effects of oxygen toxicity is on the central nervous system
- High partial pressure of oxygen also damages the capillary endothelium in the lungs. Later on patient may develop pulmonary arterial hypertension.
- The goal should be to use the lowest possible FiO2 to achieve adequate tissue oxygenation.

- Depression of Ventilation: This is a common observation in patients with COPD and chronic hypercapnia.
- Hypoxemia is primarily sensed by peripheral chemoreceptors. Increase in blood oxygen level in these patients suppresses the peripheral chemoreceptors which depresses the ventilatory drive and elevates the $PaCO_2$.


PaO2 decrease
hypoxemia
High FiO2 for long duration
Distractions of lung tissue (type 1 pneumocytes destroyed) and AC membrane thickness
Functional Surface area decrease
V/Q mismatch (shunt)

Central nervous system (CNS)

Exposure to hyperbaric oxygen at more than 2 atmospheres of pressure may cause generalized tonic-clonic seizures. Higher oxygen pressures can induce seizures with shorter durations of exposure. This side effect constitutes the main limitation to increasing oxygen pressures in hyperbaric chambers. Other CNS manifestations of oxygen toxicity include dizziness, nausea, headaches, disorientation, and facial twitching.


Tonic phase
Clonic phase

Ocular

Neonatal exposure to high oxygen concentrations can lead to retinopathy of prematurity (ROP). The risk seems to be greatest in the premature and continues up to 44 weeks of post-conceptual age. Oxygen therapy in neonates at risk should therefore be very tightly controlled and their saturations should never be allowed to exceed 95%.

Pulmonary

Hyperoxia-induced lung injury has long been recognized as a major limitation to the liberal administration of oxygen in the world of critical care. Breathing 100% oxygen over prolonged periods of time can adversely affect the entire respiratory tract.

- Atelectasis is increased due to denitrogenation and alveolar absorption.
- Acute tracheobronchitis may be the earliest manifestation of oxygen toxicity, generally appearing after 4 to 22 hours of breathing 100% oxygen at one atmospheric pressure.
- Diffuse alveolar damage (DAD) develops soon thereafter, starting after 48 hours of inspiring FiO2s greater than 60%.

Pulmonary

- With prolonged hyperoxia, ARDS-like changes are observed at the alveolar level and capillary endothelial damage takes place resulting in interstitial edema.
- The long-term effects of oxygen toxicity are similar to those of ARDS with pulmonary fibrosis, emphysema and worsening gas exchange being the end-result.
- Retrospective studies suggest that survivors of ARDS who received more than 24 hours of an inspired oxygen concentration >60%, may have worsened lung diffusion capacity at 1 year follow-up.

Absorption Atelectasis

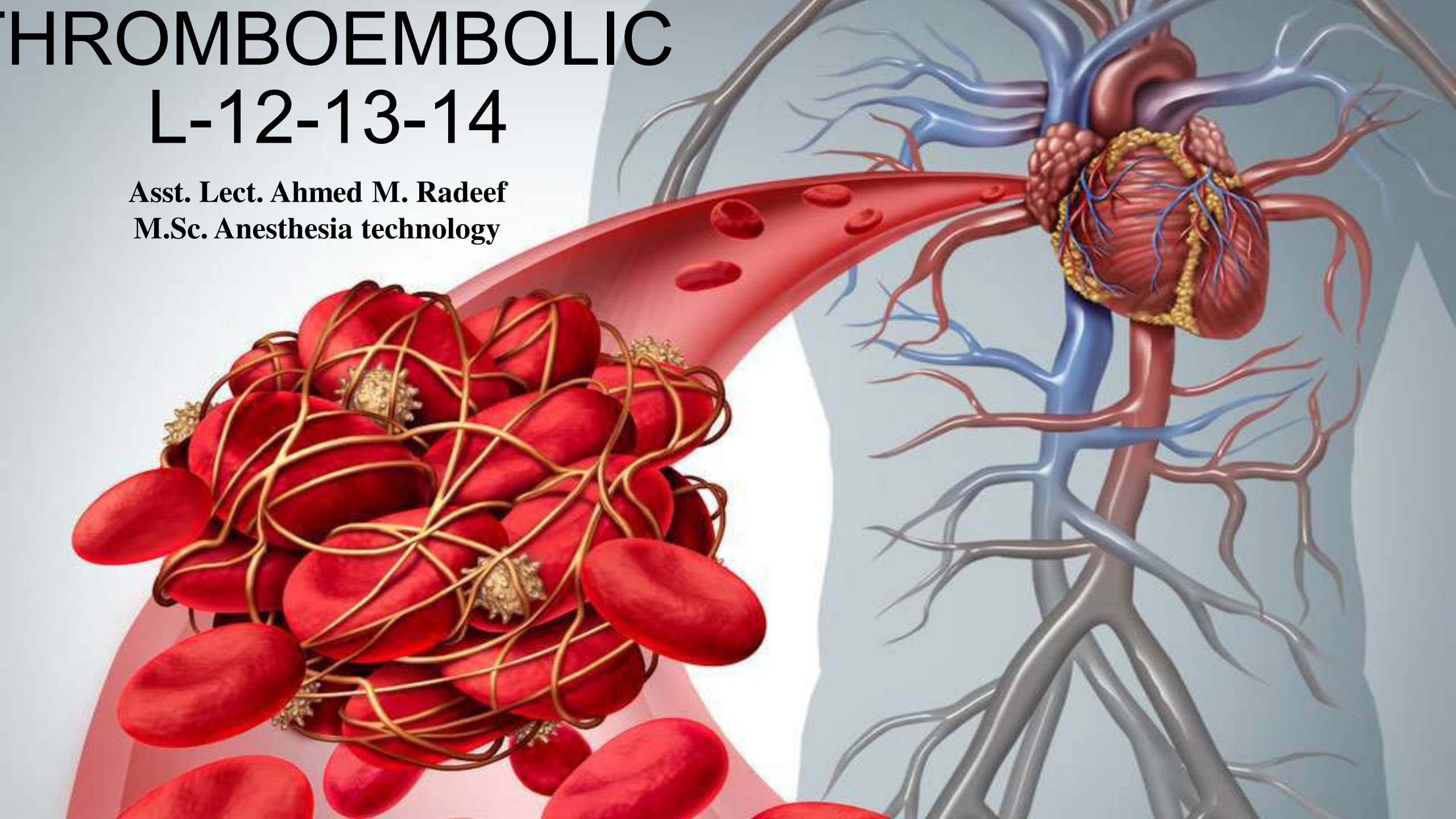
- FiO2 of greater than 0.5 presents a risk of absorption atelectasis, it happens because of denitrogenation. Breathing high level of oxygen quickly depletes the nitrogen level in alveoli and blood. With rapid denitrogenation the total gas pressure in the alveolus progressively decreases.
- In the absence of any repletion this ultimately leads to alveolar collapse. Absorption atelectasis also increases physiologic shunt and worsen blood oxygenation.
- Conditions like sedation, surgical pain and CNS dysfunction give rise to more rapid collapse as compared to normal lung.

THANK YOU FOR ATTENTION!

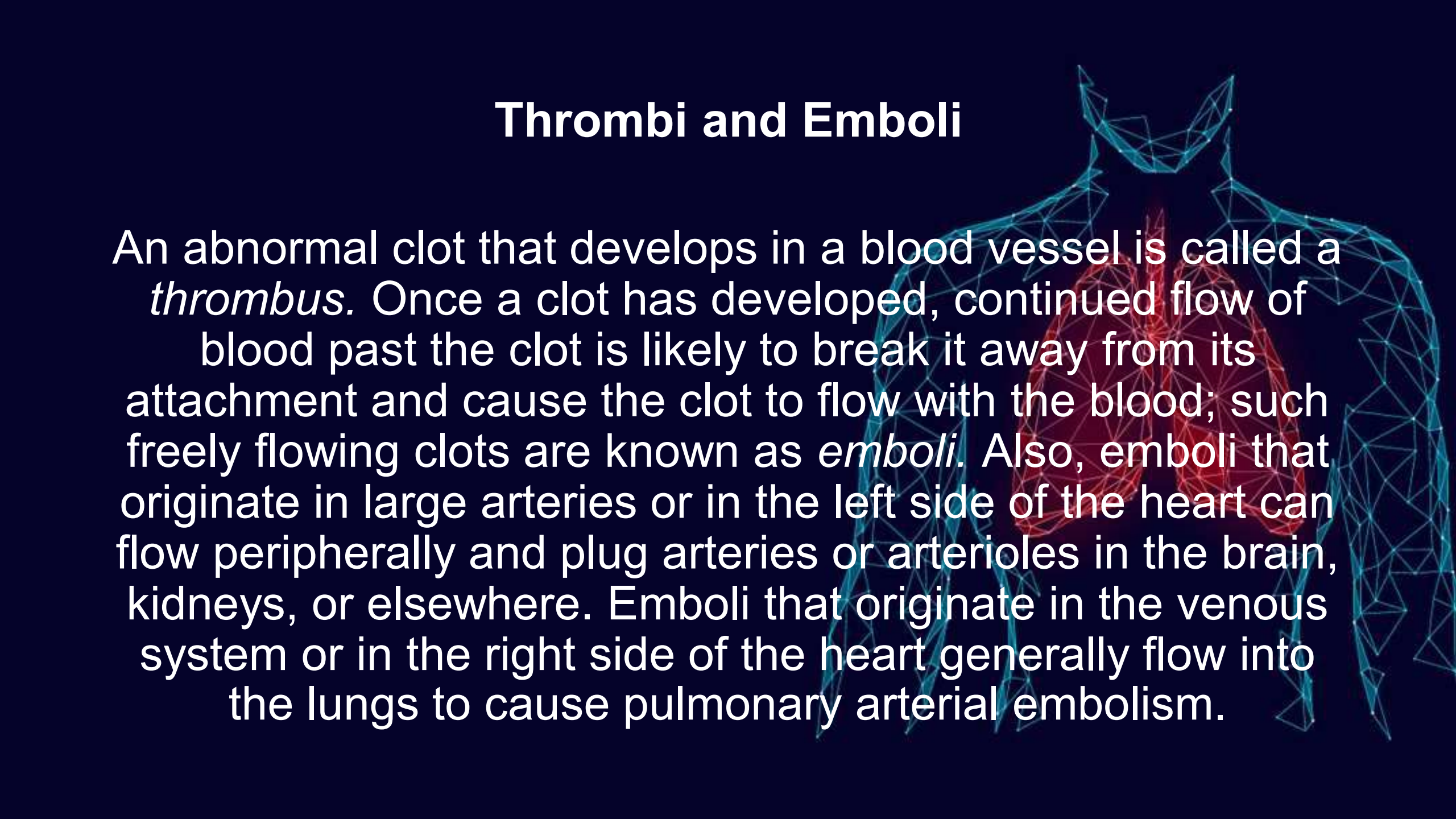
Have a nice day!!!

THROMBOEMBOLIC
L-12-13-14

Asst. Lect. Ahmed M. Radeef
M.Sc. Anesthesia technology



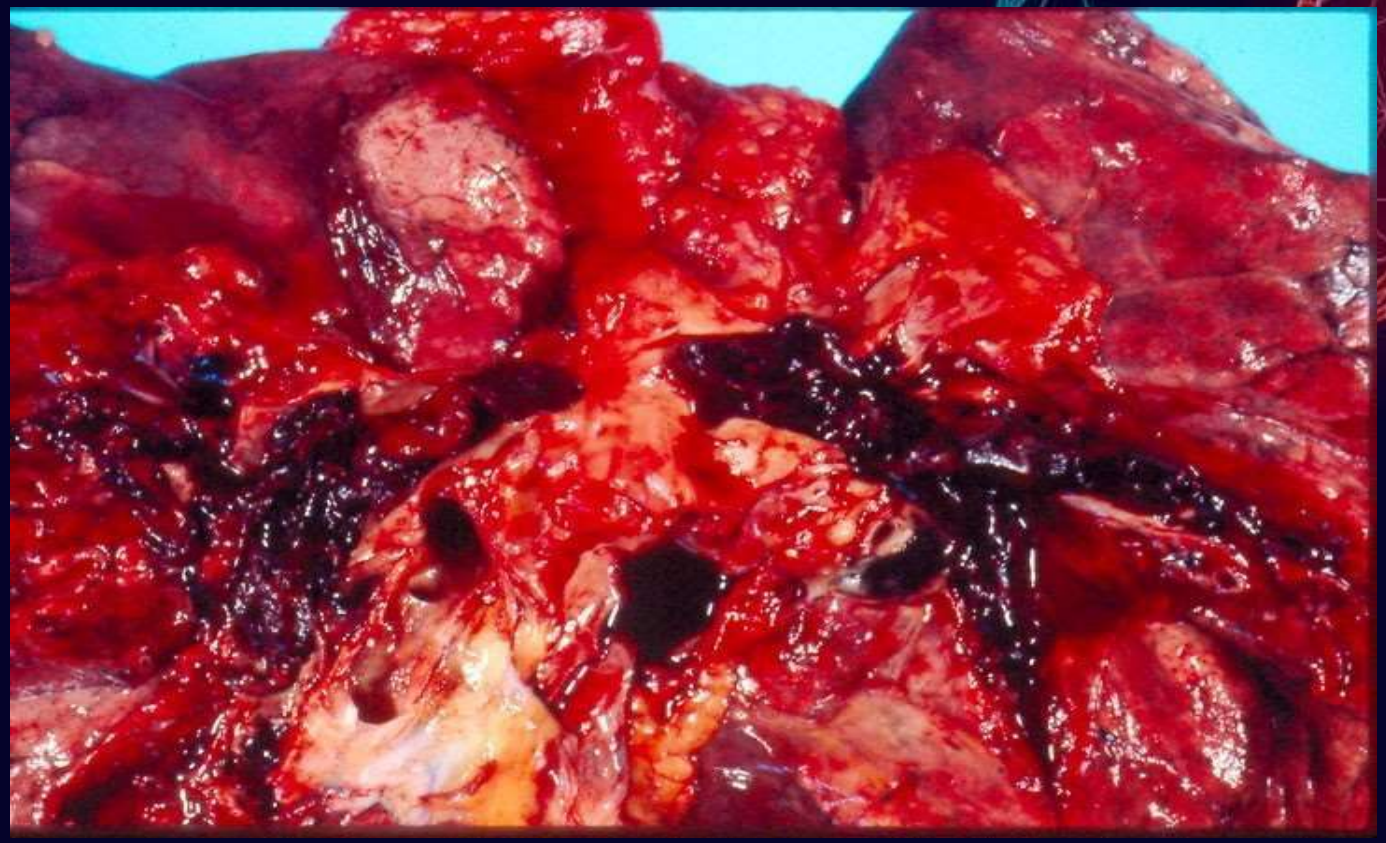
Thrombi and Emboli



An abnormal clot that develops in a blood vessel is called a *thrombus.* Once a clot has developed, continued flow of blood past the clot is likely to break it away from its attachment and cause the clot to flow with the blood; such freely flowing clots are known as *emboli.* Also, emboli that originate in large arteries or in the left side of the heart can flow peripherally and plug arteries or arterioles in the brain, kidneys, or elsewhere. Emboli that originate in the venous system or in the right side of the heart generally flow into the lungs to cause pulmonary arterial embolism.

Venous Thromboembolism in ICU

Pathophysiology of Thrombosis

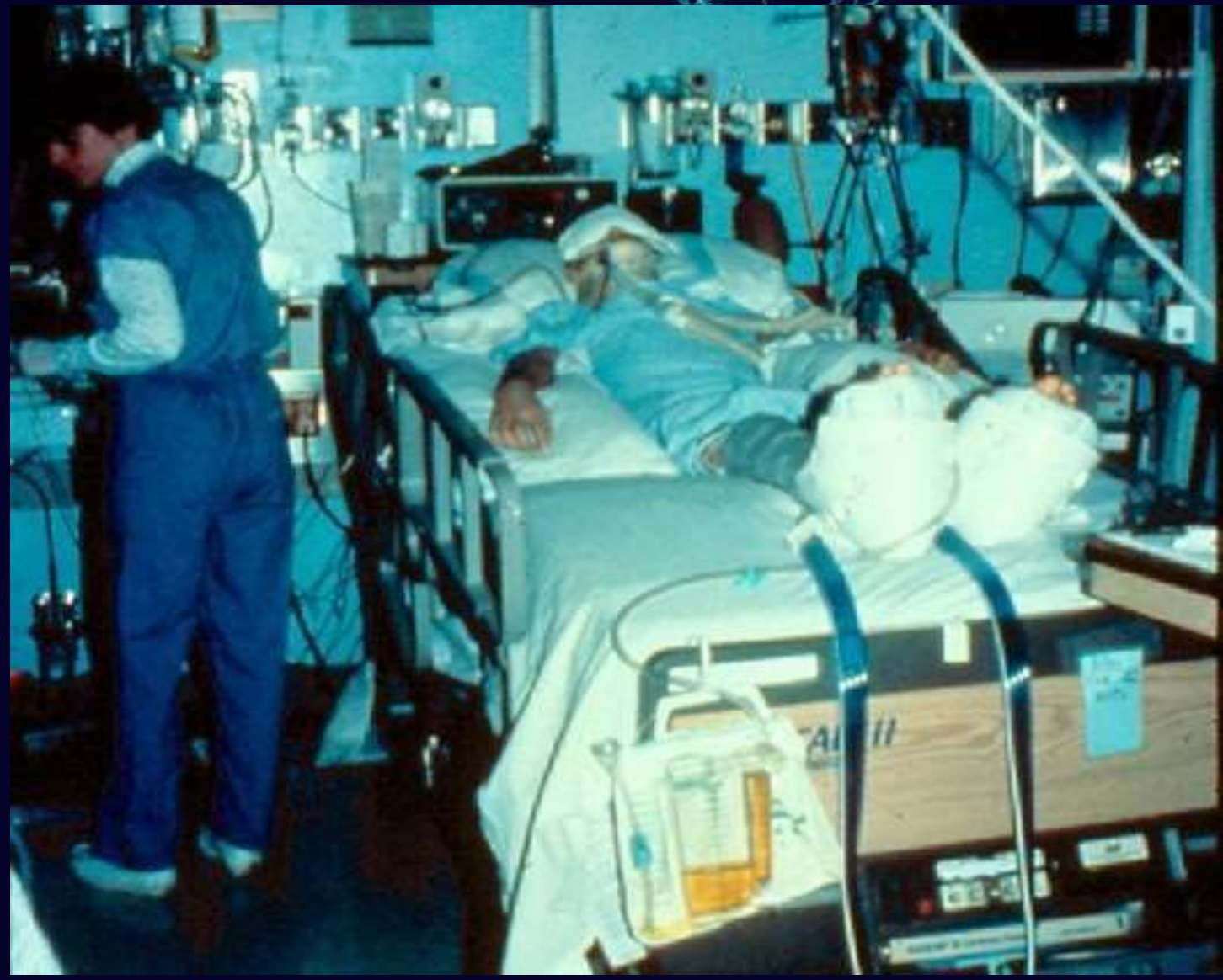


Deep venous thrombosis usually arises in the **lower extremities**. Most DVTs form in the **calf veins**.

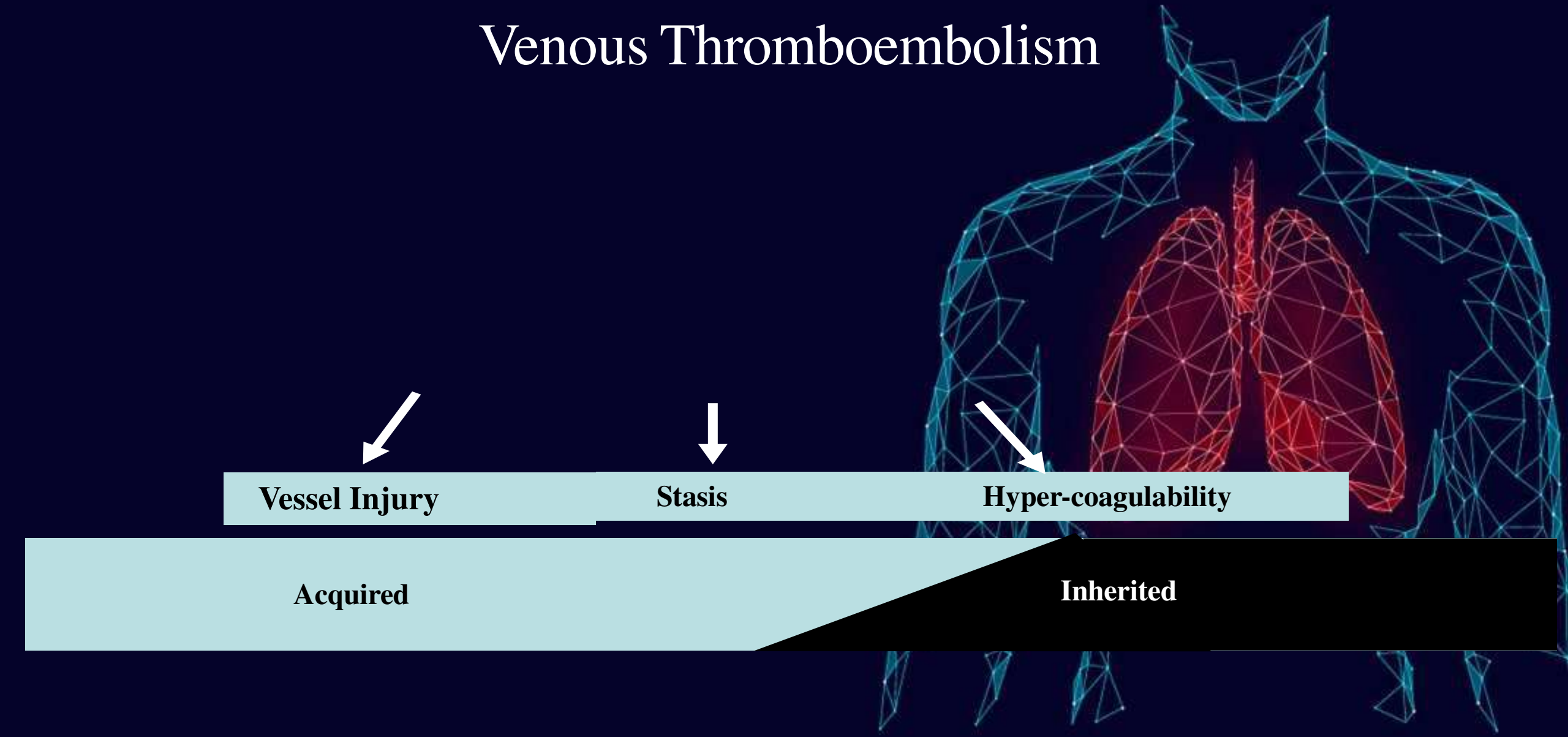
- **Venous valves** are avascular, which, in conjunction with reduced flow of oxygenated blood in veins, predisposes the endothelium to be **hypoxemic**. The endothelium around valves responds by expressing adhesion molecules that attract leukocytes. These cells transfer **tissue factor** to the endothelium, which can complex with **activated factor VII** to begin the coagulation cascade via the extrinsic pathway. The main component of these venous thrombi is **fibrin** (as product of coagulation cascade) and **red blood cells**, which get trapped in the clot. *Platelets* also contribute, but to a lesser extent.
- The **skeletal muscle pump** helps prevent DVT by moving blood past the valves (i.e. reducing venous stasis), which washes away activated clotting factors that can otherwise propagate the initial thrombus.
- If a clot forms and does not resolve, it will extend proximally into the **popliteal and femoral veins** ("proximal veins"). 25% of calf DVTs will extend proximally within 7 days. the calf DVTs are usually asymptomatic and do not give rise to significant PEs, proximal DVTs are more likely symptomatic and can embolize to form dangerous PEs.

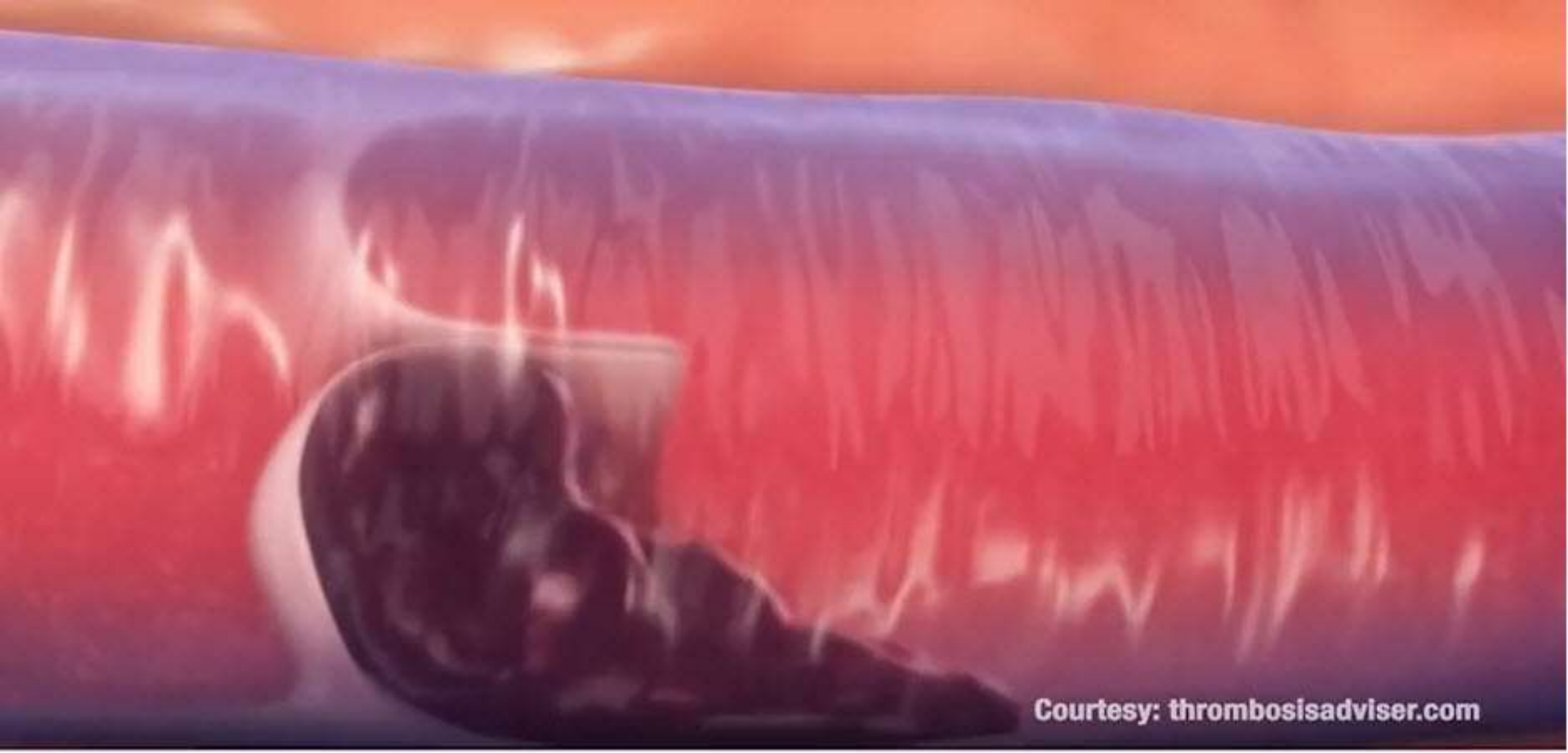
Over a century ago, Rudolf **Virchow** described 3 factors that are critically important in the development of venous thrombosis:

- **Hypercoagulable**
- **Stasis**
- **Vessel Damage**



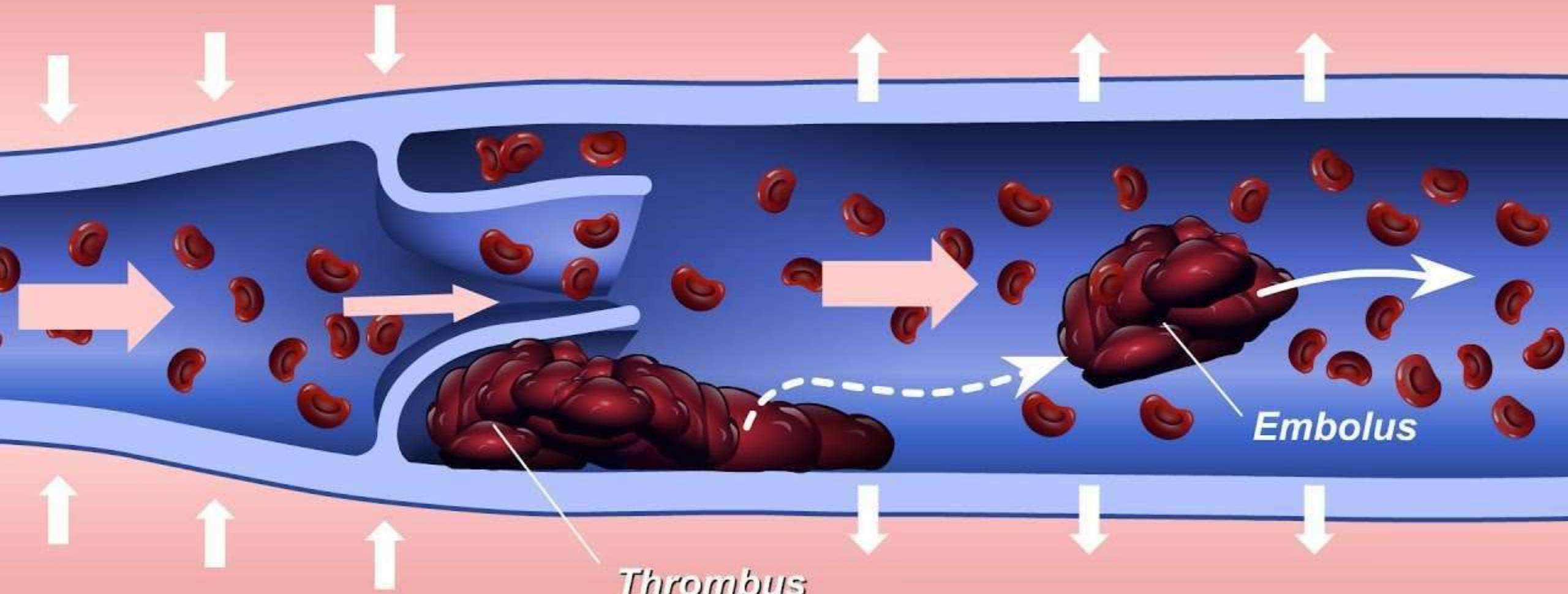
Venous Thromboembolism





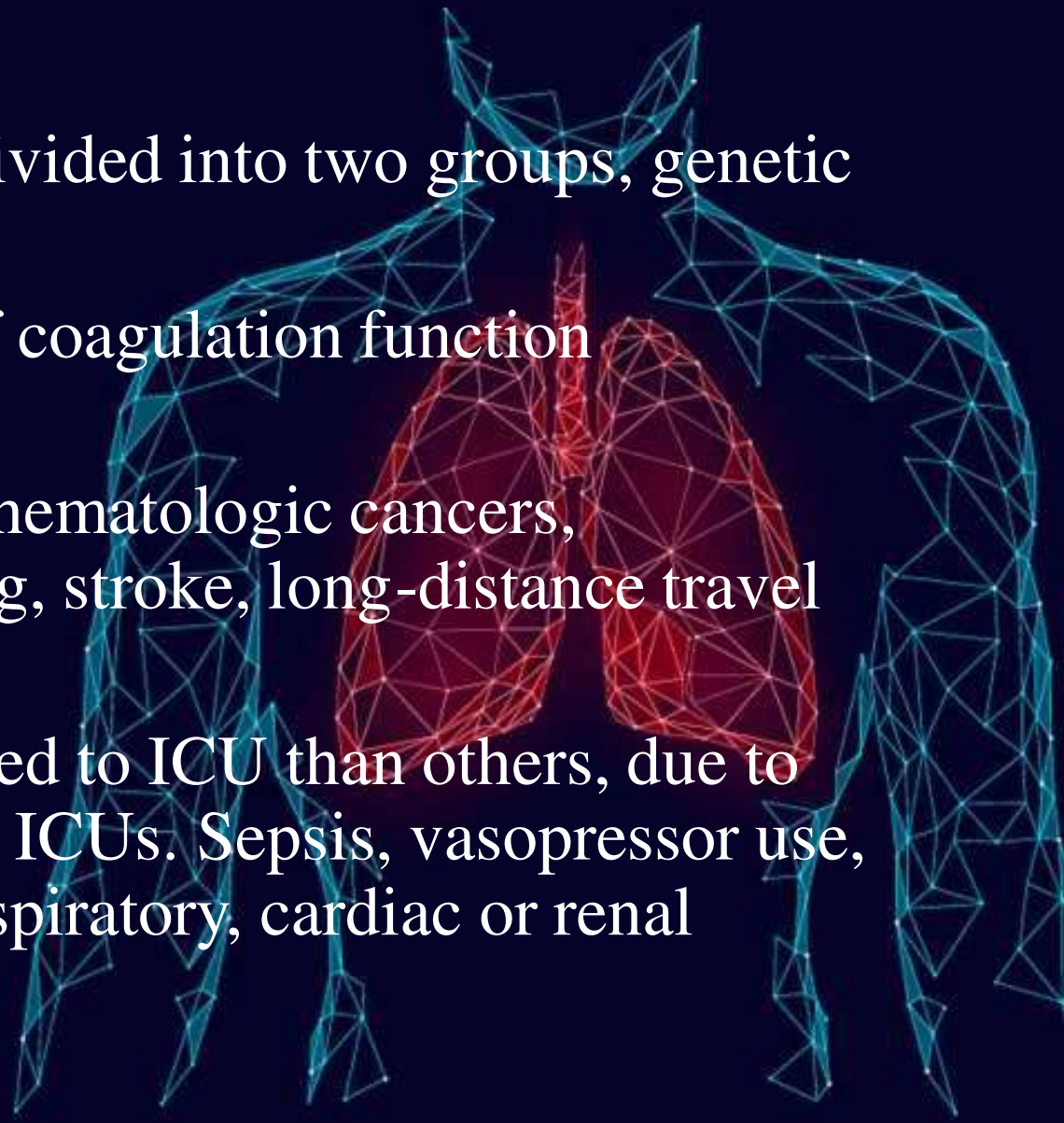
Venous Thromboembolism (VTE)

What is venous thromboembolism (VTE)?



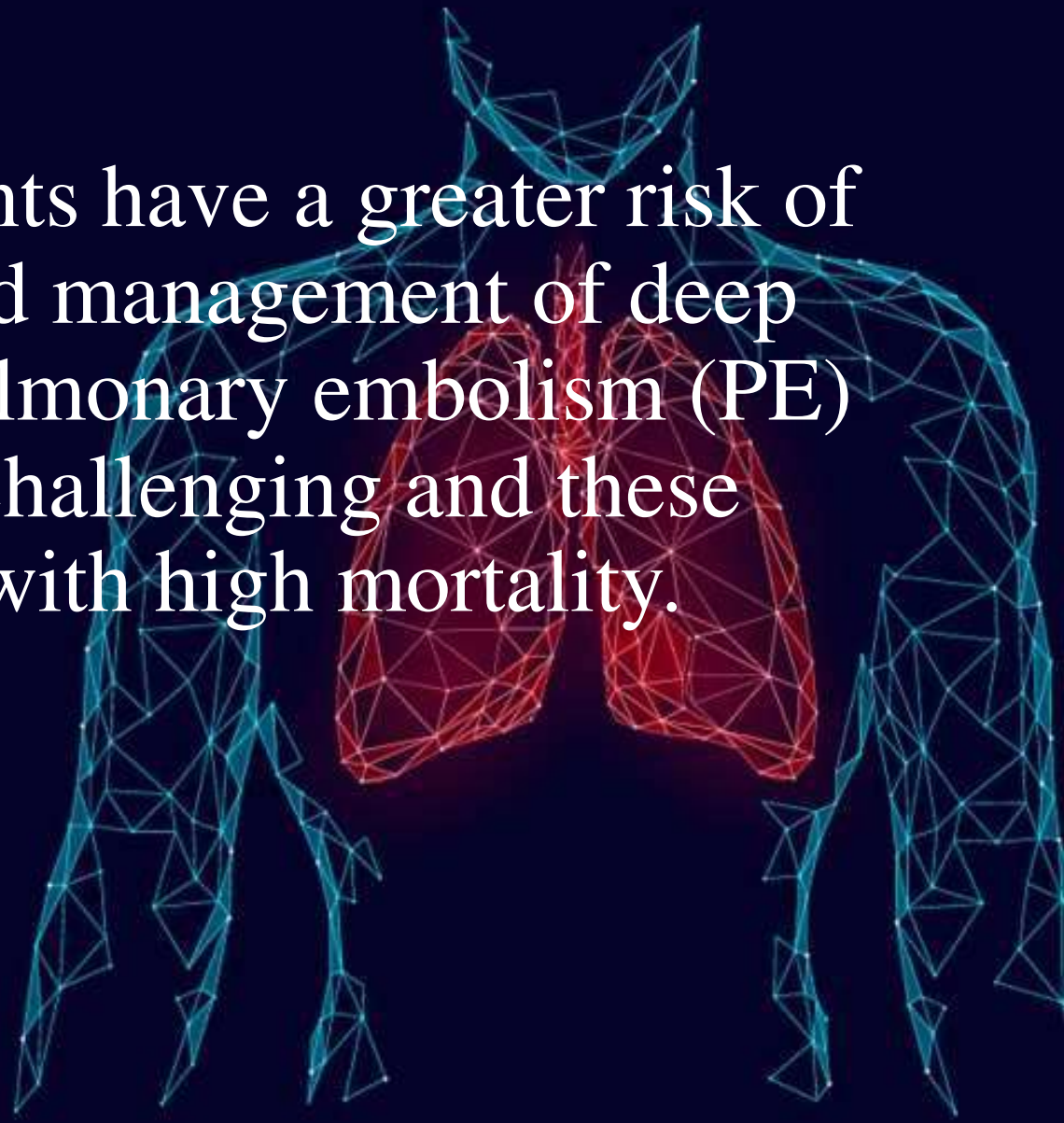
VENOUS THROMBOSIS

Risk factors

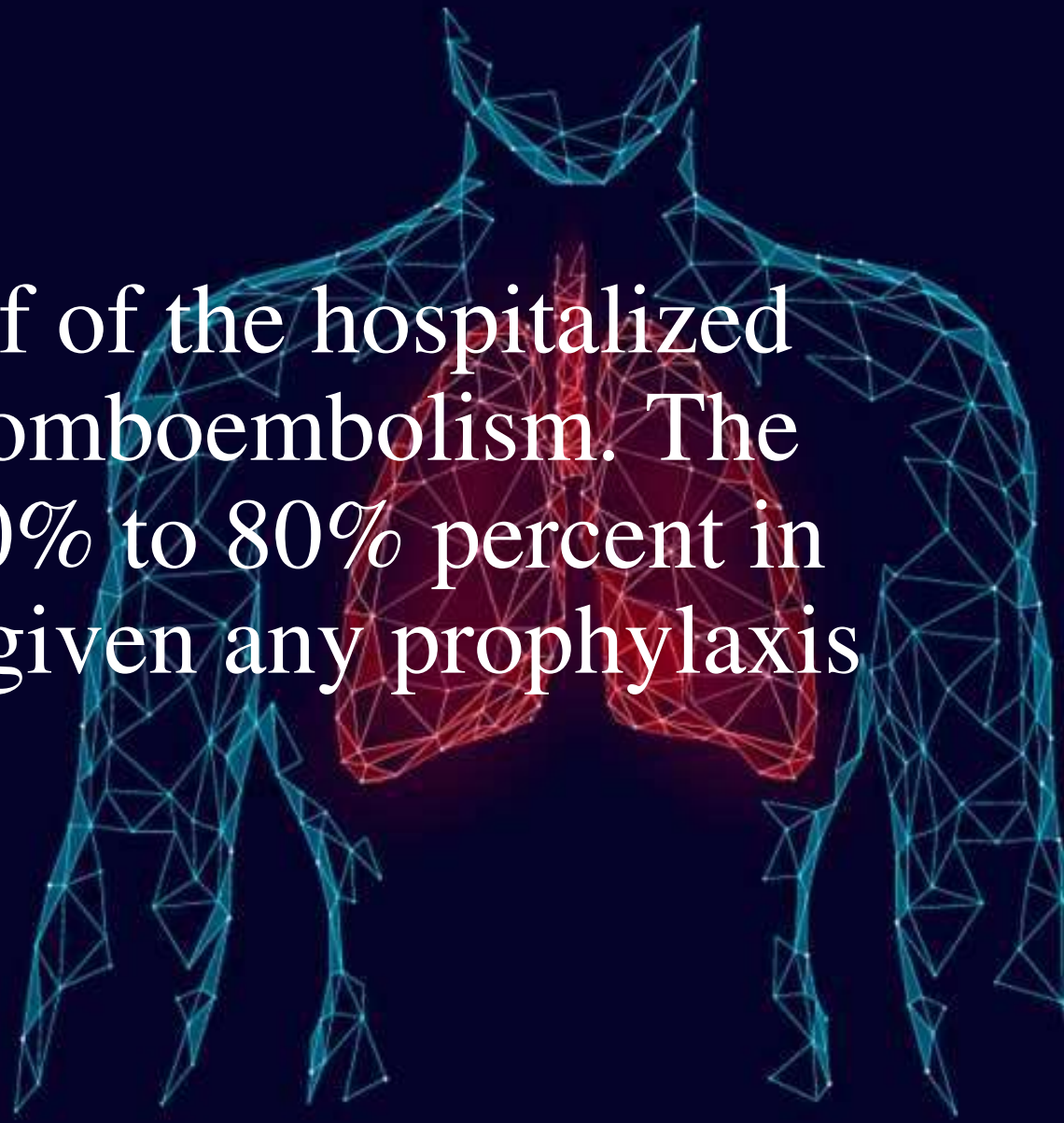


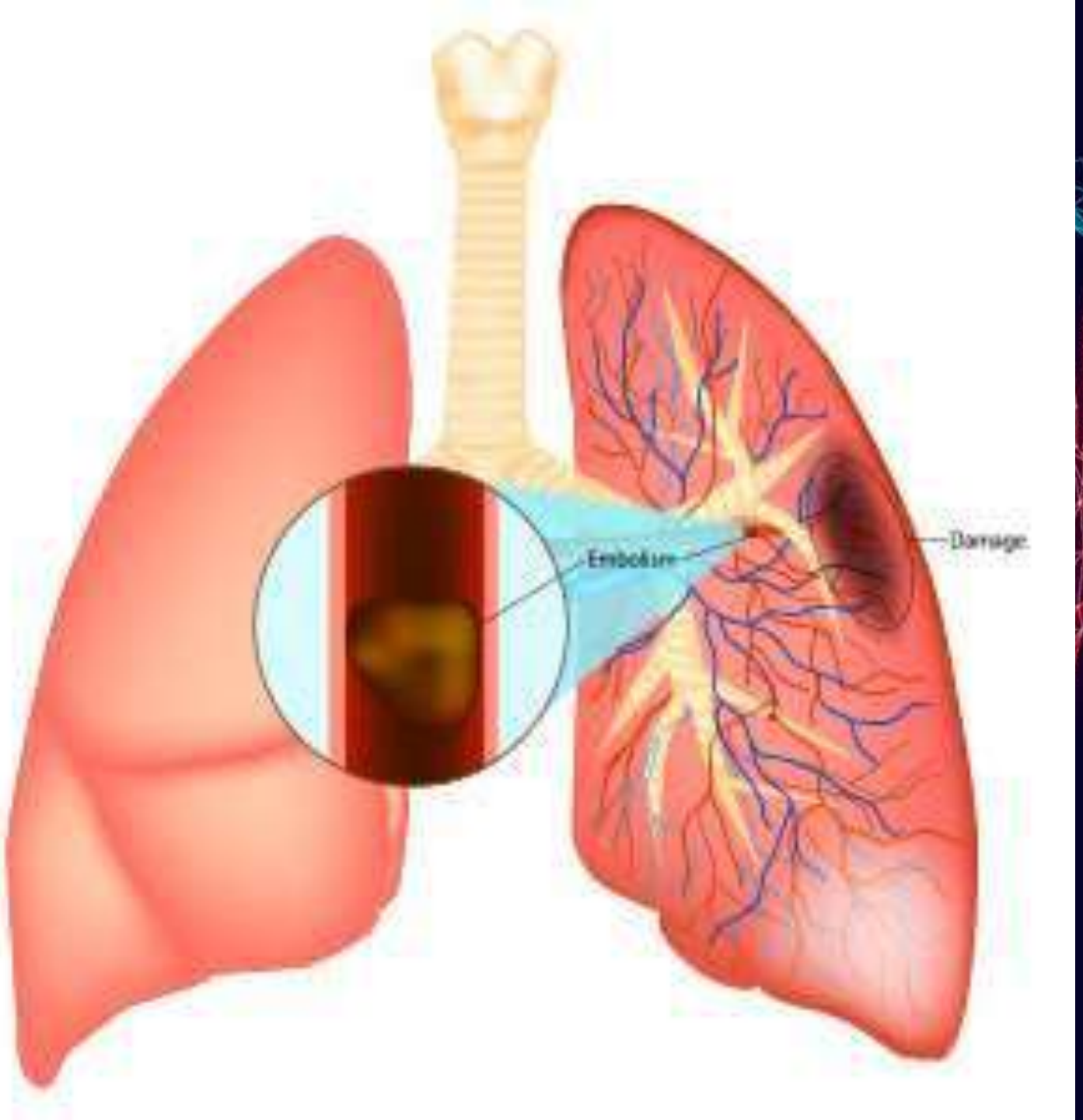
- Risk factors for thromboembolism can be divided into two groups, genetic and acquired.
- Genetic risk factors include loss and gain of coagulation function disorders.
- Acquired risk factors include bed rest, age, hematologic cancers, immobilization, obesity, pregnancy, smoking, stroke, long-distance travel and certain inflammatory conditions
- The risk of VTE is higher in patients admitted to ICU than others, due to the higher number of risk factors specific to ICUs. Sepsis, vasopressor use, central catheters, mechanical ventilators, respiratory, cardiac or renal failure are common ICU.

Intensive care unit (ICU) patients have a greater risk of thrombotic. The diagnosis and management of deep vein thrombosis (DVT) and pulmonary embolism (PE) in critically ill patients are challenging and these conditions are associated with high mortality.



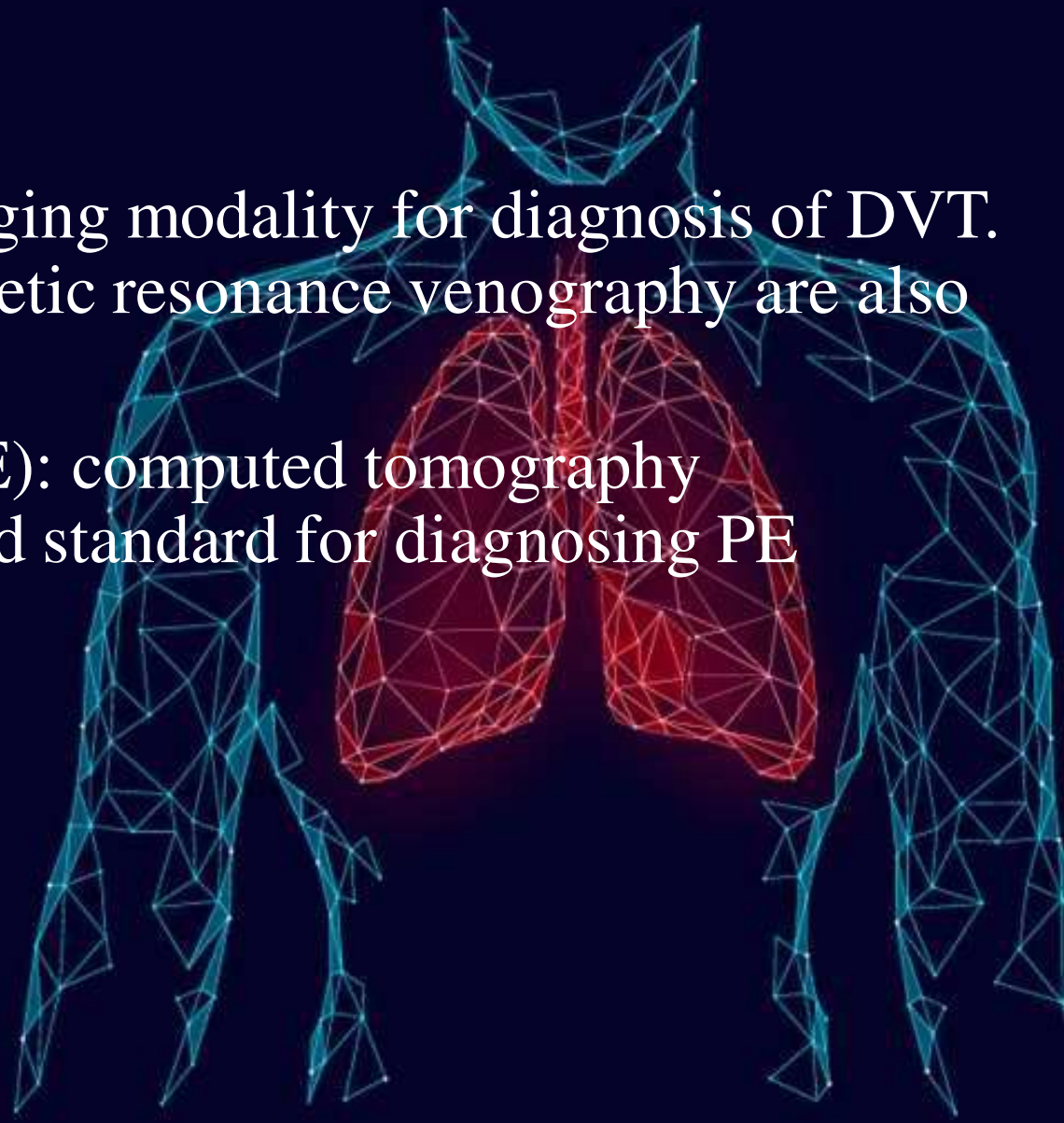
According to research, half of the hospitalized patients are at a risk of thromboembolism. The rate of VTE ranges from 10% to 80% percent in patients, who are not being given any prophylaxis

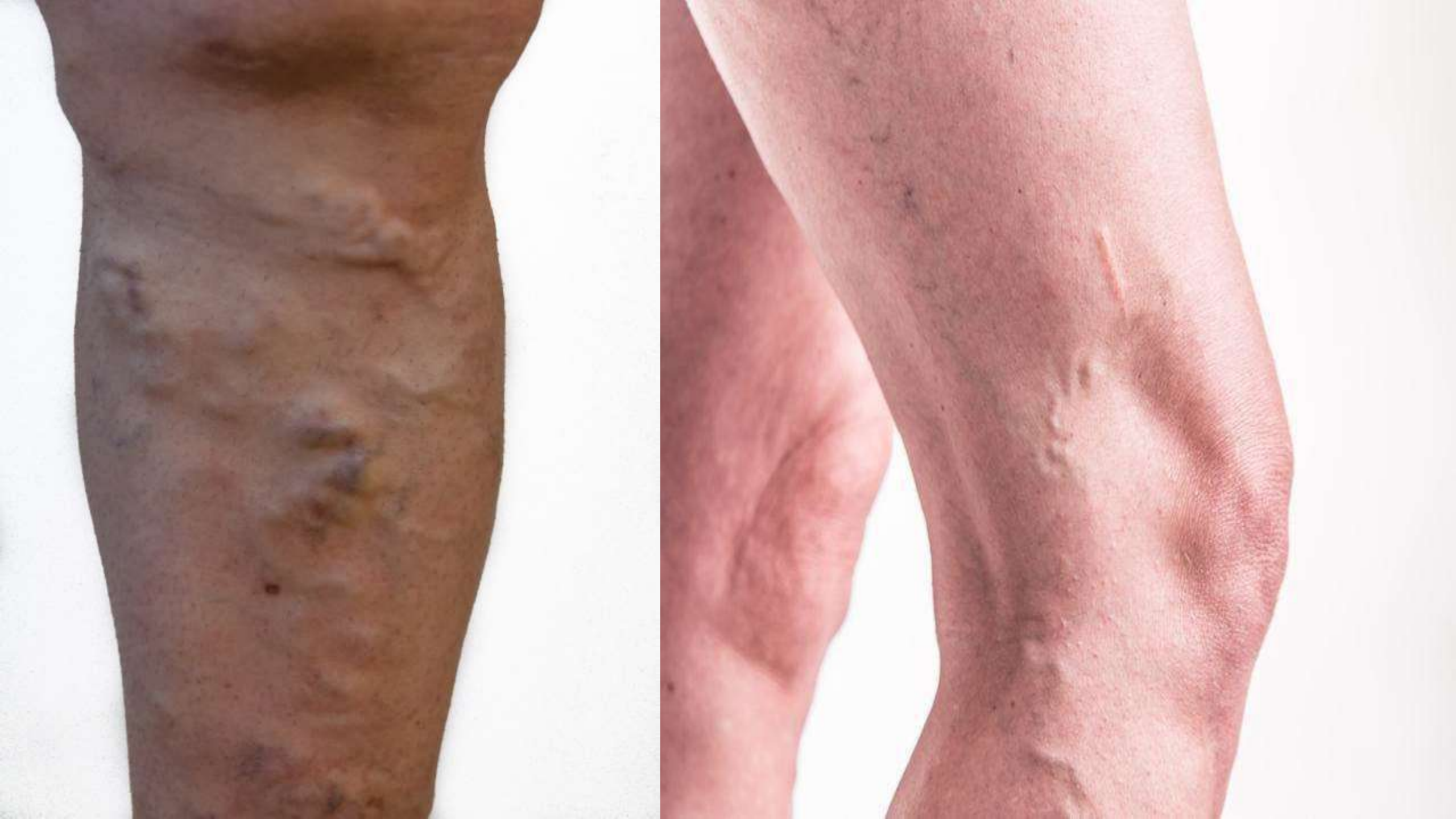



Embolism
Damage

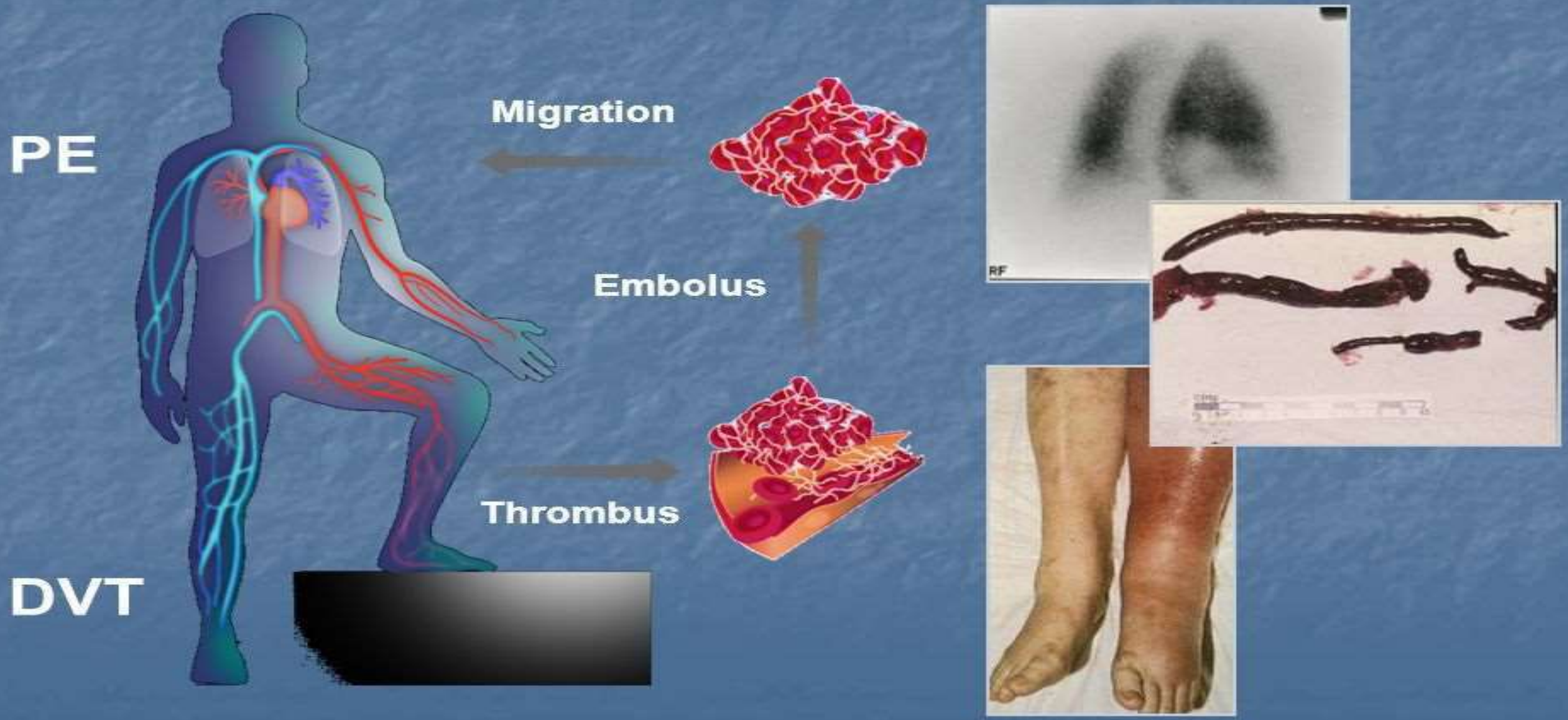
diagnosis

- Doppler venous ultrasound is the best imaging modality for diagnosis of DVT. Others like contrast venography and magnetic resonance venography are also being use, but they have limitations
- The diagnosis of pulmonary embolism (PE): computed tomography pulmonary angiography (CTPA) is the gold standard for diagnosing PE





VTE - deep vein thrombosis (DVT) & pulmonary embolism (PE)



Thromboprophylaxis

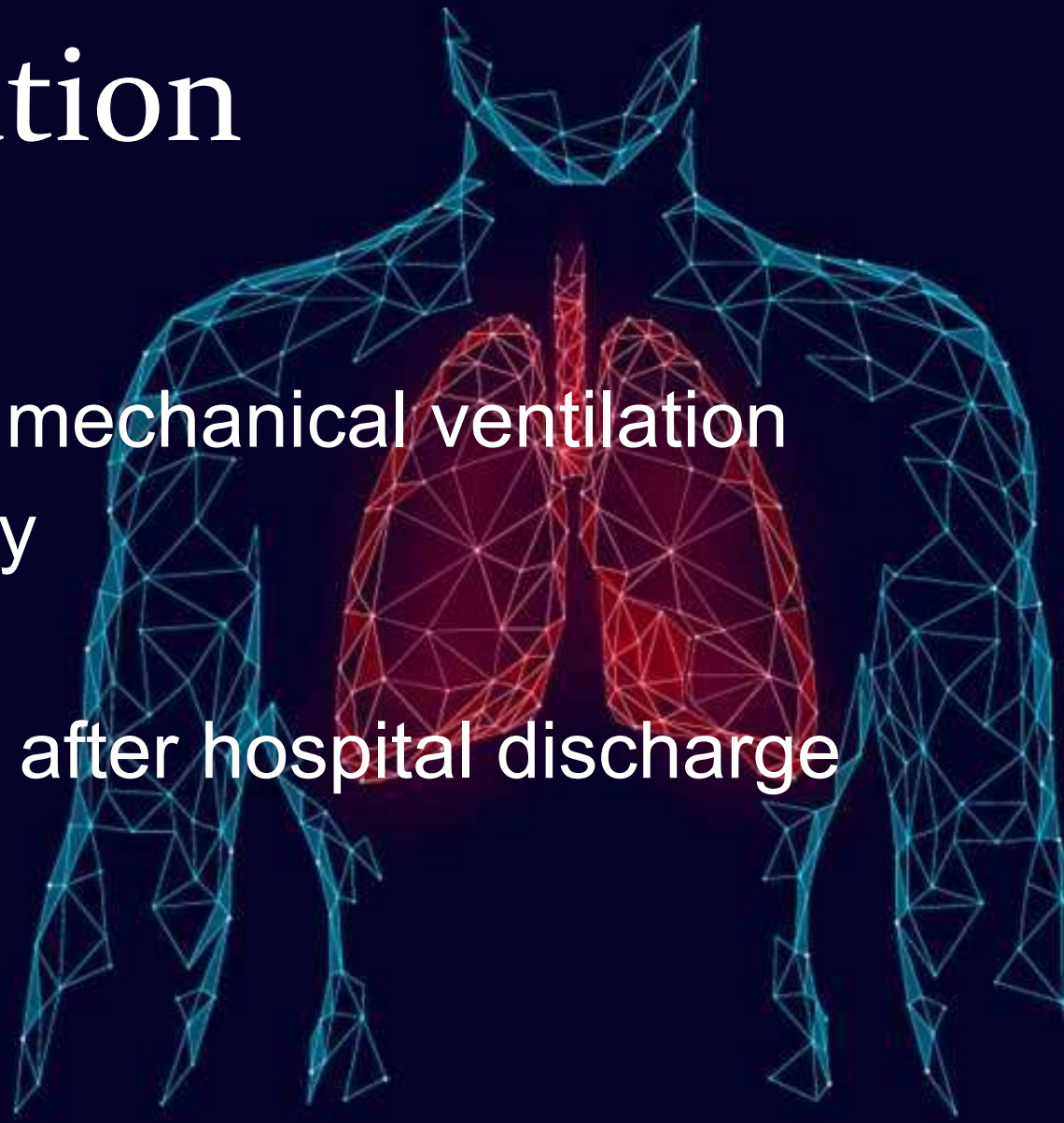
Thromboprophylaxis is of two varieties, primary and secondary. Primary prophylaxis is the one which is given to prevent the occurrence of deep vein thrombosis (DVT), which includes pharmacologic therapy like unfractionated heparin (UFH) (**Heparin**), low molecular weight heparin (LMWH)9 "**Enoxaparin**" (**Clexane**)), or mechanical therapy like pneumatic and graduated compression stockings. Secondary prophylaxis involves early detection and management of venous thrombosis. The method of primary prophylaxis is determined by factors like the risk of thrombosis and hemorrhage, nature of the illness. These factors help to classify patients into low, moderate and high-risk categories, each having a different method of prophylaxis. Moreover, the duration of prophylactic treatment differs from patient to patient depending on the risk classification.


CLEXANE®
4000 anti-Xa IU 0.4 ml
énoxaparine sodique/enoxaparin sodium
4000 UI anti-Xa est équivalent à 40 mg/4000 anti-Xa IU is equivalent to 40 mg
Solution injectable/Injectable solution
Voie sous-cutanée/Subcutaneous route
2 seringues pré-remplies/pre-filled syringes
sanofi aventis

Mobilization

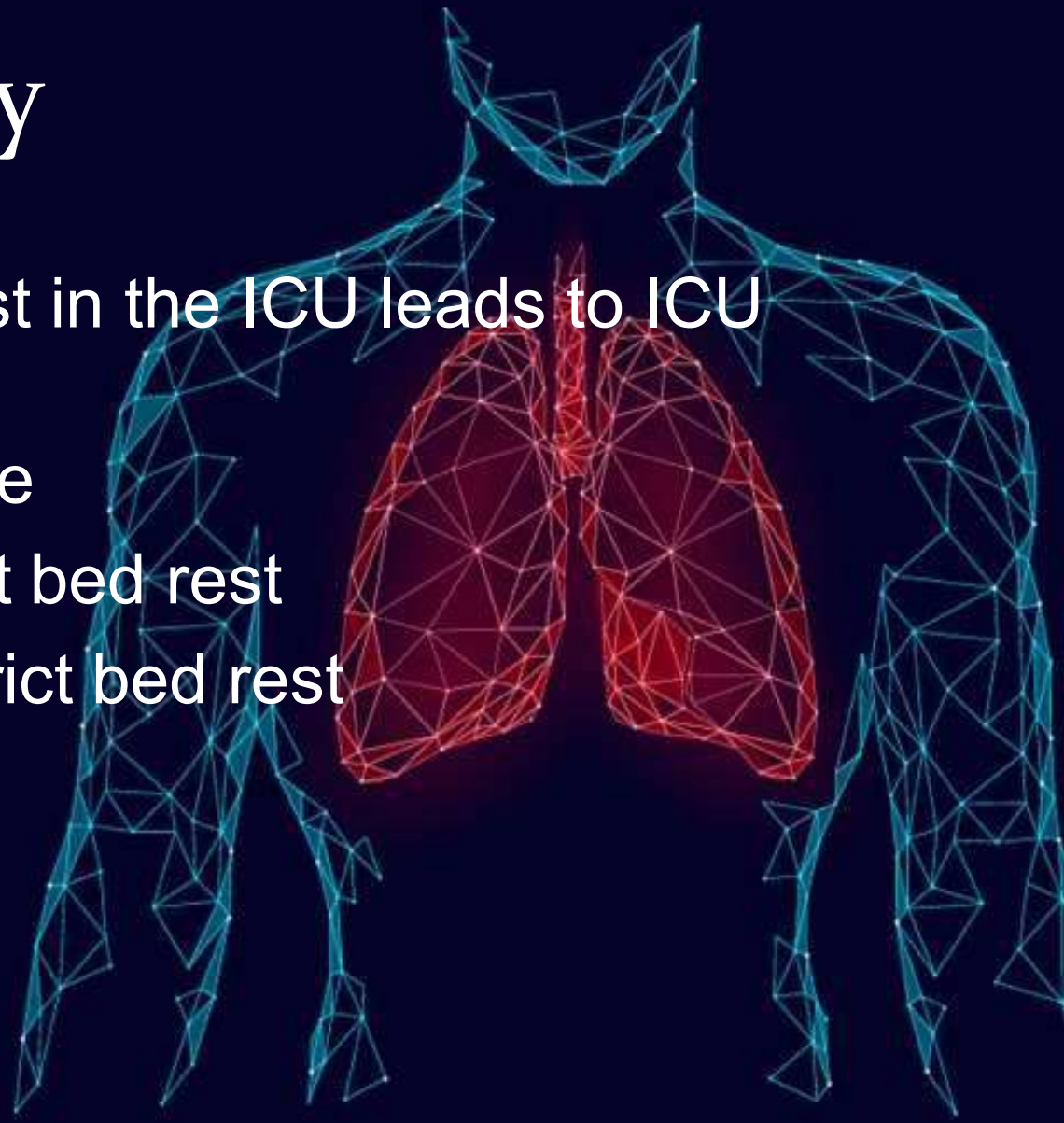
The Effects of Immobility

- Immobility prolongs duration of mechanical ventilation
- Increases hospital length of stay
- Increases mortality
- Immobility can persist for years after hospital discharge

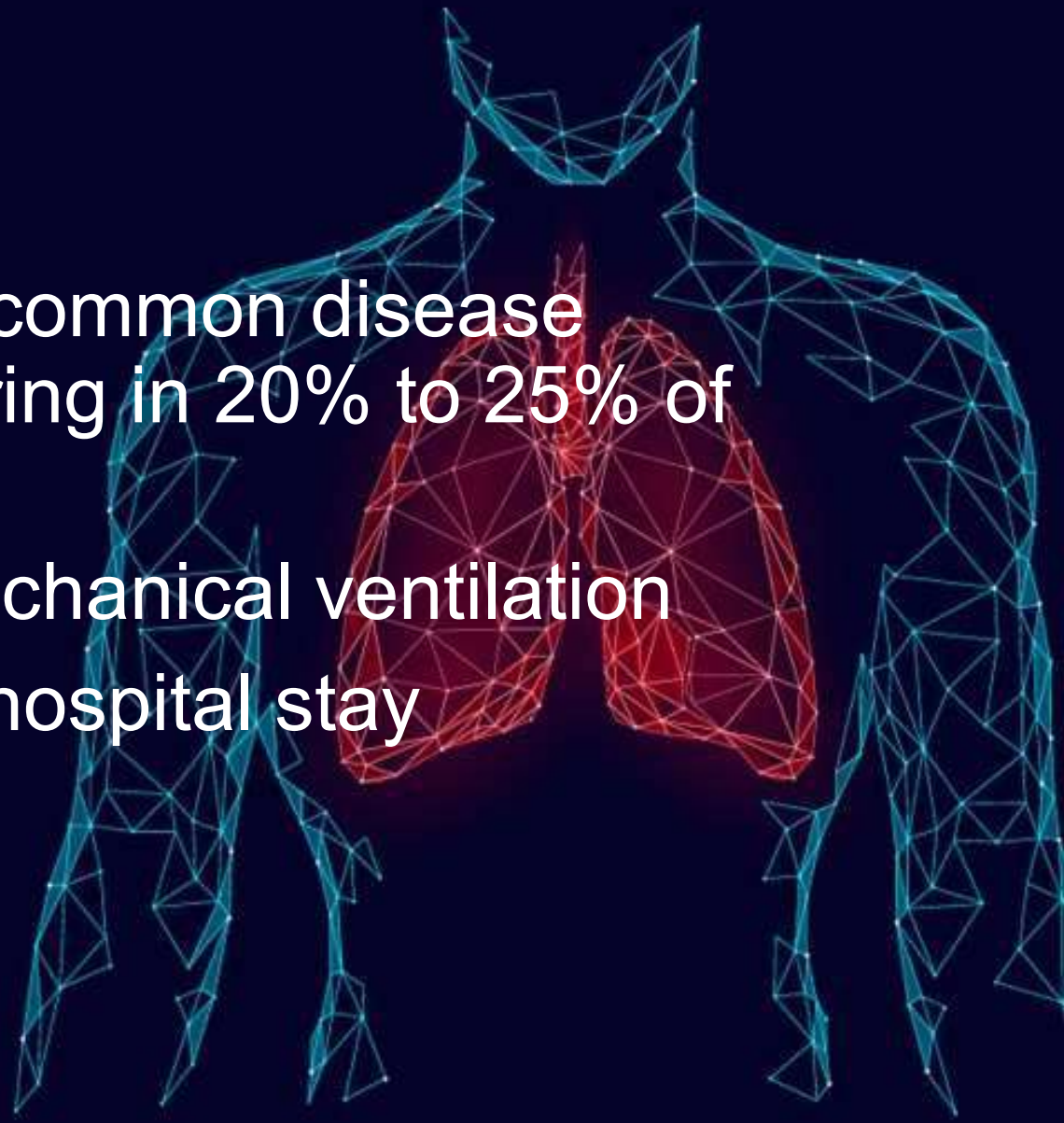


The effects of Immobility

- Immobility due to prolonged bed rest in the ICU leads to ICU acquired-weakness
- Skeletal muscle strength can decline
    - ❑1% to 1.5% per day from strict bed rest
    - ❑4% to 5% for each week of strict bed rest



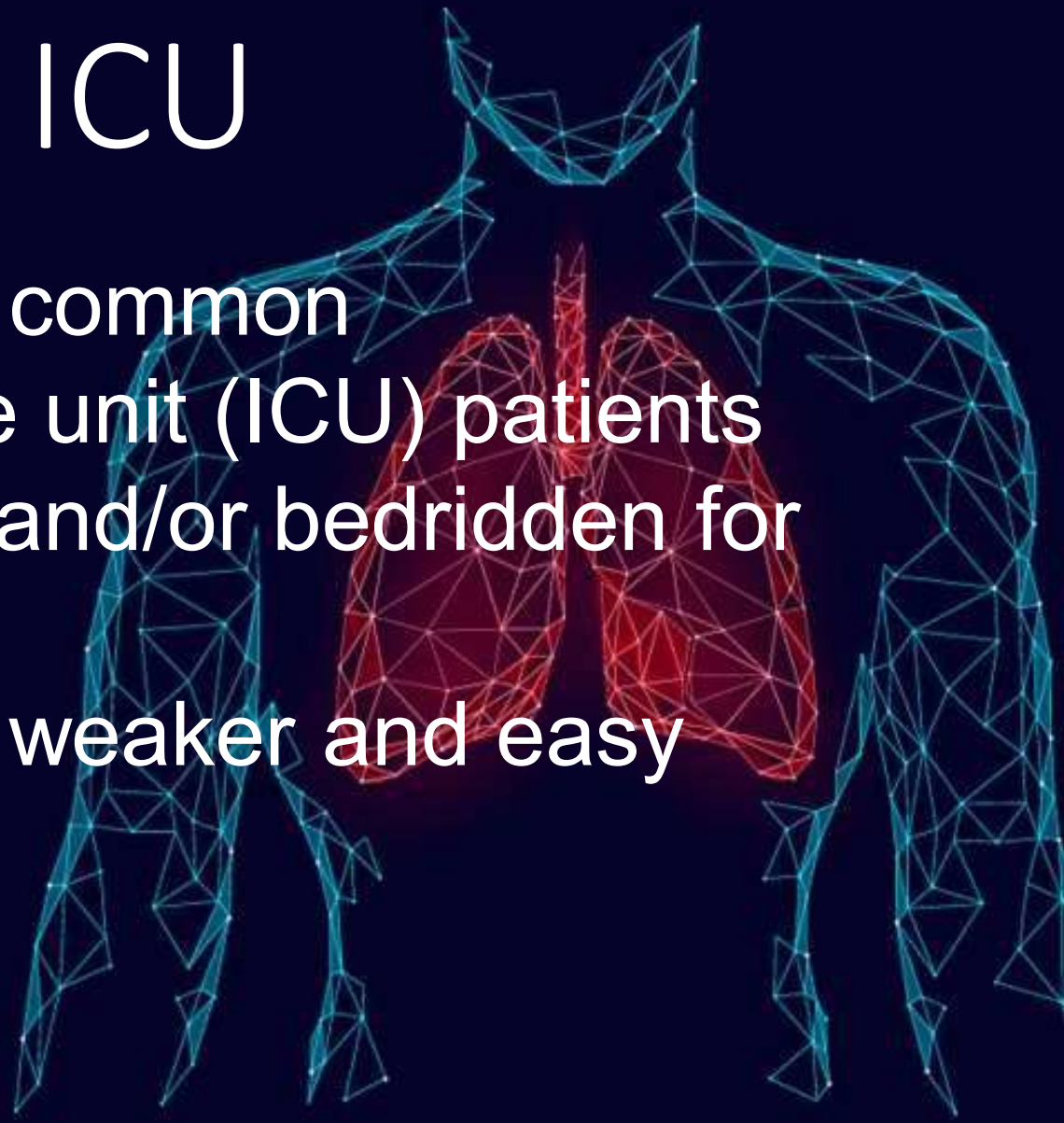
- Neuromuscular weakness is a common disease process of critical illness occurring in 20% to 25% of ICU patients
  - ❑Extends the duration of mechanical ventilation
  - ❑Prolongs the length of the hospital stay
  - ❑Increases mortality

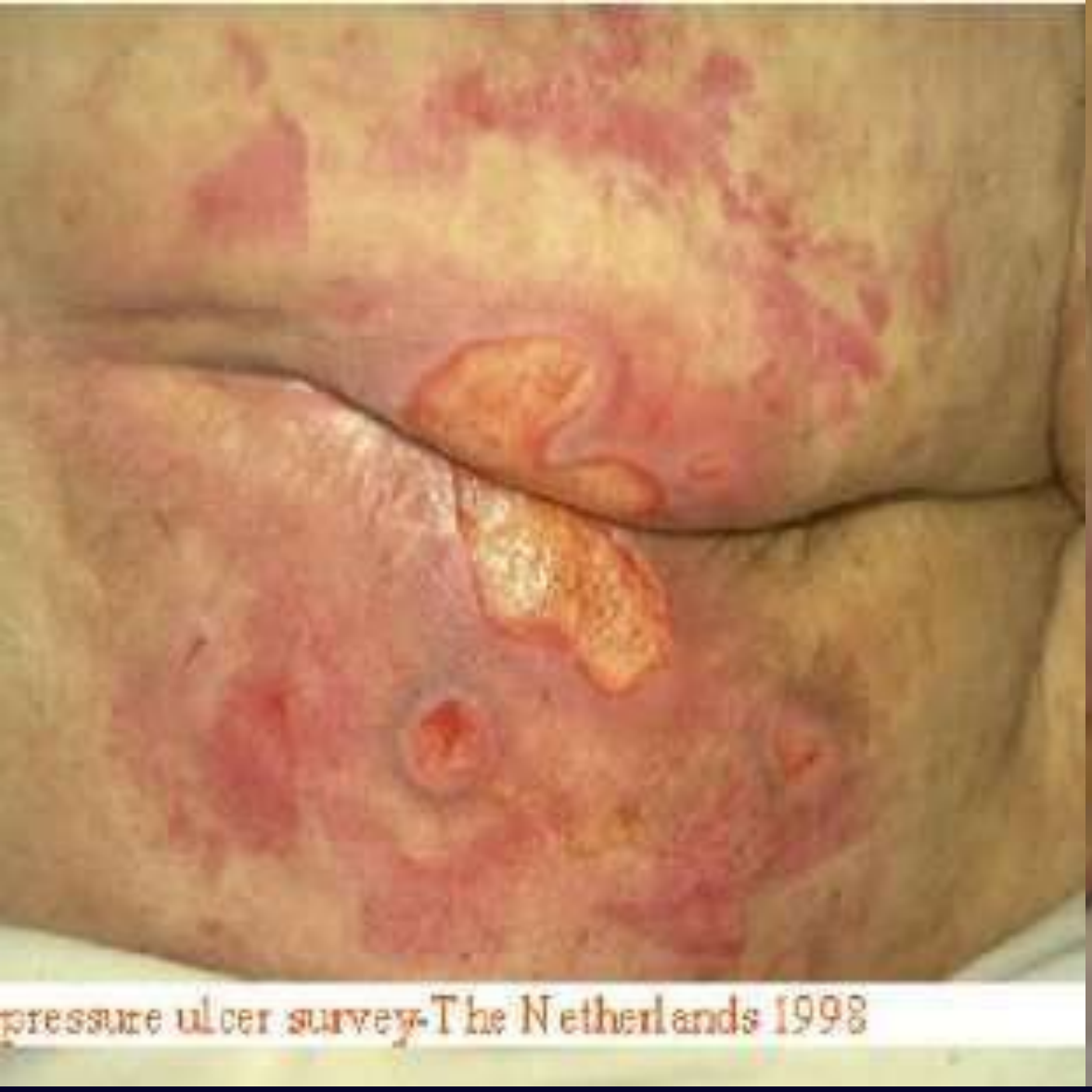


- Prolonged immobilization depresses insulin-induced glucose transport in skeletal muscle and leads to a catabolic state in the affected areas: MUSCLE WASTING

sores in ICU

- Pressure ulcers (PrUs) are a common complication in intensive care unit (ICU) patients who are sedated, ventilated, and/or bedridden for long periods.
- In older adults skin becomes weaker and easy breakdown
- Pressure ulcers are formed




pressure ulcer survey-The Netherlands 1998

Depression and Anxiety

- Patients who are critically ill often develop depression and anxiety symptoms during their stay in ICU.
- Delirium is the most prevalent mental disorder among older patients in the ICU associated with…
  - ❑Poor prognosis
  - ❑Increased length of hospital stay

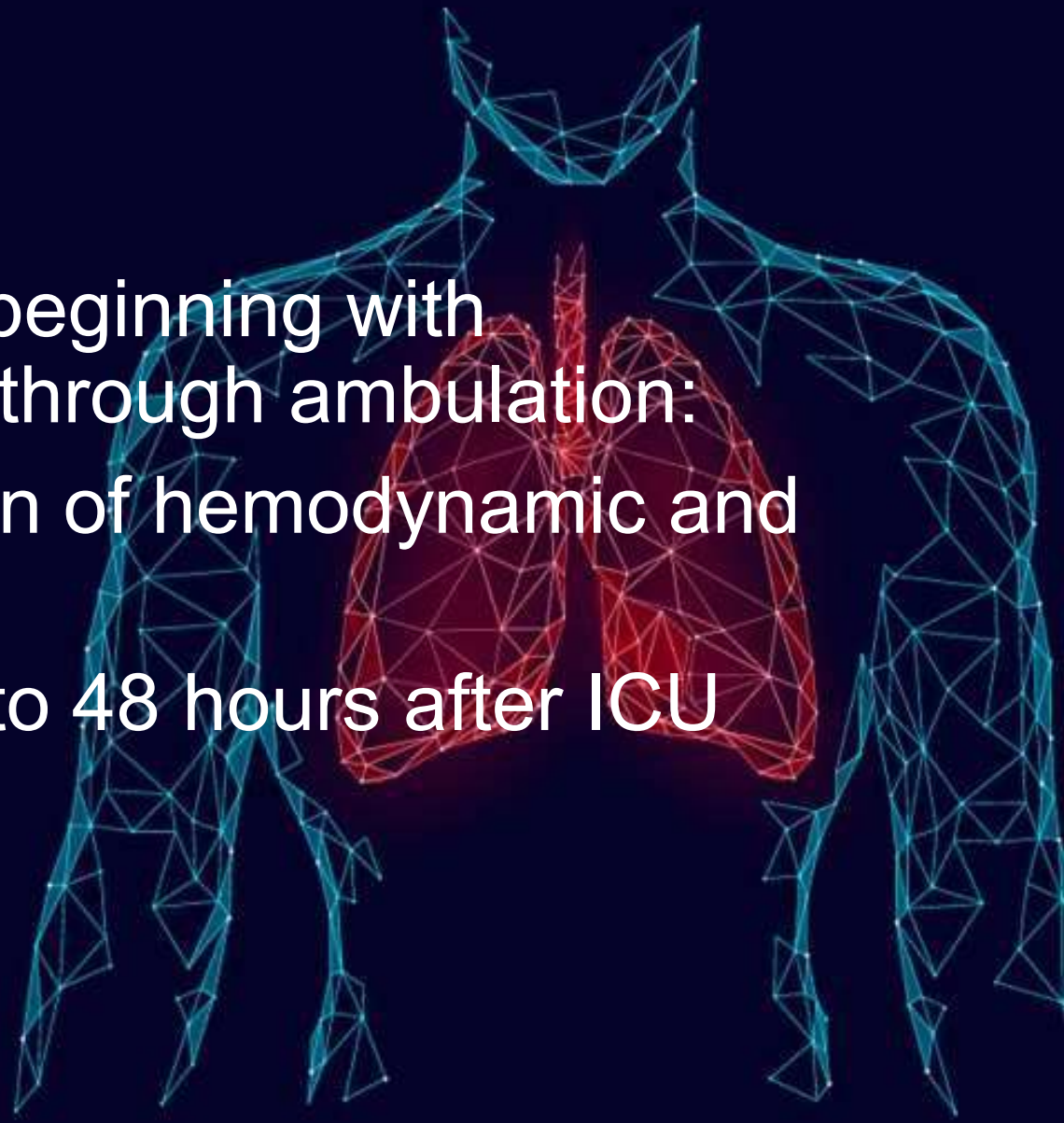
Benefits of Early Mobility

- Safe
- Feasible
- Improves patients ICU outcomes
- Discharge patients home earlier
- Decreases neuromuscular weakness
- Decreases bed sores
- Decreases anxiety and depression

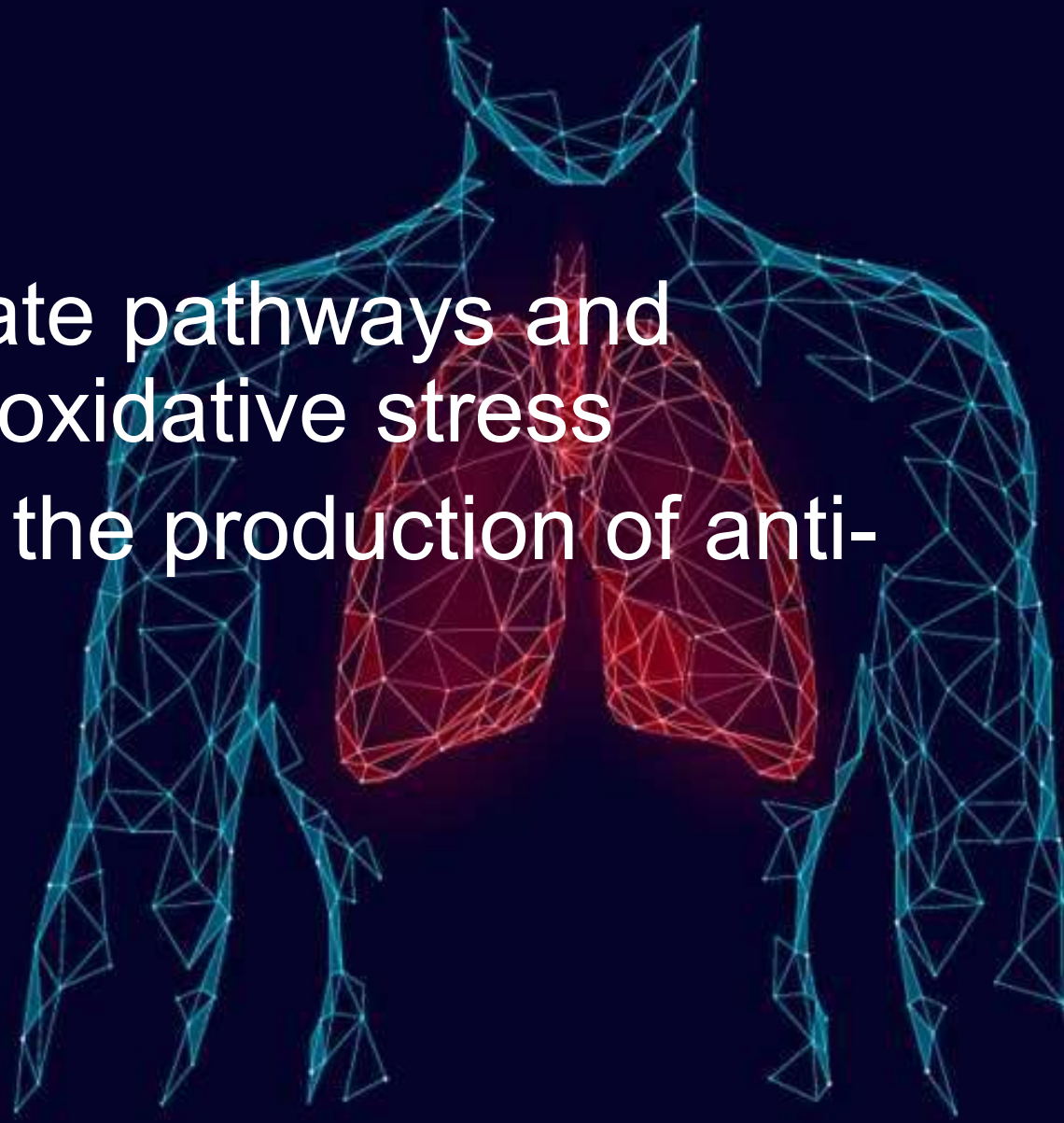
- Provides psychological support for the…
  - ❑ Patient
  - ❑ Family members

Early Mobilization

- A pattern of increasing activity beginning with active/passive range of motion through ambulation:
  - ❑ Begins upon stabilization of hemodynamic and respiratory physiology
  - ❑ Should begin within 24 to 48 hours after ICU admission

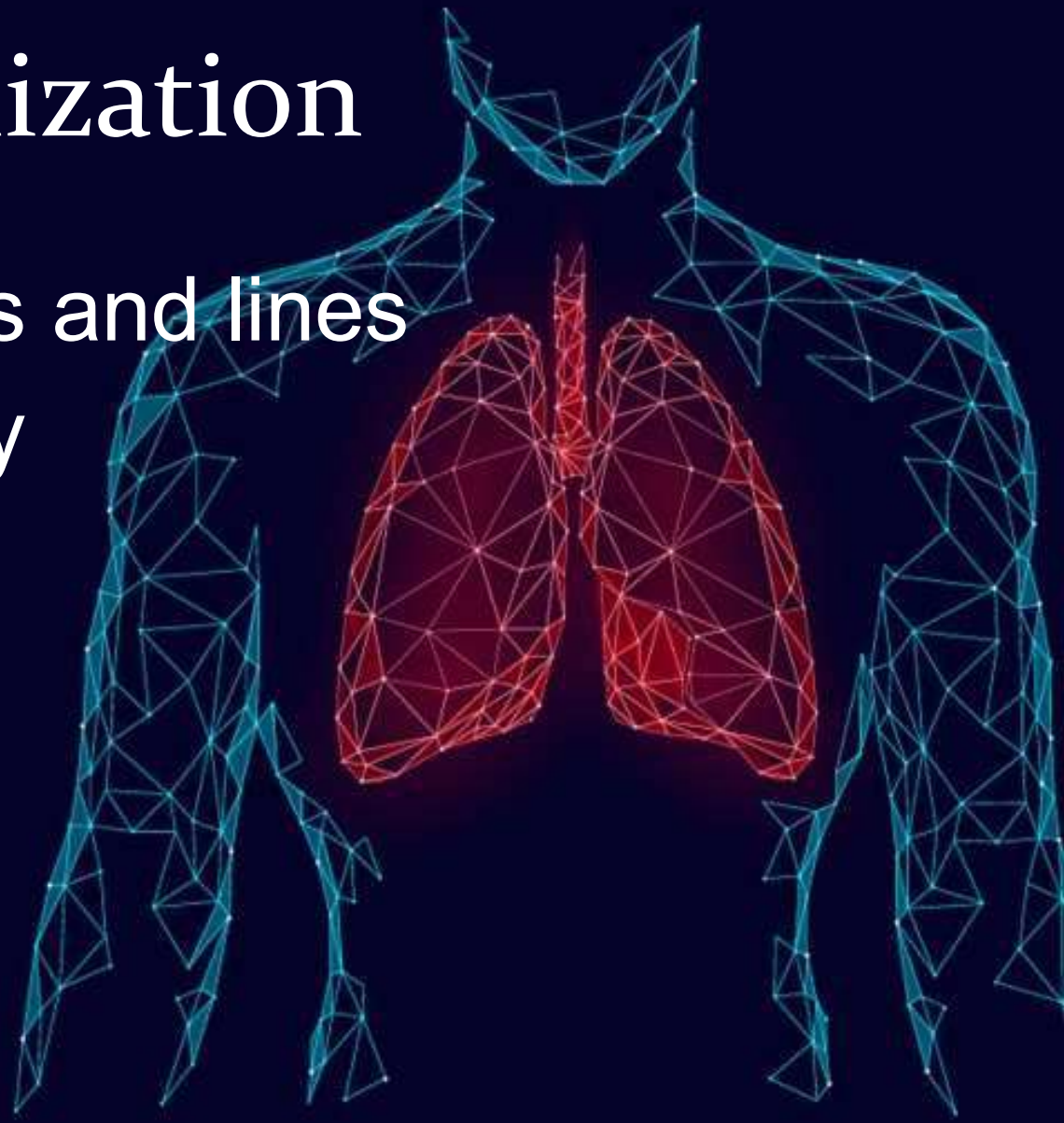


- Physical exercise can activate pathways and increase protection against oxidative stress
- Moderate exercise triggers the production of anti-inflammatory cytokines



Risks with Early Mobilization

- Accidental removal of tubes and lines
- Hemodynamically instability
- Oxygen desaturation
- Accidental extubation
- Patient discomfort
- Limited staff and time



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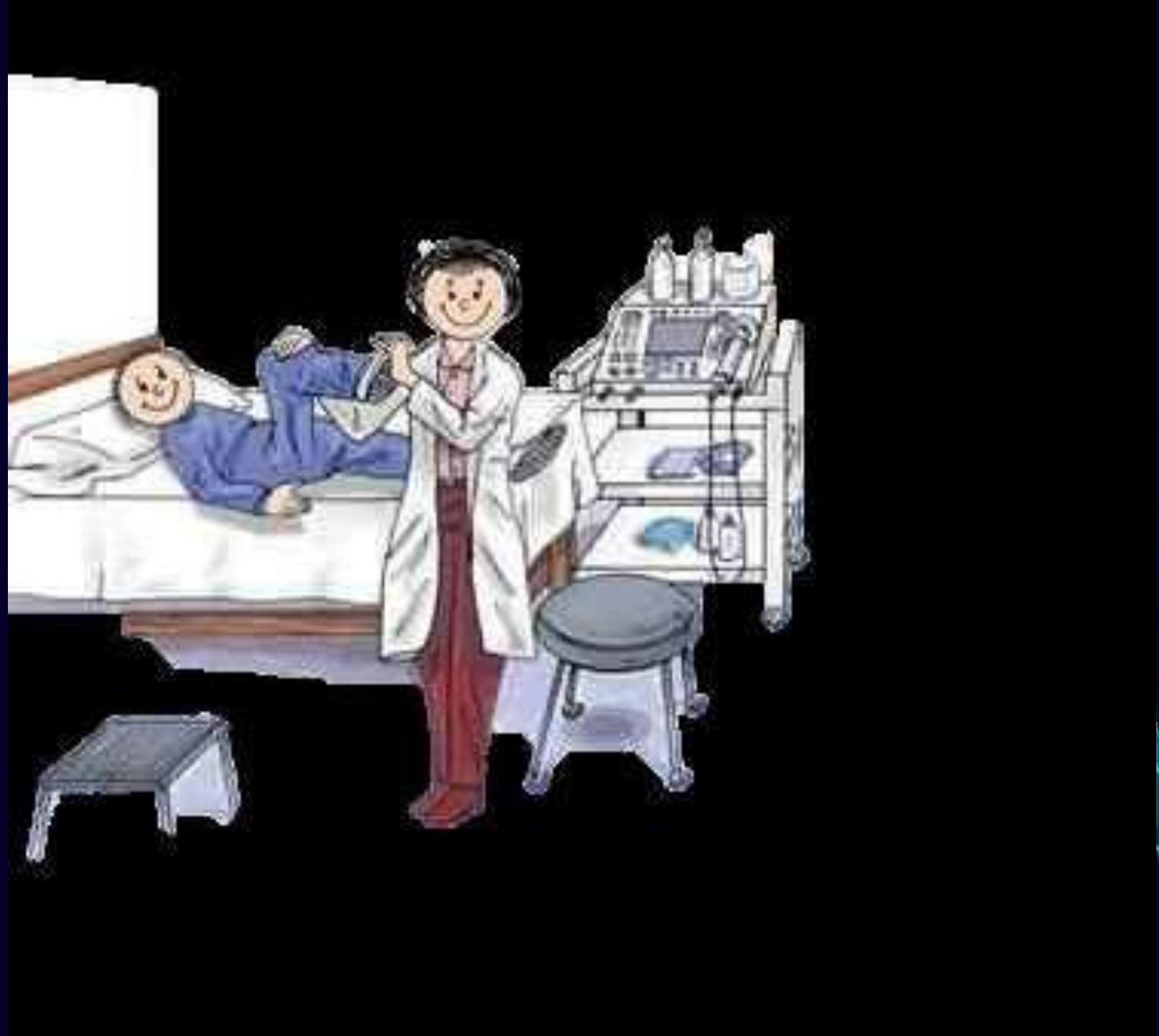
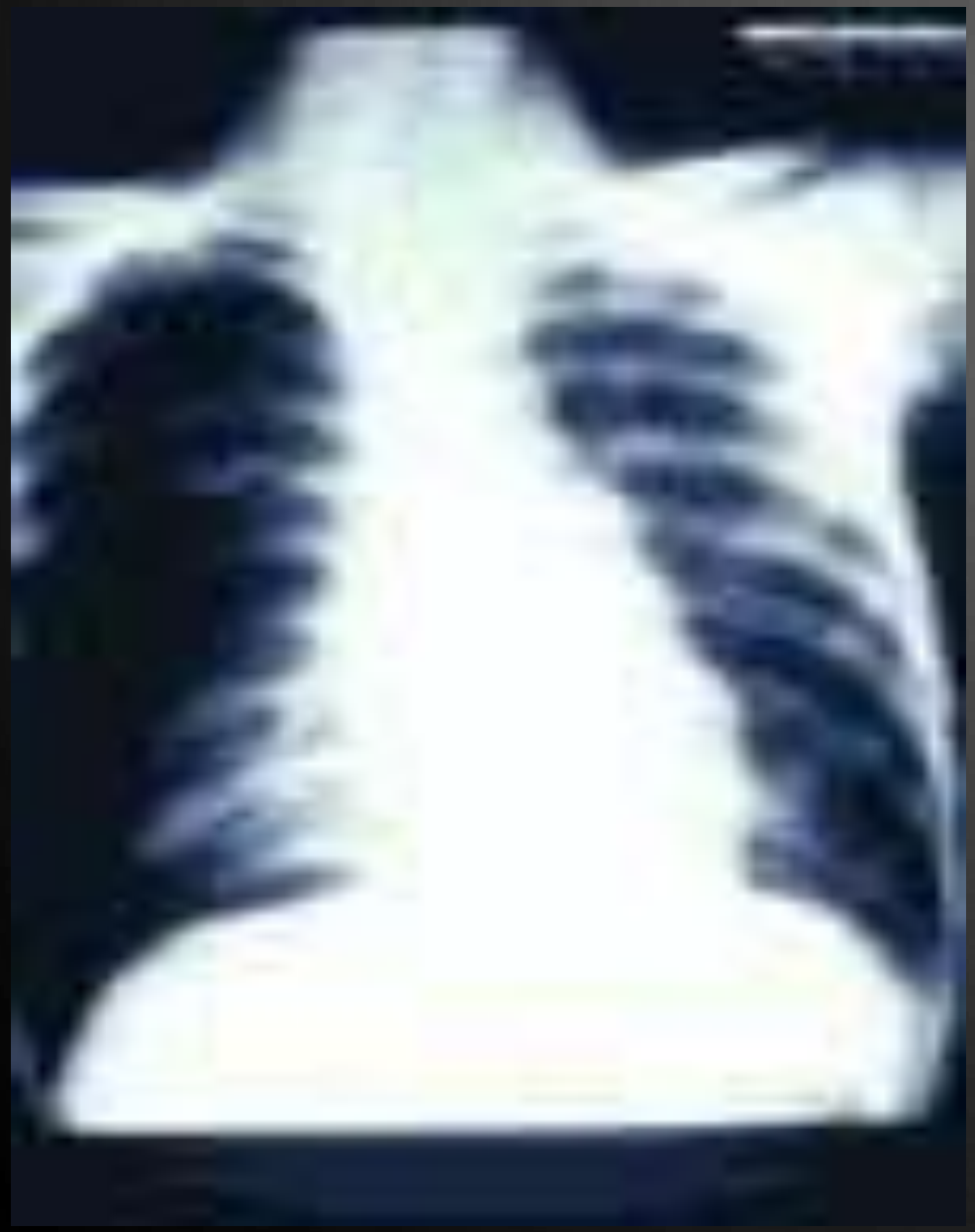




PNEUMOTHORAX IN ICU

Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

Introduction

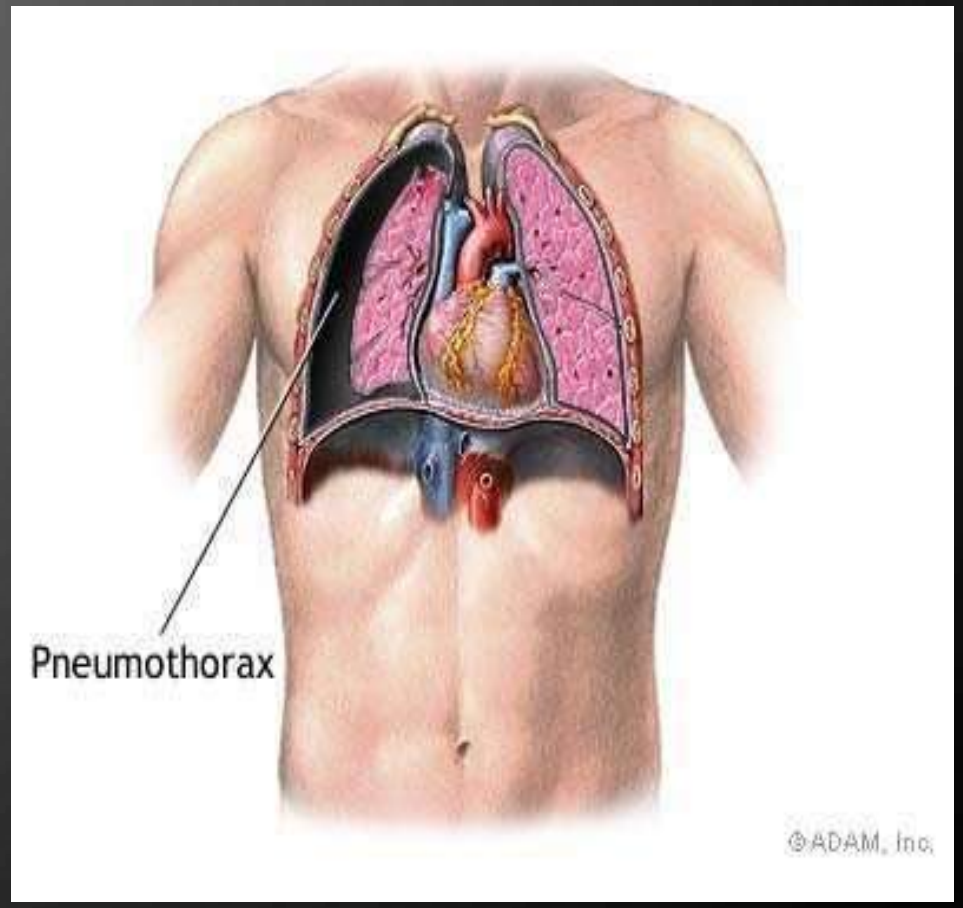
- Pneumothorax is a condition in which air is noted in the pleural space. It is classified as spontaneous (primary or secondary), traumatic or iatrogenic. Primary spontaneous pneumothorax occurs in individuals with no clinically apparent lung disease, whereas secondary spontaneous pneumothorax occurs as a complication of preexisting lung disease.

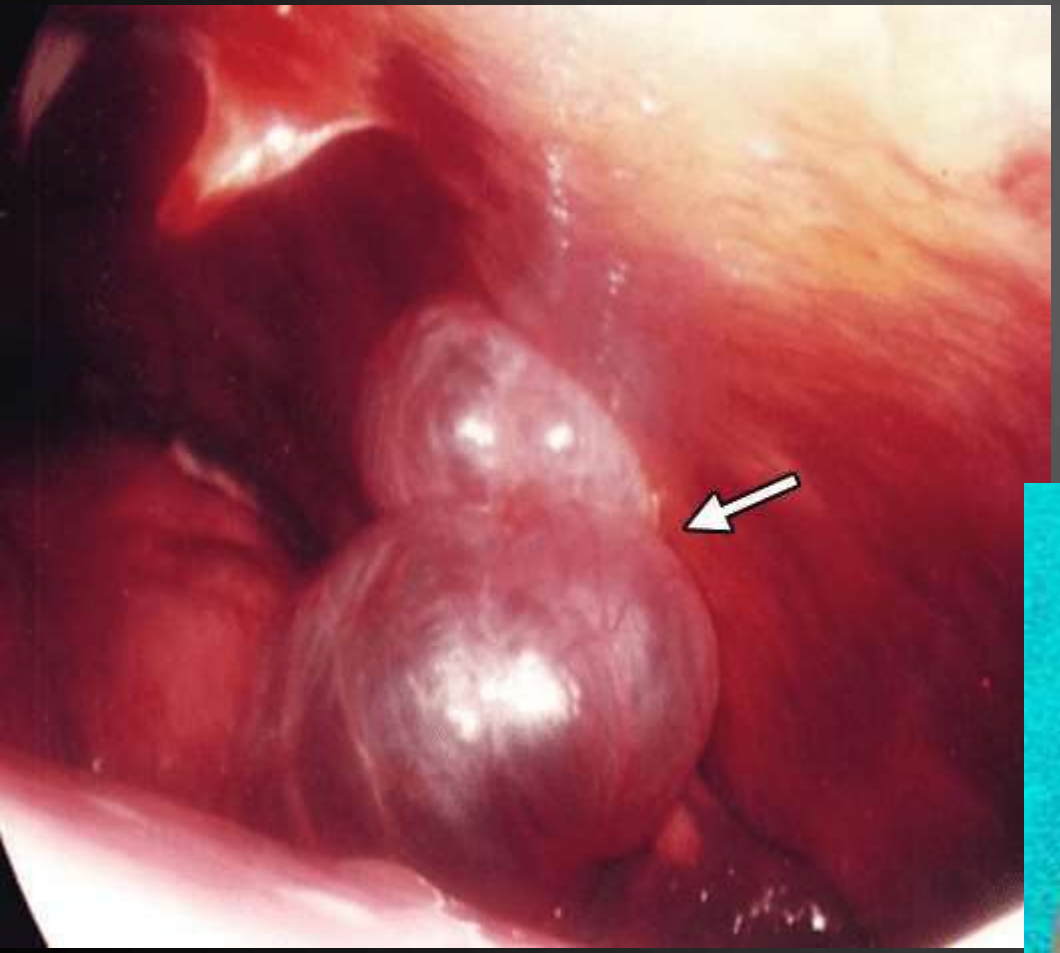
Introduction

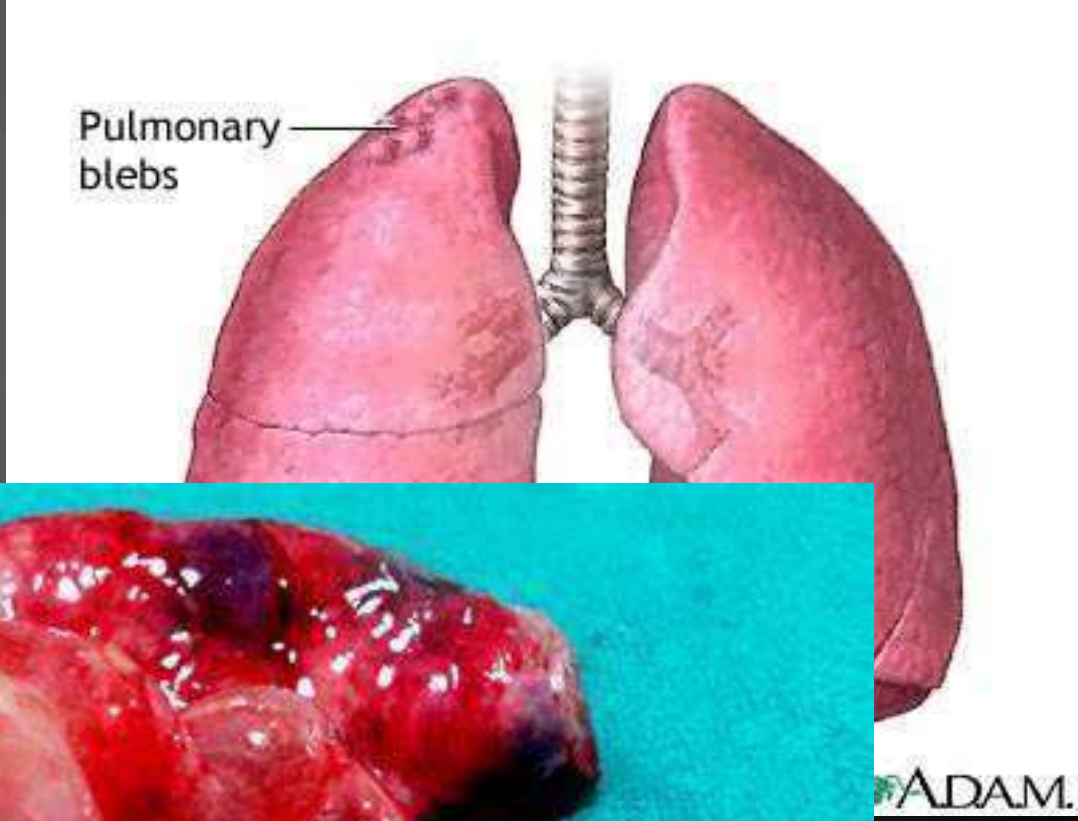
- Traumatic pneumothorax occurs as a result of penetrating or blunt trauma to the chest wall. Iatrogenic pneumothorax occurs as a result of diagnostic and therapeutic intervention and is the most common cause of pneumothorax in the ICU setting. Incidence in the 1990s was up to 24%, but more recent data show that the incidence has come down to about 3%. This is likely from a change in the medical management of ICU patients over time.

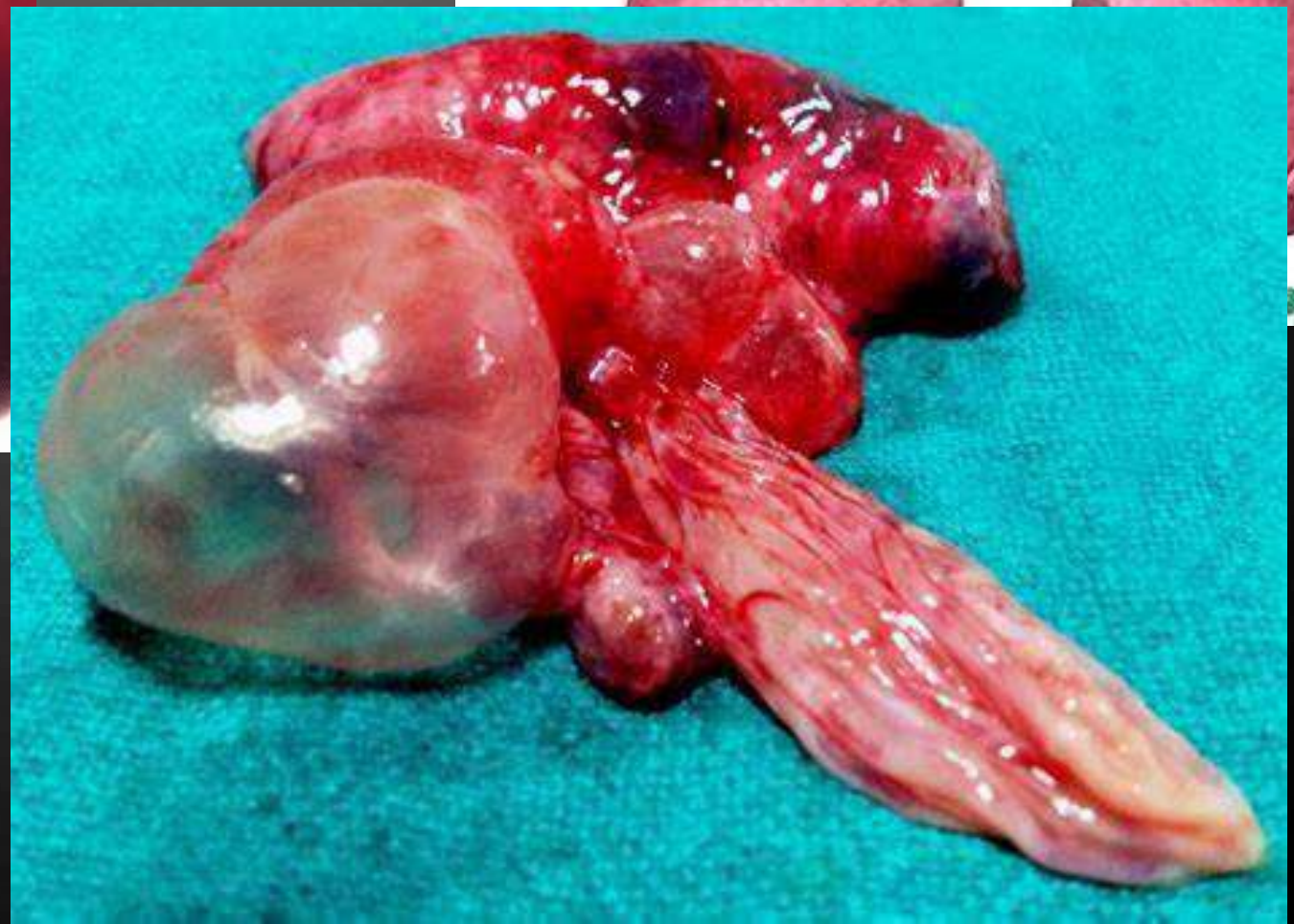
- Pneumothorax; air in pleural space

  - **Air can enter from the outside; injury penetrated the chest wall**
  - **Air can enter from inside, if the lung is torn or ruptured e.g. (pulmonary bleb).**






Pulmonary
blebs
A.D.A.M.

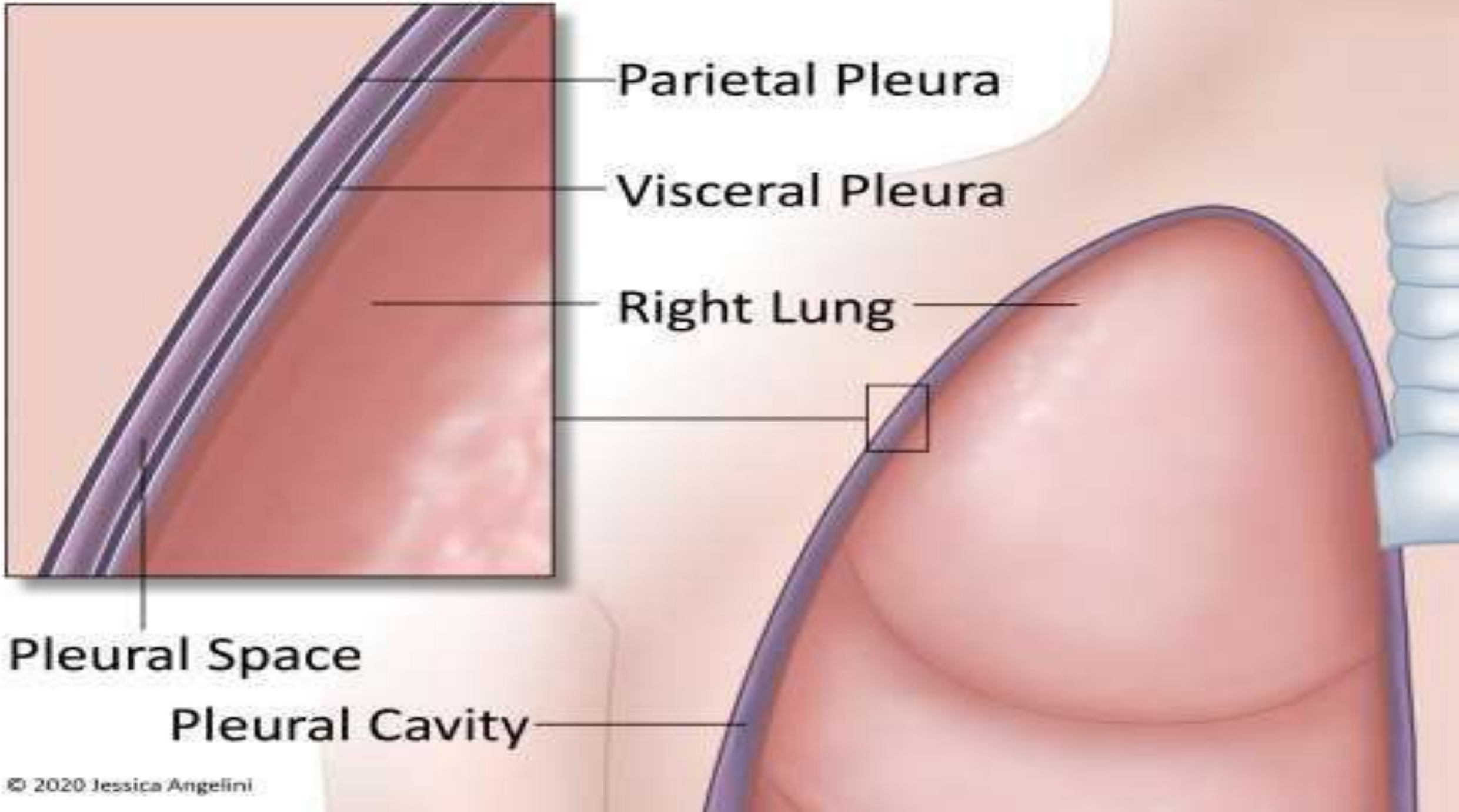


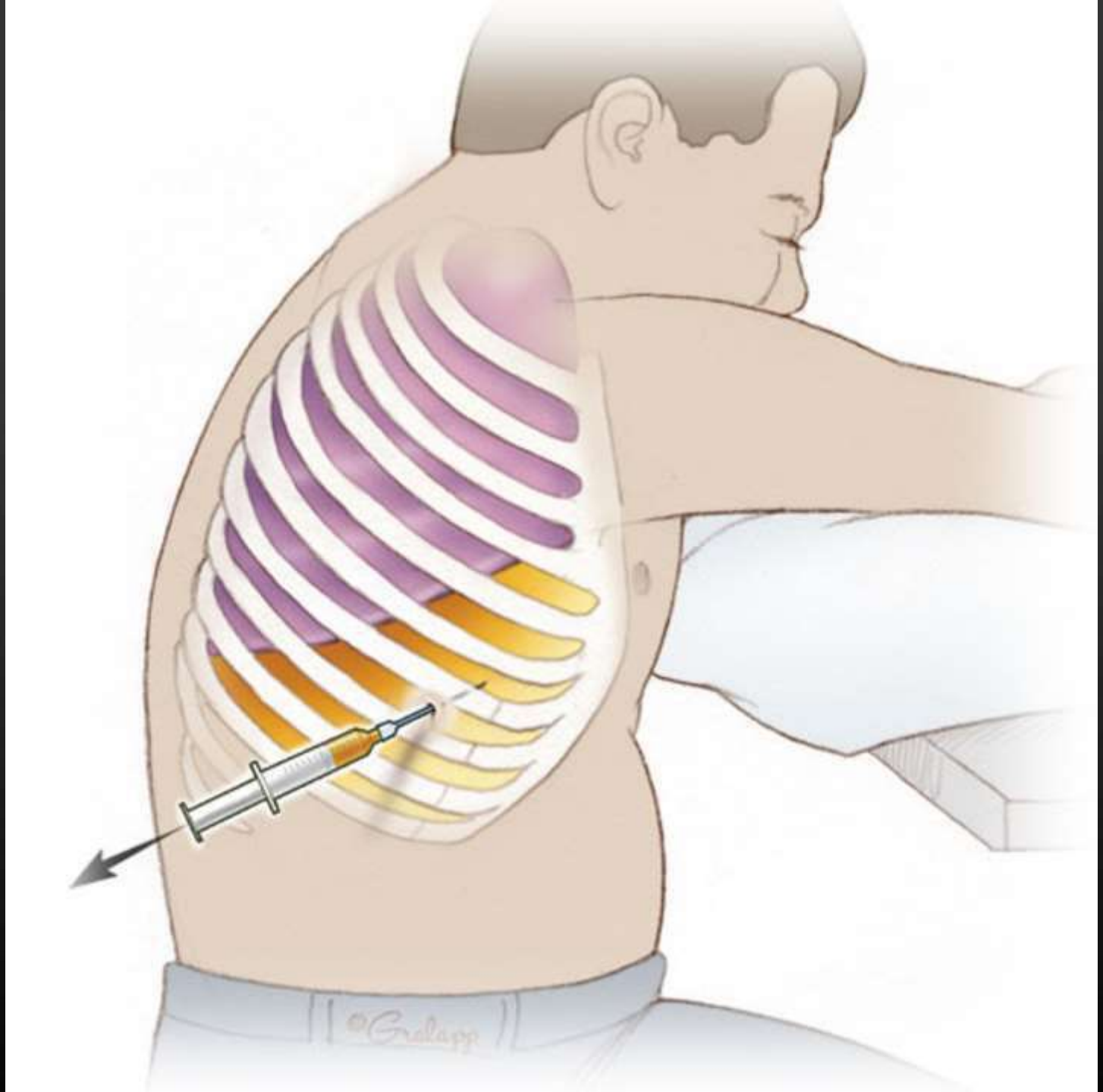
PATHOPHYSIOLOGY OF PNEUMOTHORAX

- The pressure gradient inside the thorax changes with a pneumothorax. Normally the pressure of the pleural space is negative when compared to atmospheric pressure. When the chest wall expands outwards, the lung also expands outwards due to surface tension between parietal and visceral pleurae. Lungs have a tendency to collapse due to elastic recoil. When there is communication between the alveoli and the pleural space, air fills this space changing the gradient, lung collapse unit equilibrium is achieved, or the rupture is sealed.

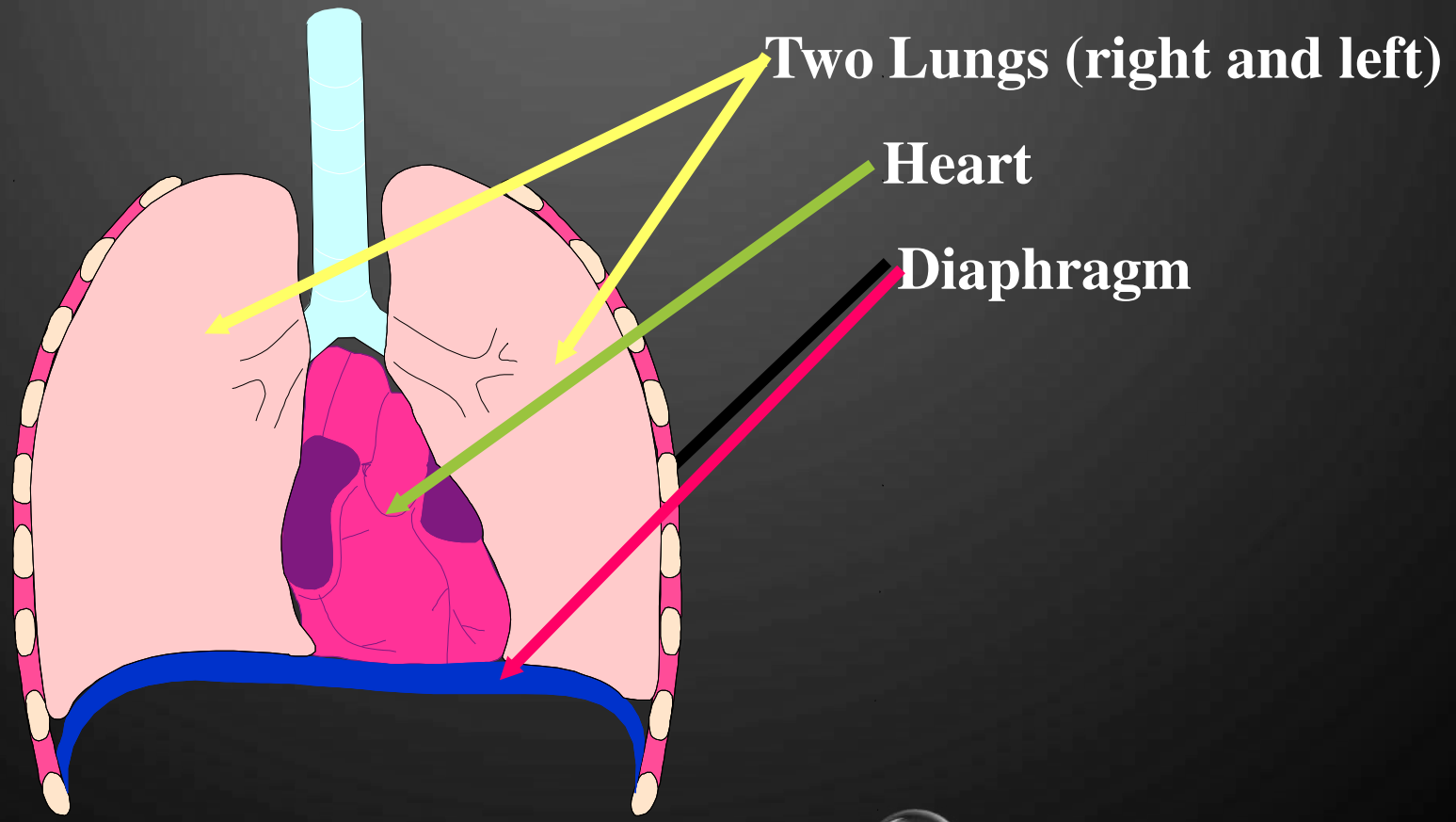
PATHOPHYSIOLOGY OF PNEUMOTHORAX

- Pneumothorax enlarges, and the lung gets smaller due to this vital capacity, and oxygen partial pressure decreases. Clinical presentation of a pneumothorax can range anywhere from asymptomatic to chest pain and shortness of breath.
- Iatrogenic pneumothorax occurs due to a complication of a medical or surgical procedure. Thoracentesis is the most common cause.

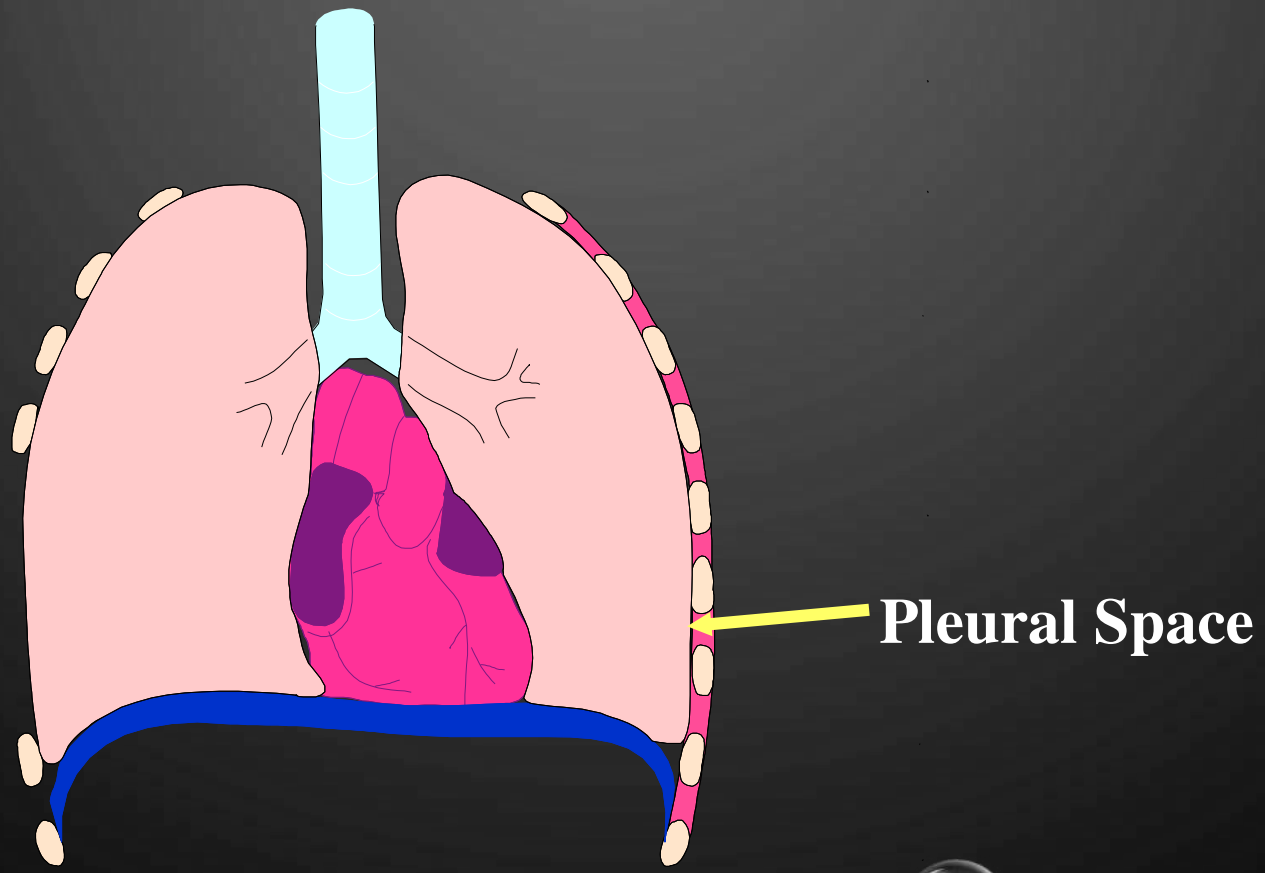


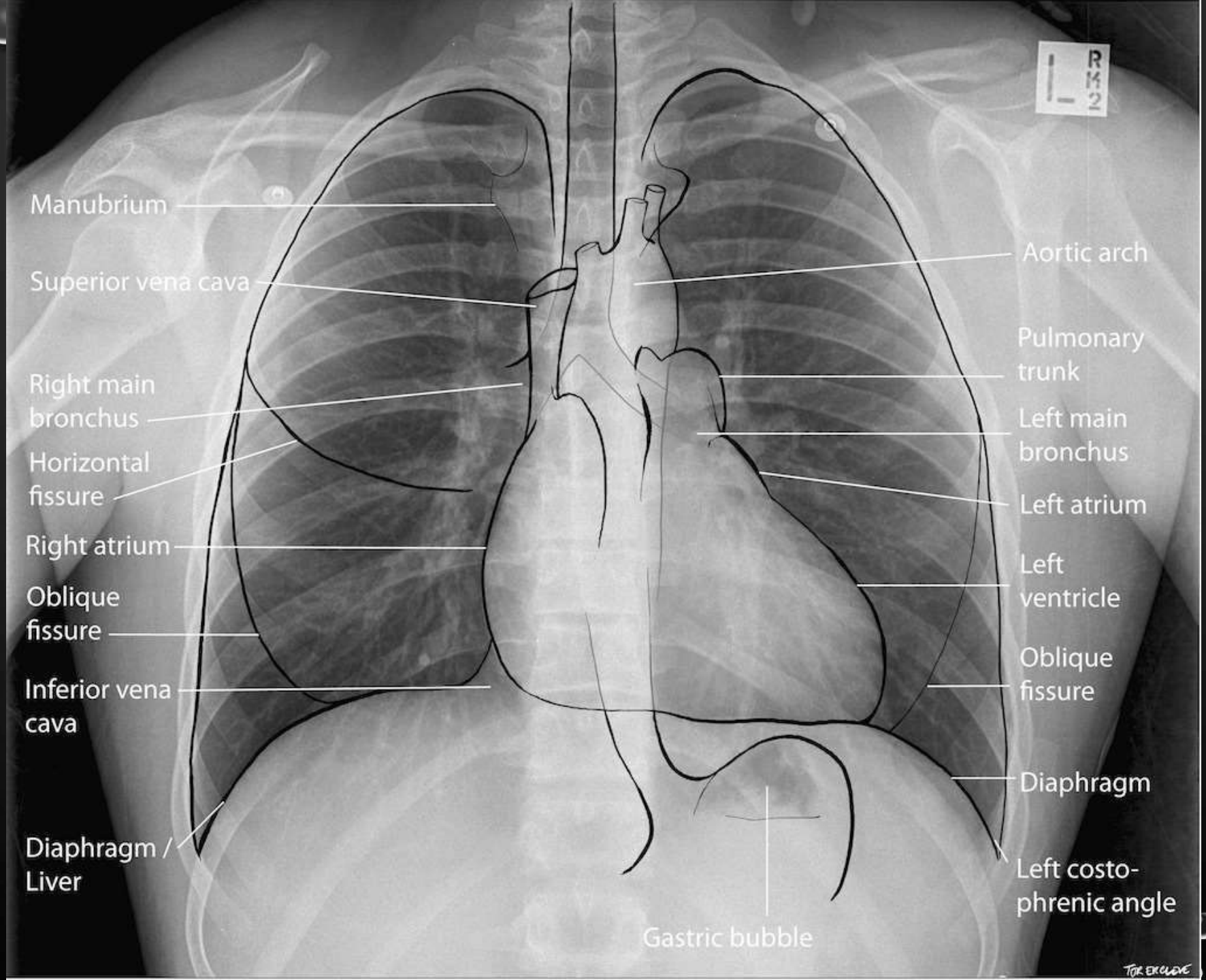


ANATOMY OF THE CHEST

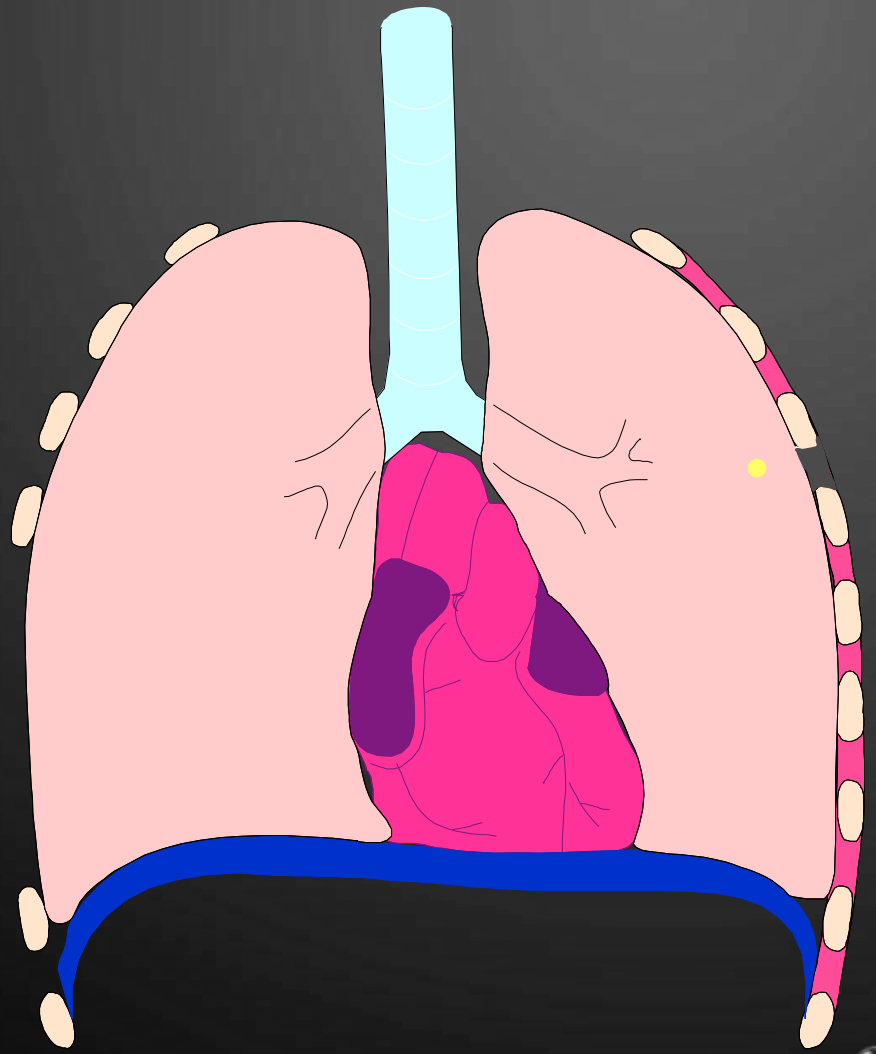


ANATOMY OF THE CHEST

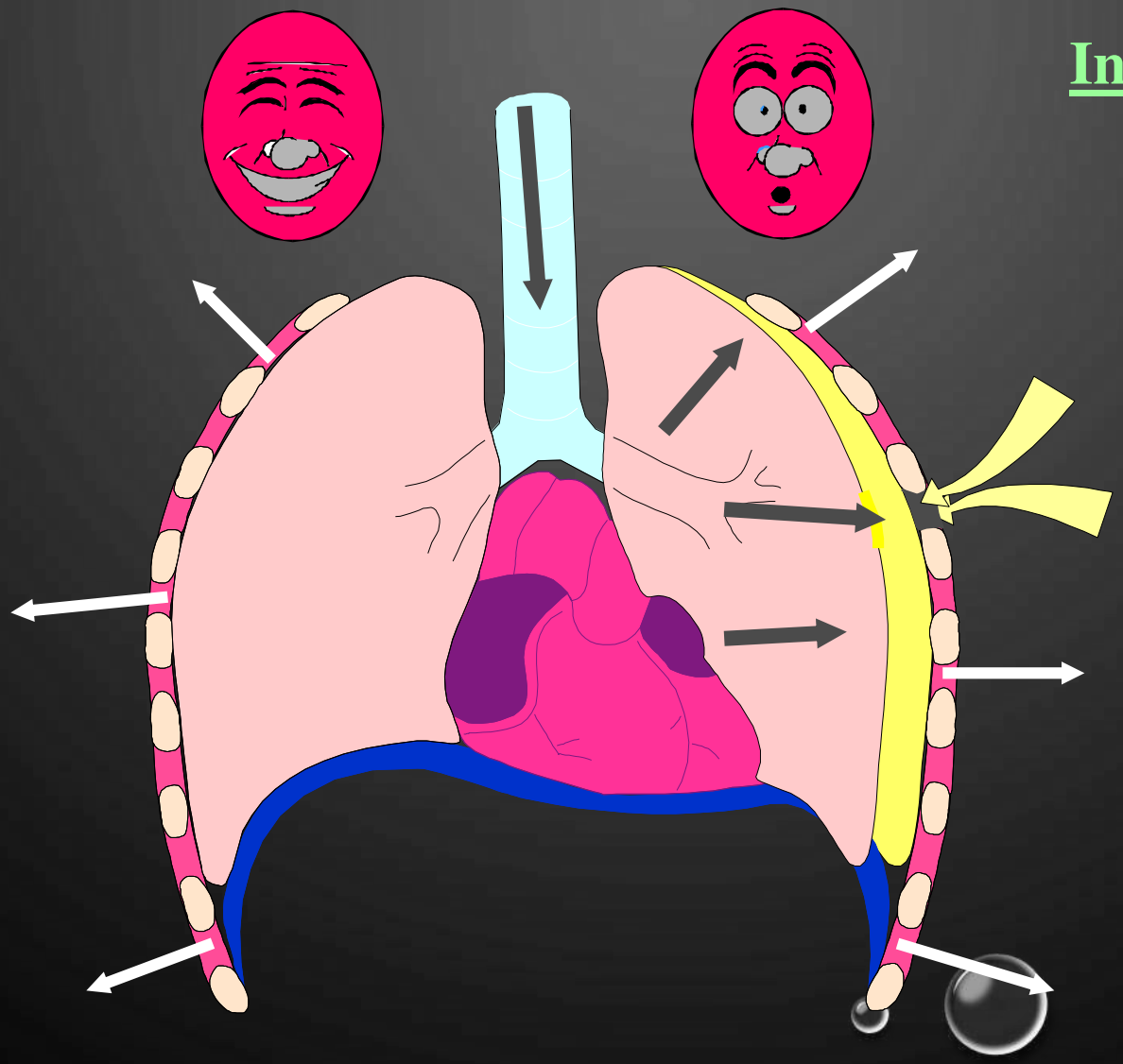



Manubrium
Superior vena cava
Right main bronchus
Horizontal fissure
Right atrium
Oblique fissure
Inferior vena cava
Diaphragm / Liver
Aortic arch
Pulmonary trunk
Left main bronchus
Left atrium
Left ventricle
Oblique fissure
Diaphragm
Left costo-phrenic angle
Gastric bubble

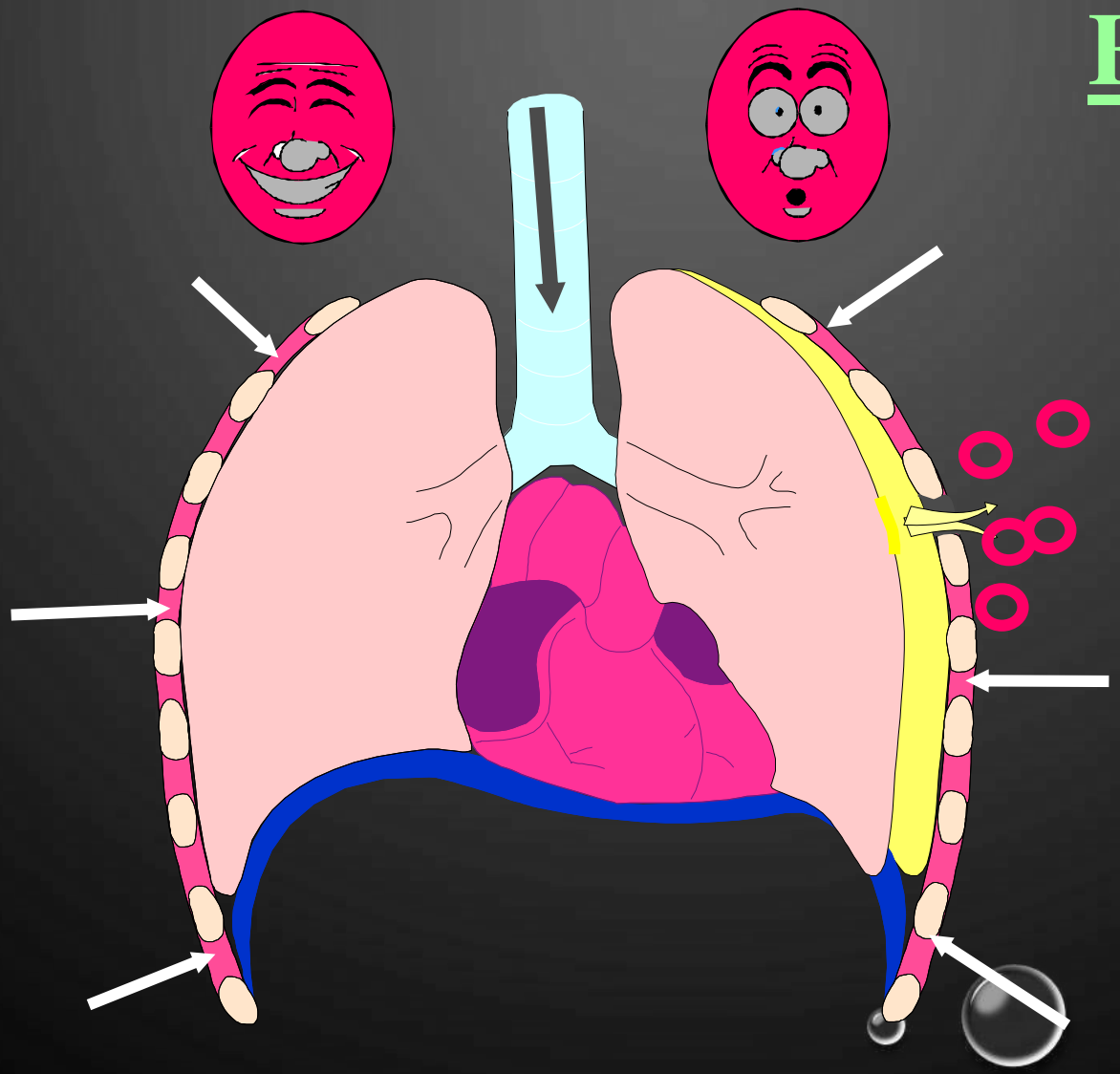
OPEN PNEUMOTHORAX



OPEN PNEUMOTHORAX

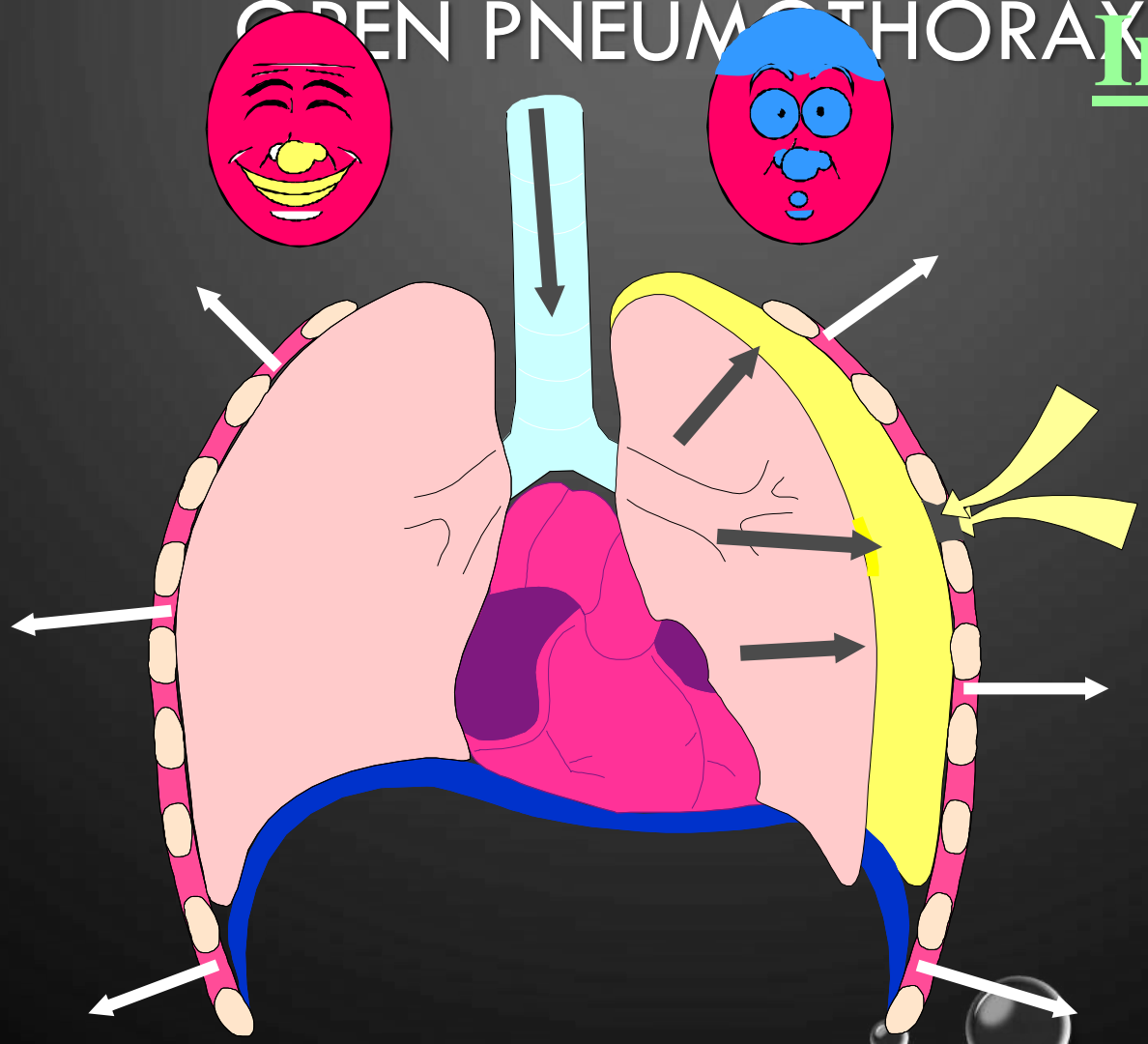


OPEN PNEUMOTHORAX

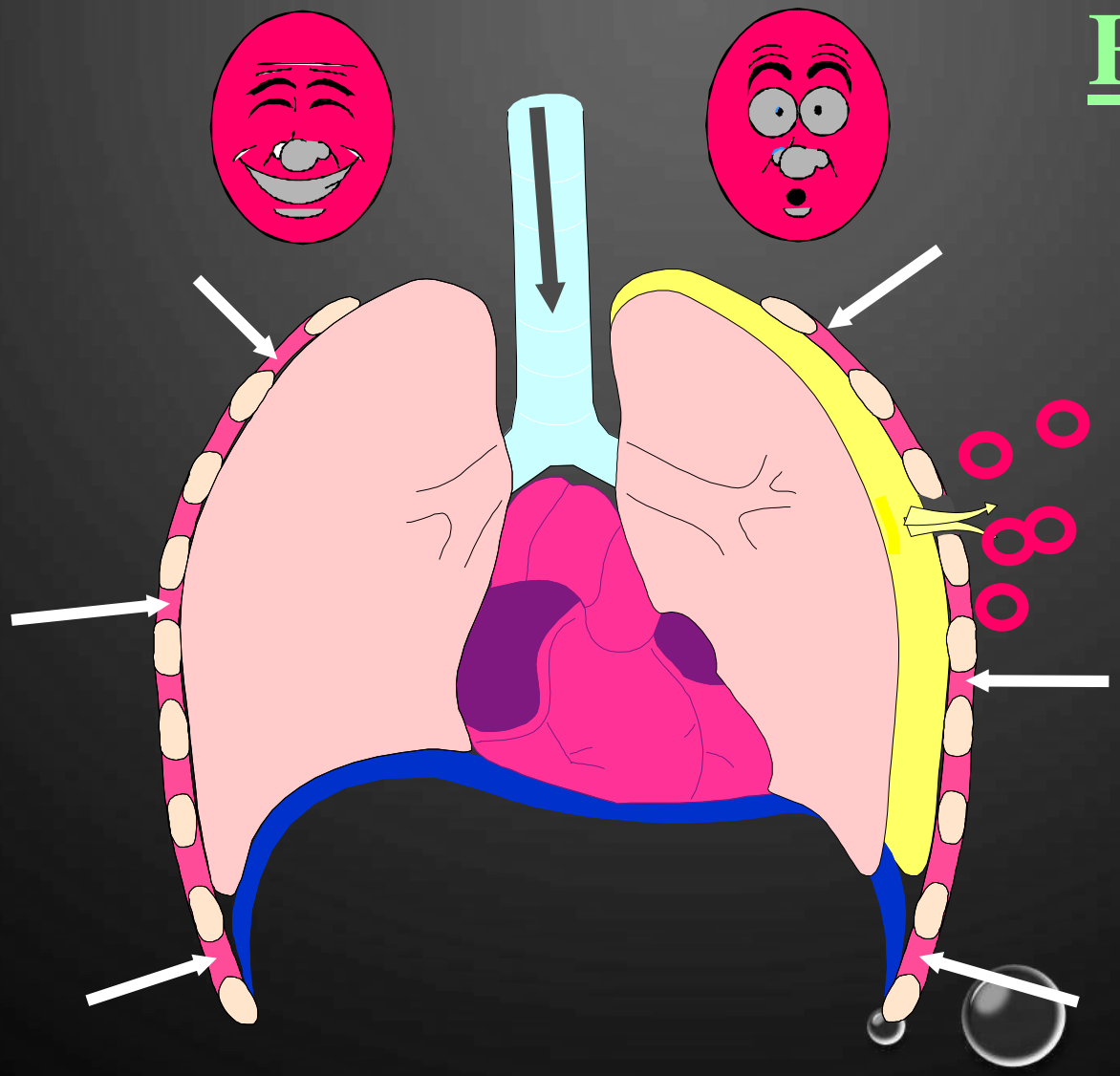


Exhale

OPEN PNEUMOTHORAX

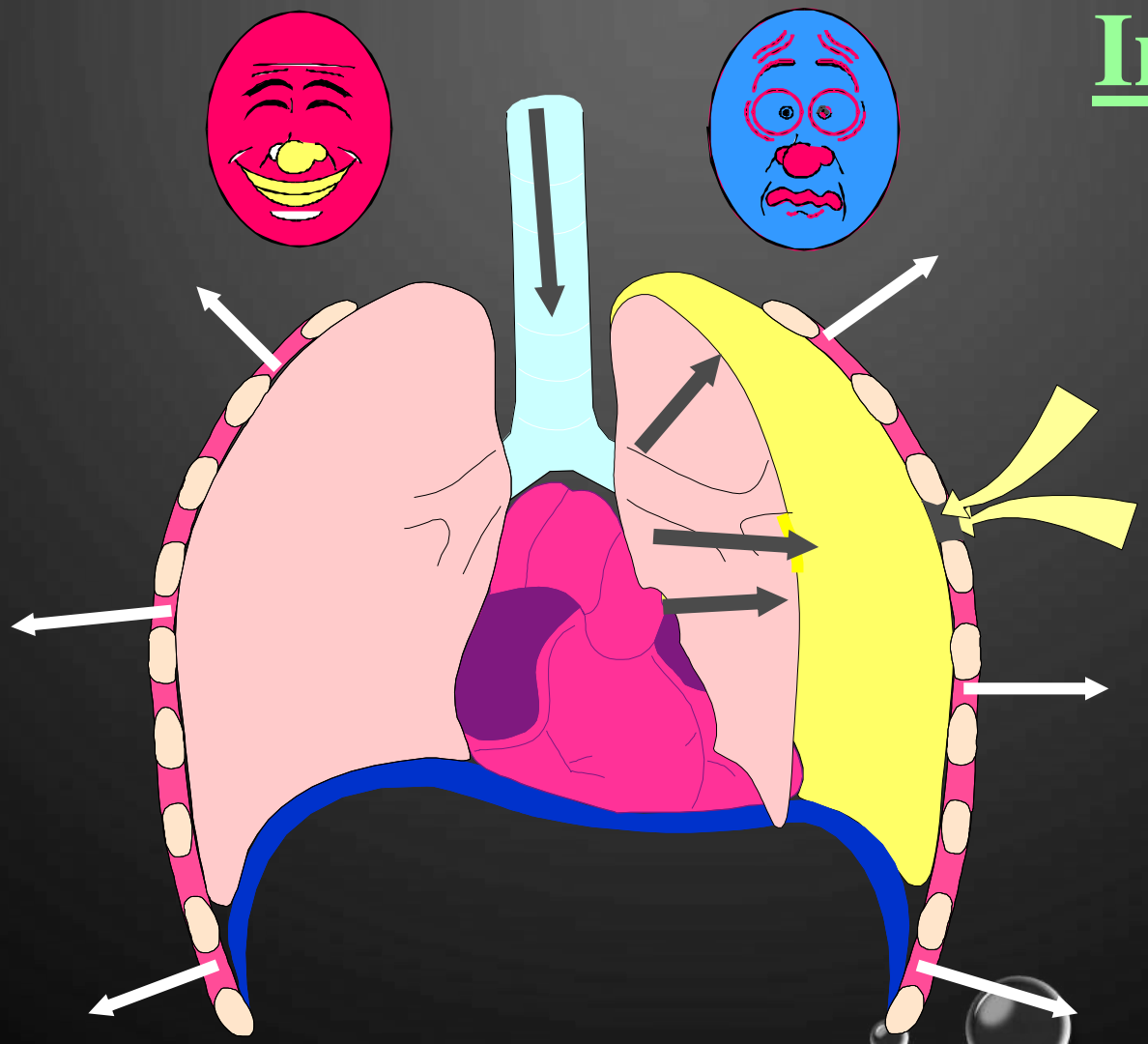


OPEN PNEUMOTHORAX



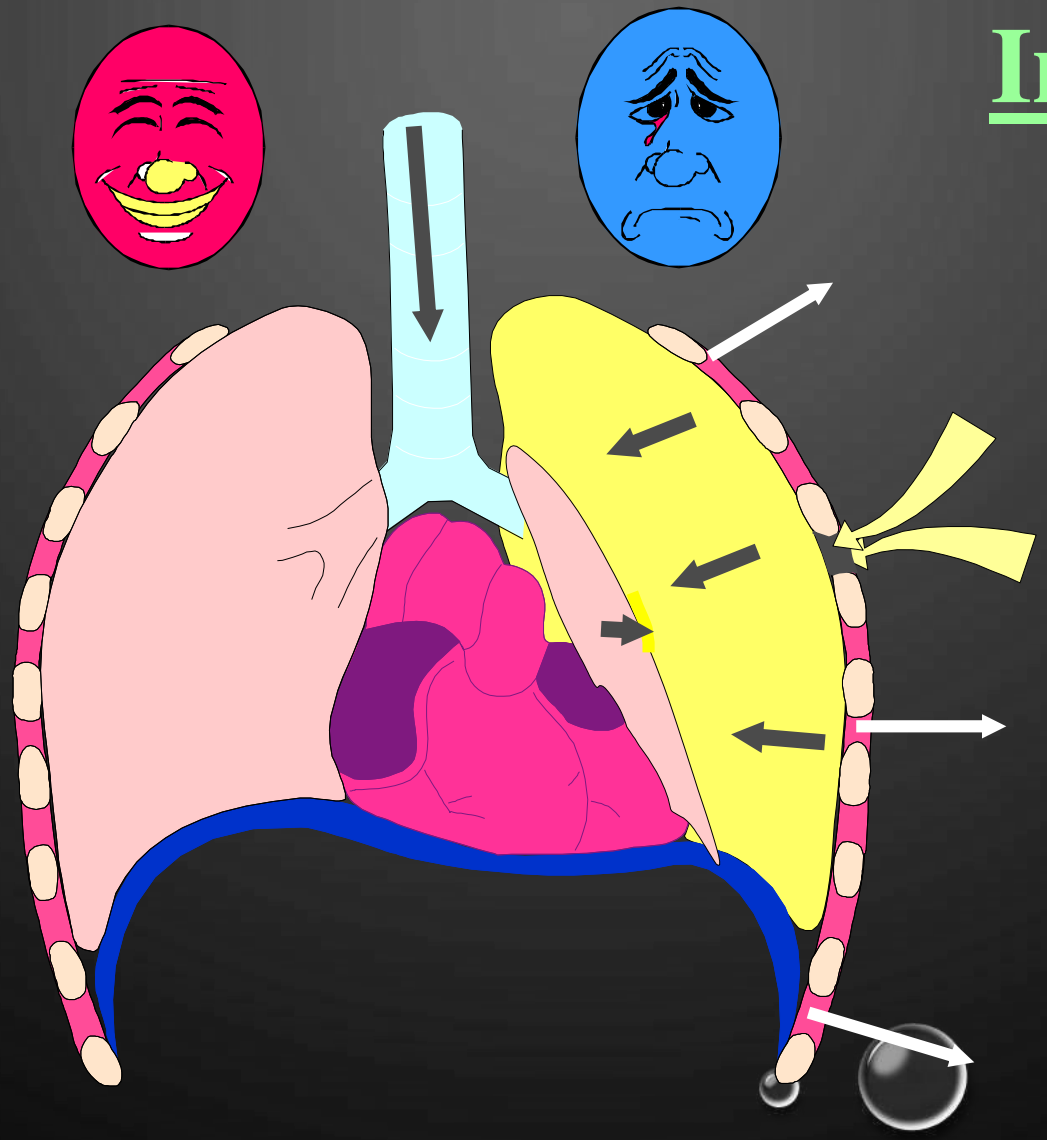
Exhale

OPEN PNEUMOTHOARX



Inhale

OPEN PNUEMOTHORAX



Inhale

Pneumothorax is the most common serious pleural complication in the ICU

ICU PTS : HIGH RISK GROUP

- Serious systemic disease
- Hemodynamically unstable; Invasive Procedures
- Ventilator/ Resuscitations
- Postoperative Patients; shifted from another invasive environment
- Trauma; admitted to ICU
  - Penetrating Injury of Chest/ Abdomen
  - # Rib
  - Central Line

PNEUMOTHORAX IN THE INTENSIVE CARE UNIT: INCIDENCE & RISK FACTORS,

- Incidence 1.4% on day 5 and 3.0% on day 30.
- Risk factors
  - History of adult immunodeficiency syndrome
  - Cardiogenic pulmonary edema at admission
  - Use of inotropic agents during the first 24 h

- Disease; ARDS
- Ventilation; Incidence (4 to 15%).
- Procedures
  - Thoracentesis,
  - Bronchoscopy
  - Pericardiocentesis
  - Tracheostomy

TYPES/ ETIOLOGY

- Spontaneous
  - Primary
  - Secondary
- Iatrogenic / Traumatic
- Open/ Close
- **Tension Pneumothorax**

SPONTANEOUS PNEUMOTHORAX

- This refers to a condition in which the lung collapses with no apparent injury or trauma
  - COPD
  - AIDS/ Lung Tumor
  - Infective or Infiltrative Lung Disease
- Cigarette smokers & drug users are at greater risk for spontaneous pneumothorax.

HOW MECHANICAL VENTILATION RESPONSIBLE FOR

Pneumothorax ?

BIOTRAUMA

Barotrauma and Volutrauma

Atelectrauma

BAROTRAUMA AND VOLUTRAUMA

- Ventilator-induced lung injury by high levels of mechanical stress that occur when **high airway pressures**(Barotrauma) and **high volumes** (Volutrauma) are delivered .

- **Atelectrauma**
  - When parts of the lung collapse at the end of expiration, due to a combination of a diseased lung state and a low functional residual capacity, then reopen again on inspiration, this repeated collapsing and reopening causes shear stress which has a damaging effect on the alveolus

RELATIONSHIP BETWEEN VENTILATORY SETTINGS AND BAROTRAUMA IN THE ARDS

- incidence of barotrauma 0% to 49%,
- High incidence correlated strongly
  - P(plat), above 35 cm H2O,
  - Compliance below 30 ml/cm H2O
- **Aspiration Pneumonia**
  - In a study 38 percent of patients developed pneumothorax

PNEUMOTHORAX TRAUMATIC/ PROCEDURE RELATED

- Direct trauma to the chest wall from either blunt or penetrating trauma causes this condition
- **Thoracentesis (54%)**
- **Central vein/pulmonary artery catheterization (40%)**
- **Bronchoscopy /transbronchial lung biopsy (23%)**

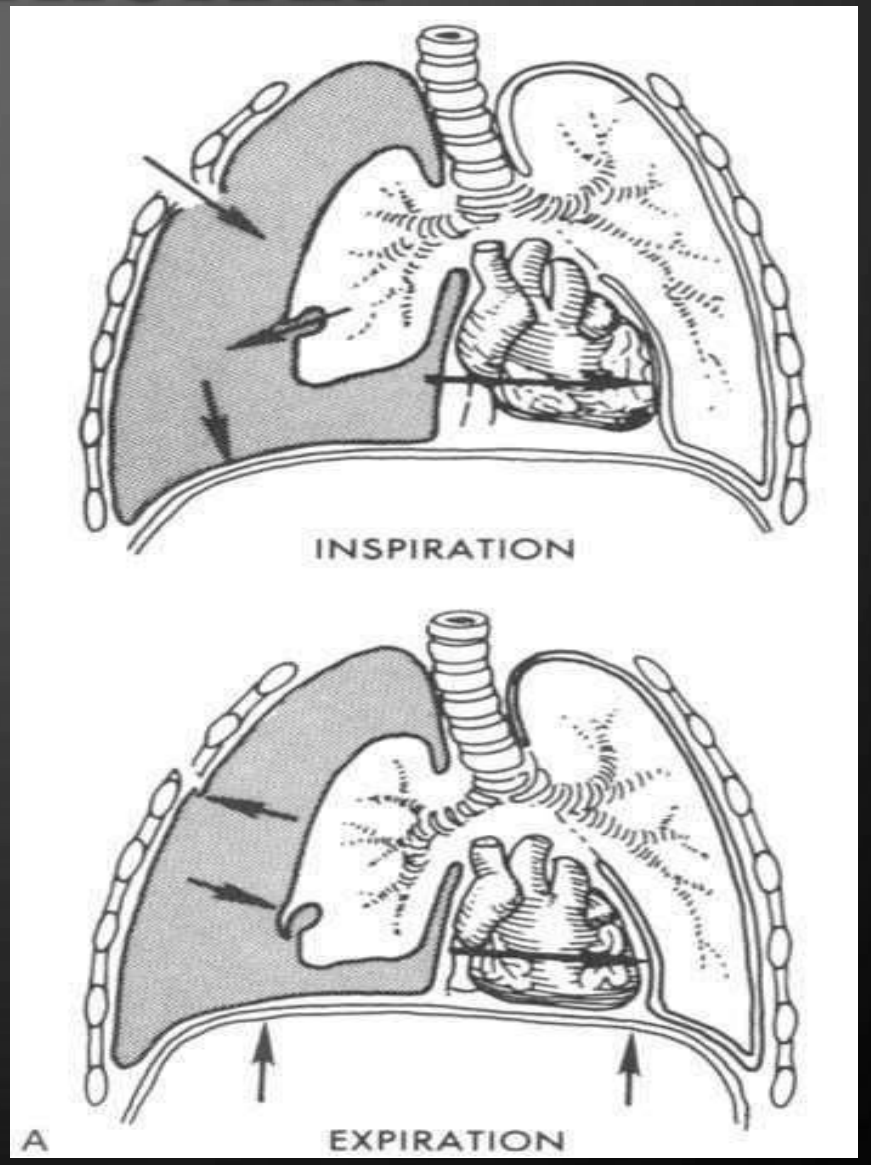
❖ **There is no difference in the rates of pneumothorax for internal jugular versus subclavian vein placement**

SYMPTOMS AFTER **CENTRAL VEIN/PULMONARY ARTERY CATHETERIZATION ASSOCIATED PNEUMOTHORAX**

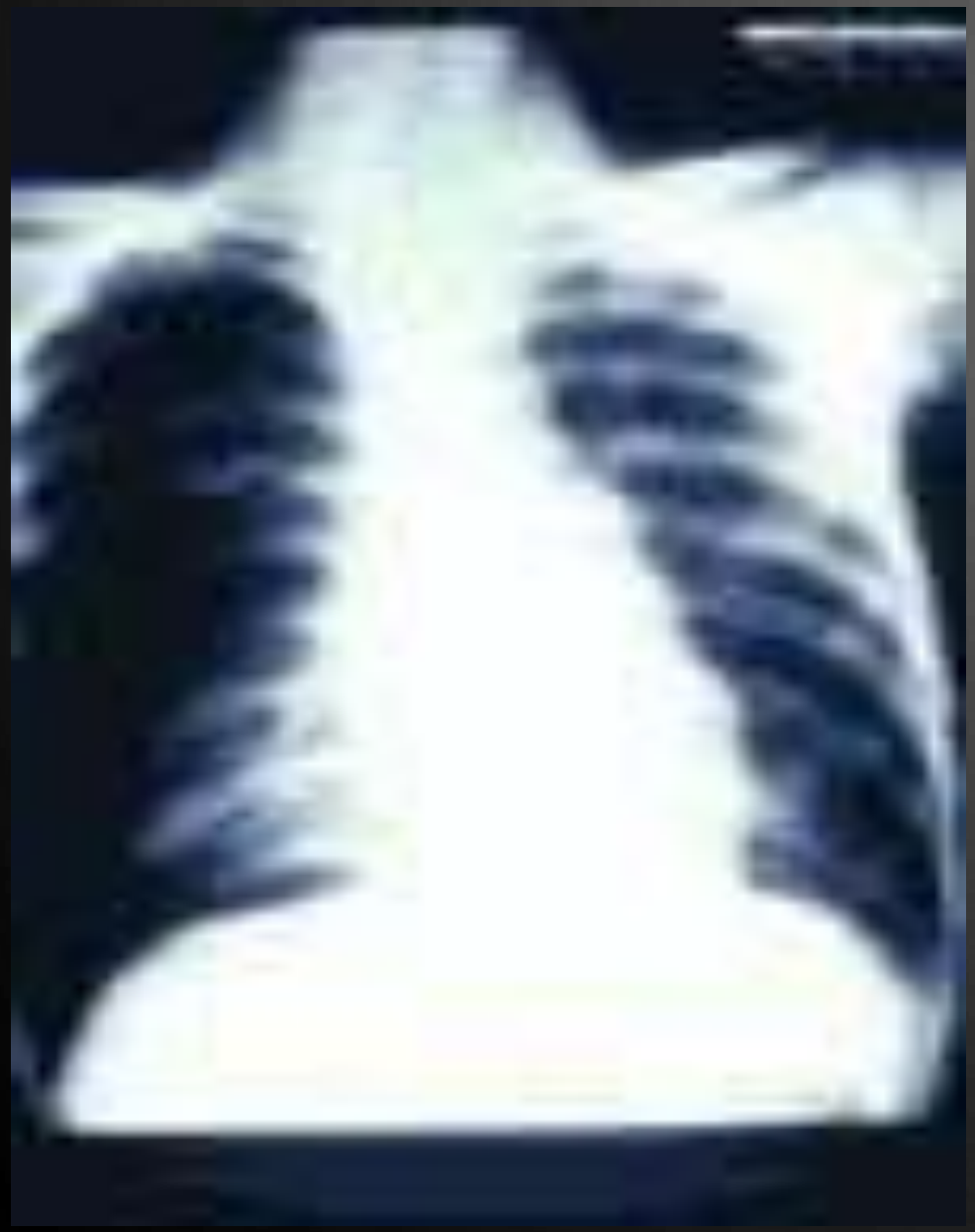
- Sharp, stabbing chest pain that worsens on breathing or with deep inspiration. Pain often radiates to the shoulder and or back
- A dry cough may occur because of irritation of the diaphragm.

TENSION PNEUMOTHORAX

- When the pleural pressure is positive throughout respiratory cycle
- "Ball-valve mechanism"
- Injury to pleura creates a **tissue flap** that opens on inspiration and closes on expiration



THANK YOU



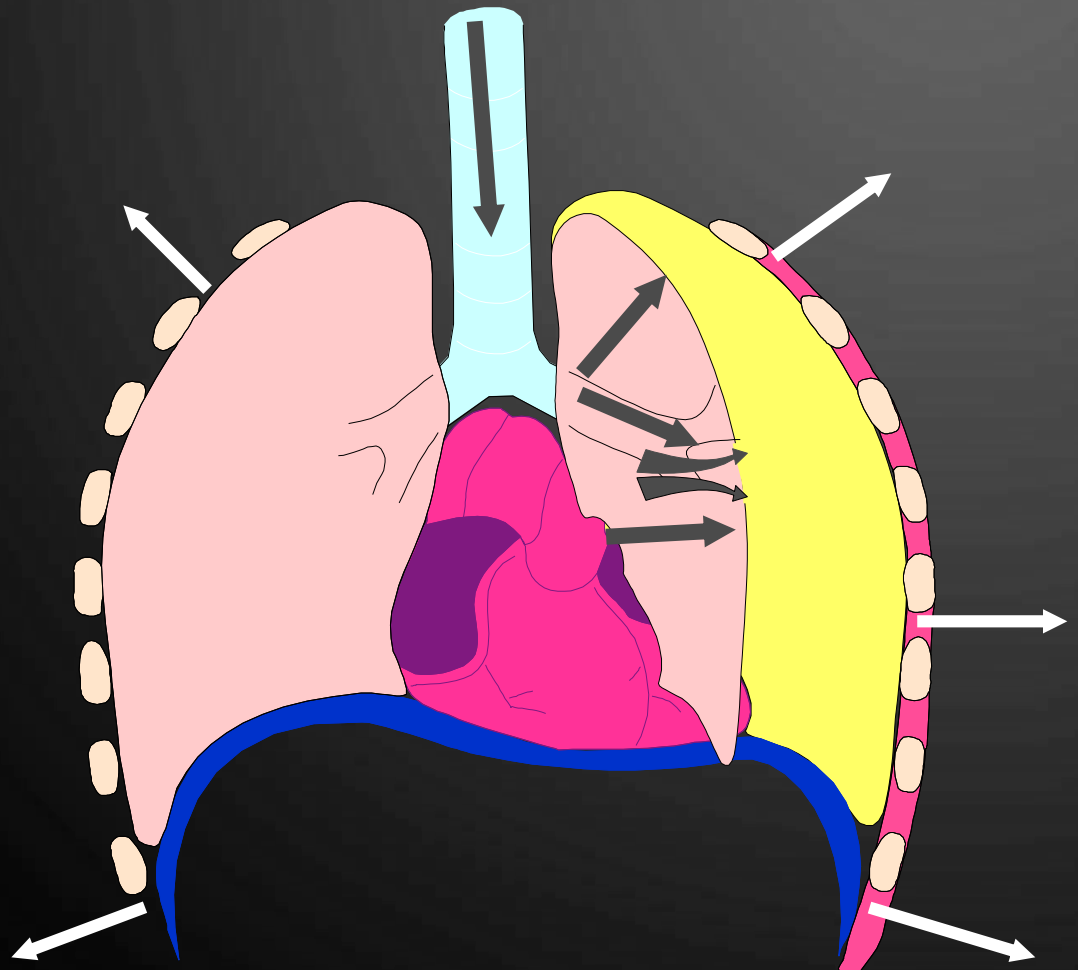
TENSION PNEUMOTHORAX
L-17

Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

Pathophysiology of Tension Pneumothorax

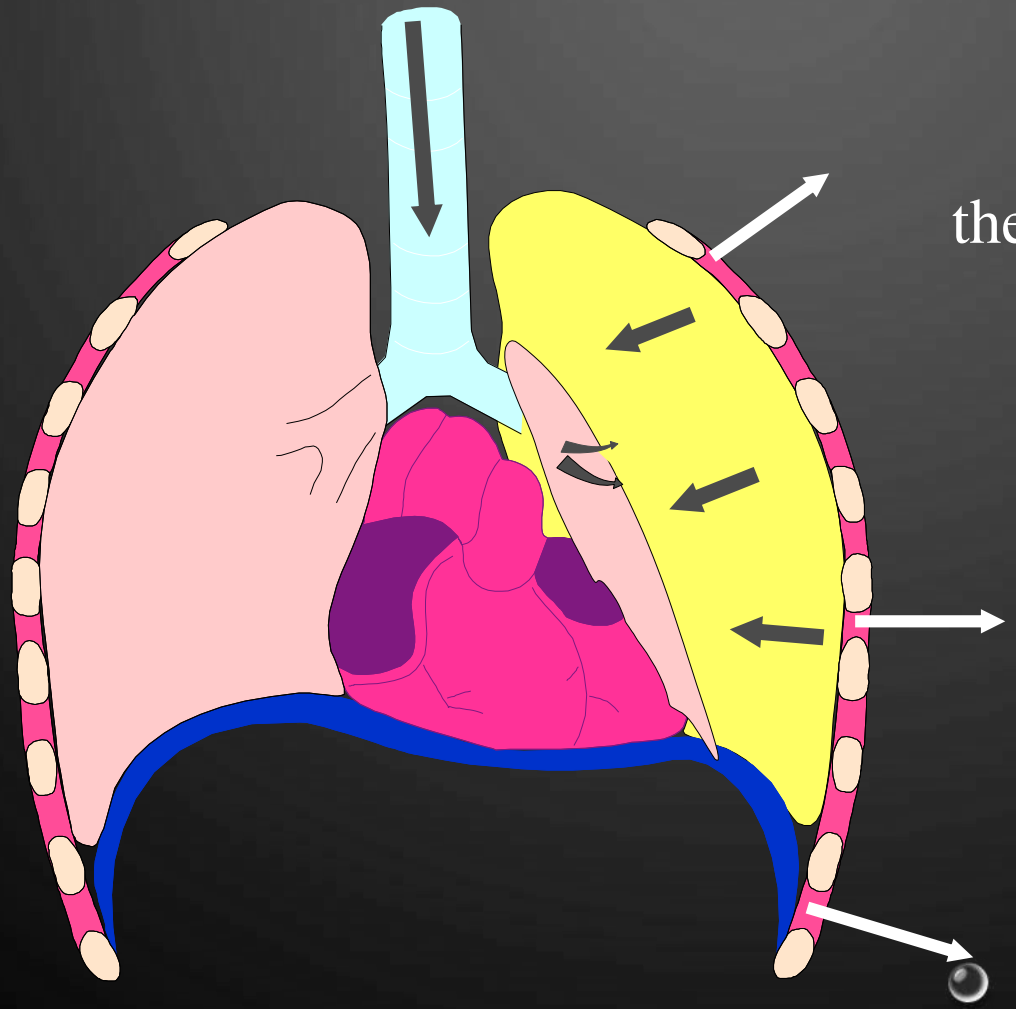
Tension pneumothorax is a pneumothorax causing a progressive rise in intrapleural pressure to levels that become positive throughout the respiratory cycle and collapses the lung, shifts the mediastinum, and impairs venous return to the heart. Air continues to get into the pleural space but cannot exit. Without appropriate treatment, the impaired venous return can cause systemic hypotension and respiratory and cardiac arrest (pulseless electrical activity) within minutes. Tension pneumothorax most commonly occurs in patients receiving positive-pressure ventilation (with mechanical ventilation or particularly during resuscitation). Rarely, it is a complication of traumatic pneumothorax, when a chest wound acts as a one-way valve that traps increasing volumes of air in the pleural space during inspiration.

TENSION PNEUMOTHORAX



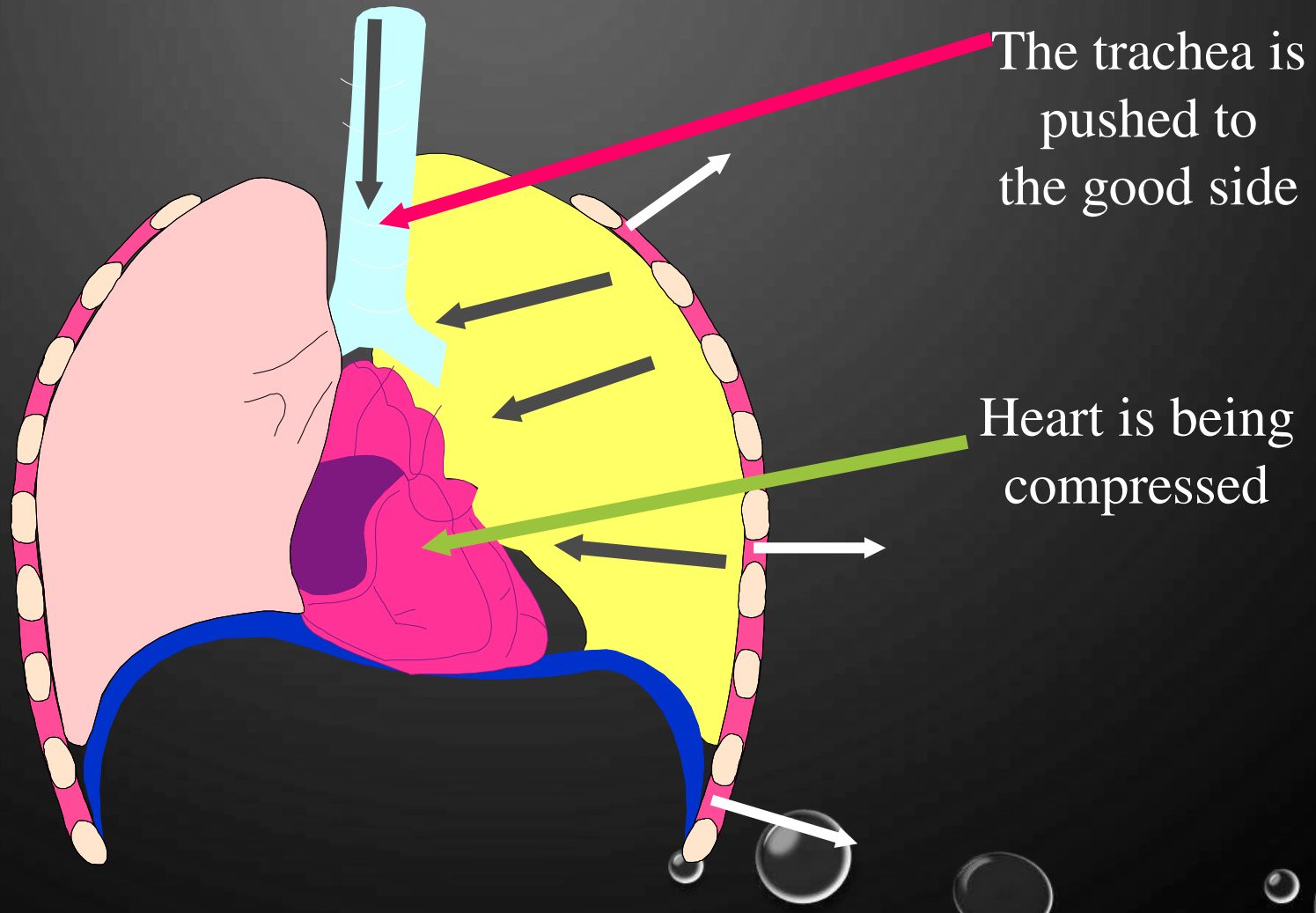
Each time we inhale, the lung collapses further. There is no place for the air to escape..

TENSION PNEUMOTHORAX

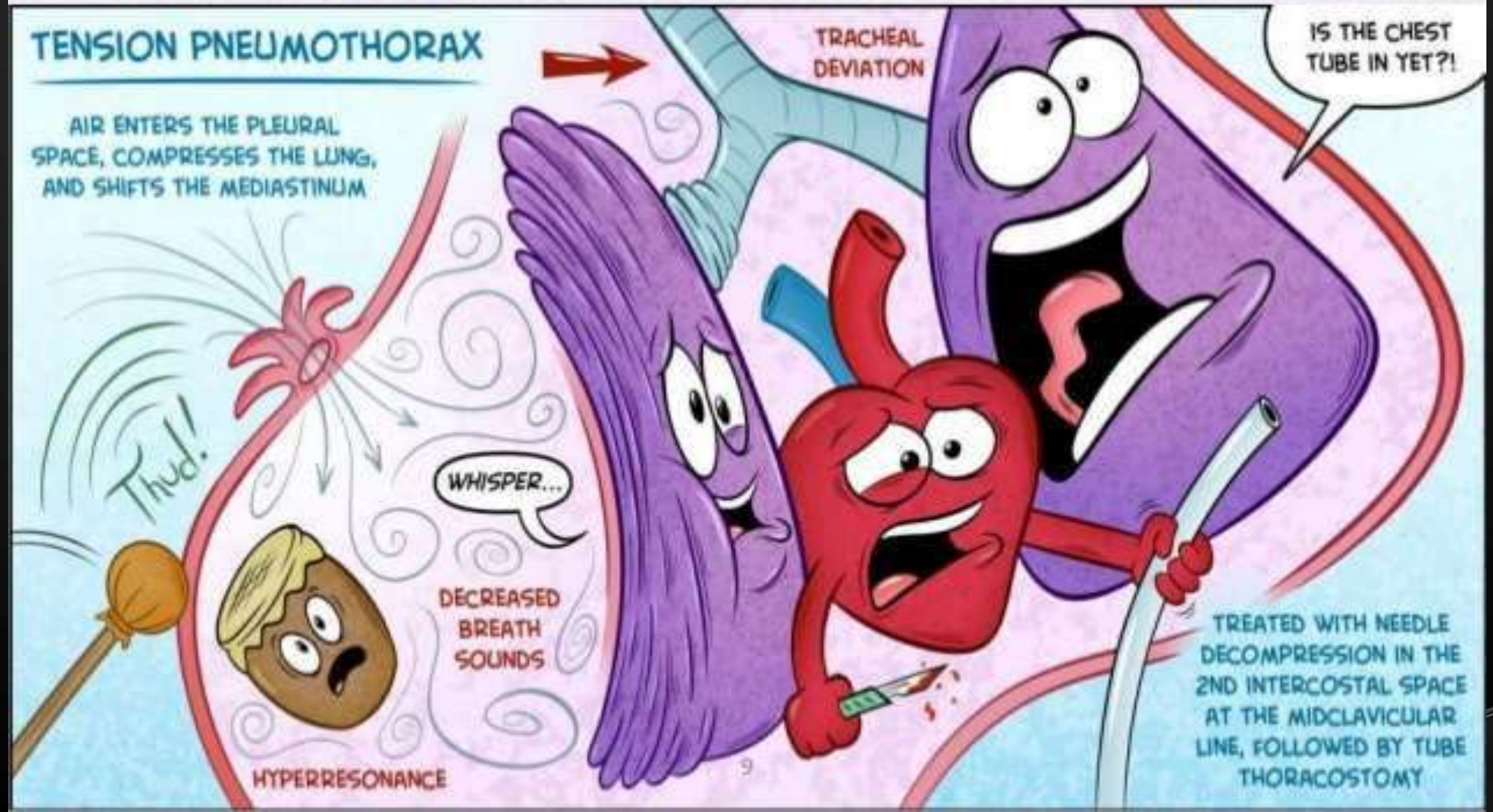


Each time we inhale, the lung collapses further. There is no place for the air to escape..

TENSION PNEUMOTHORAX



Tension Pneumothorax: Medical Emergency

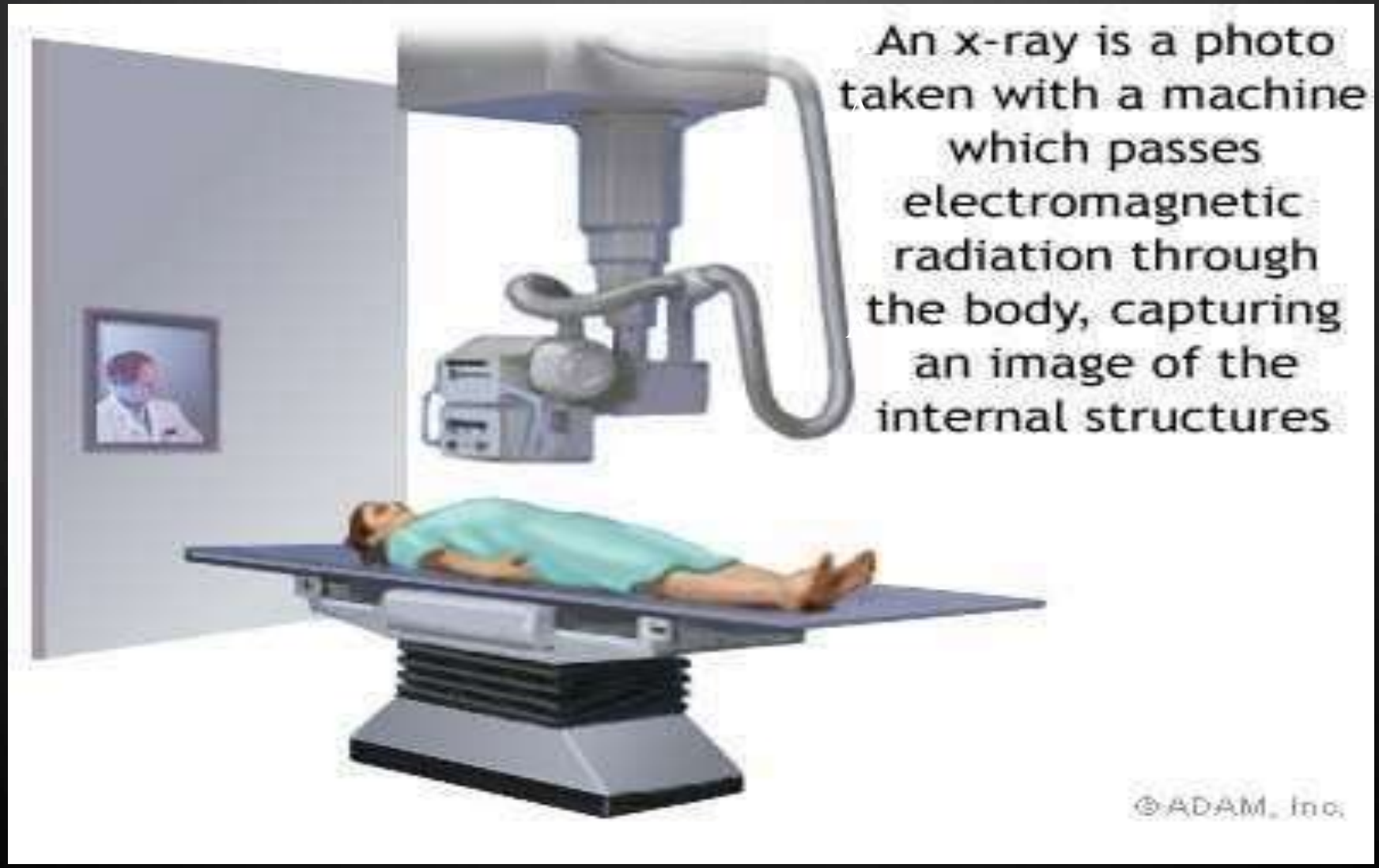


CLINICAL PICTURE

- Distressed
- Difficult breathing
- Cyanosis
- Marked tachycardia
- Hypotension
- Decreased breath sounds
- Hyper resonance on percussion

- Hypoxia
  - Decreased PaO2
  - Perfusion of atelectatic lung
- Decrease venous return
  - increase intrathoracic pressure
  - Decreased CO & SV

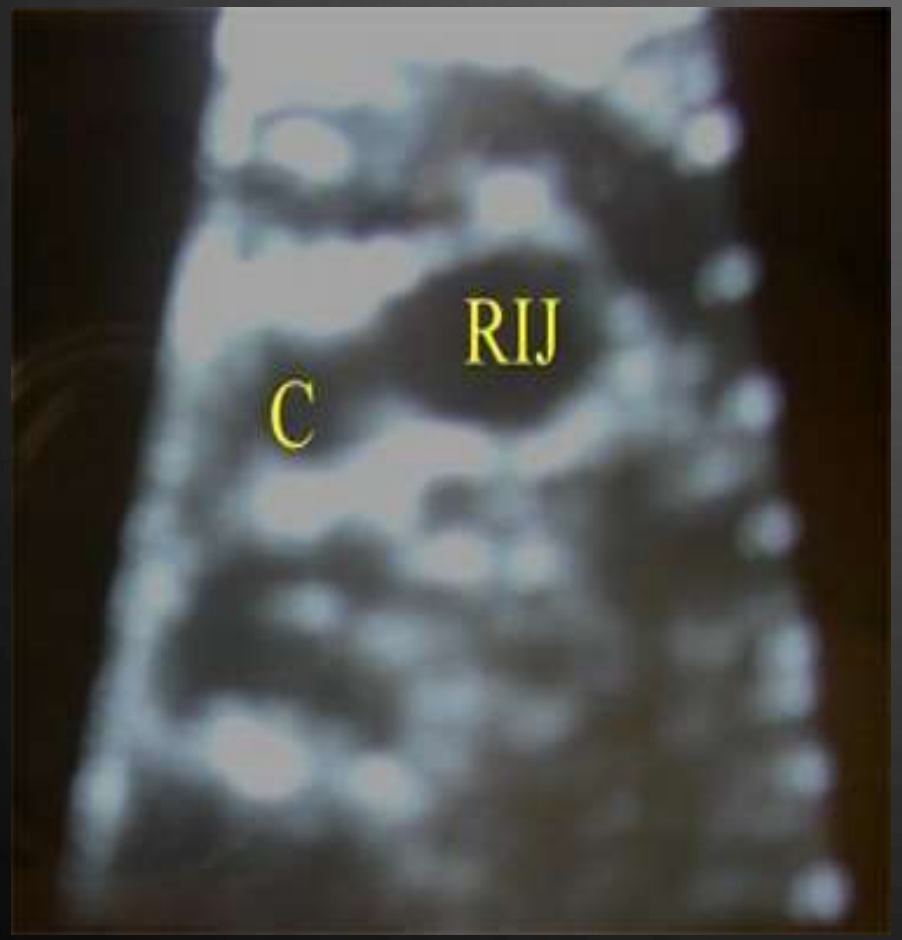
INVESTIGATIONS



PNEUMOTHORAX PREVENTION DURING CVCS

- Remove patient from ventilator before advancing the needle.
- Choose the right side rather than left,
- Avoid multiple attempts when possible
- Check post procedure x-ray,

ULTRASOUND GUIDED CVCS



- SUCCESS WITH ULTRASOUND GUIDANCE WAS 100%, COMPARED WITH 88% WHEN ULTRASOUND WAS NOT USED.
- INCIDENCE OF CAROTID PUNCTURE WAS REDUCED FROM 8.3% TO 1.7%.

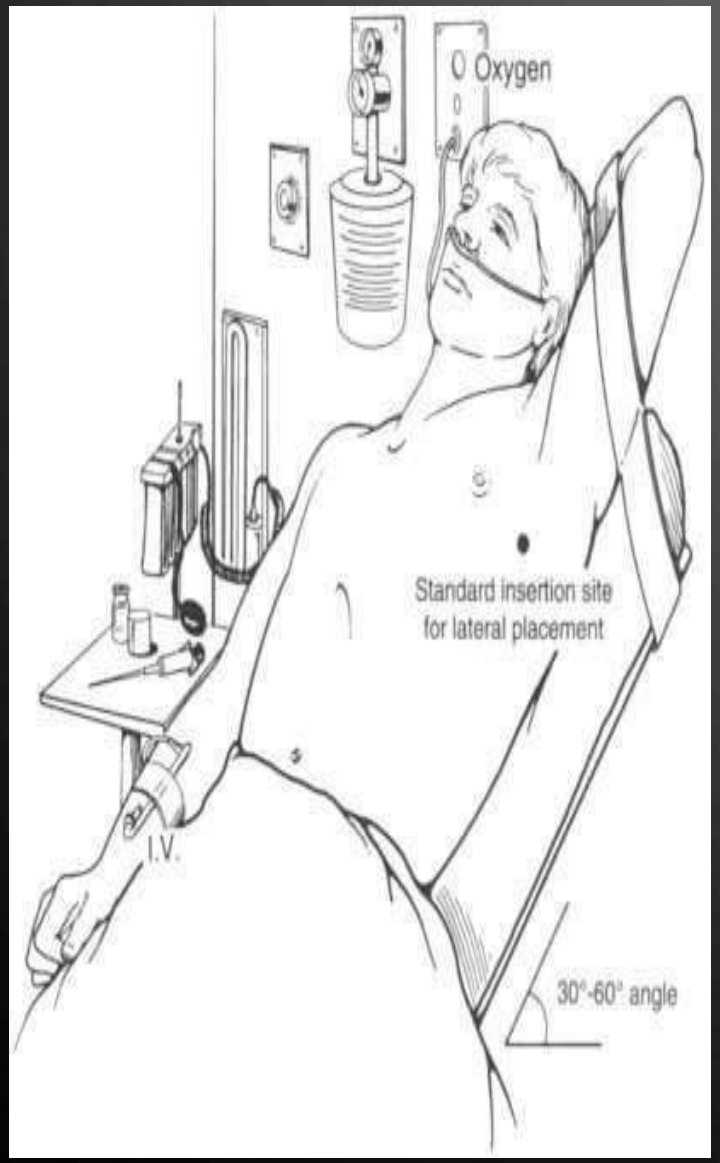
TREATMENT

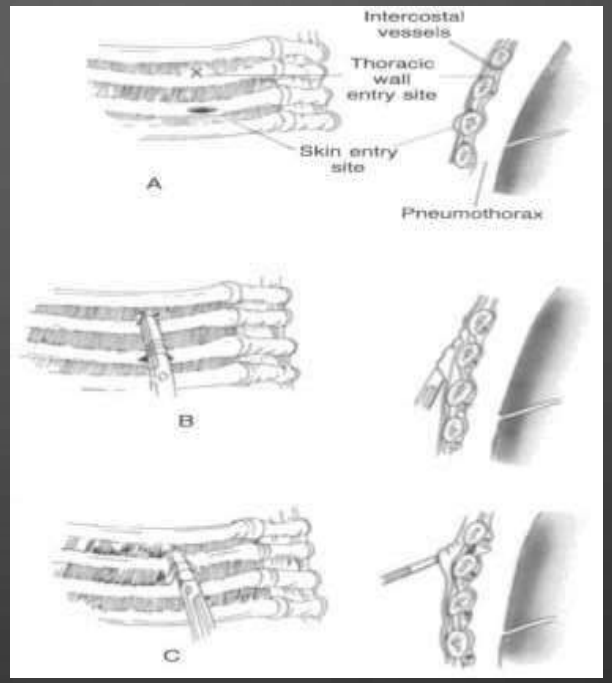
- Small pneumothorax
  - Resolve over days to weeks
  - Supplemental oxygen and observation
- Spontaneous pneumothorax
  - Asymptomatic –f/u with serial CXR
  - Symptomatic –chest tube
  - Recurrent pneumothorax – CT to evaluate need for thoracotomy

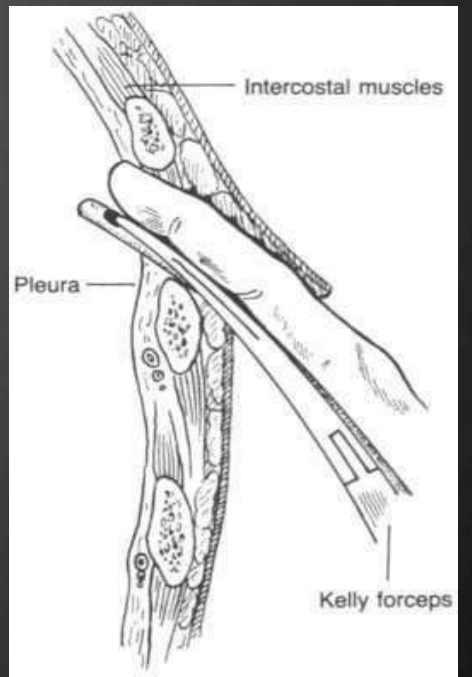
TENSION PNEUMOTHORAX; TREATMENT

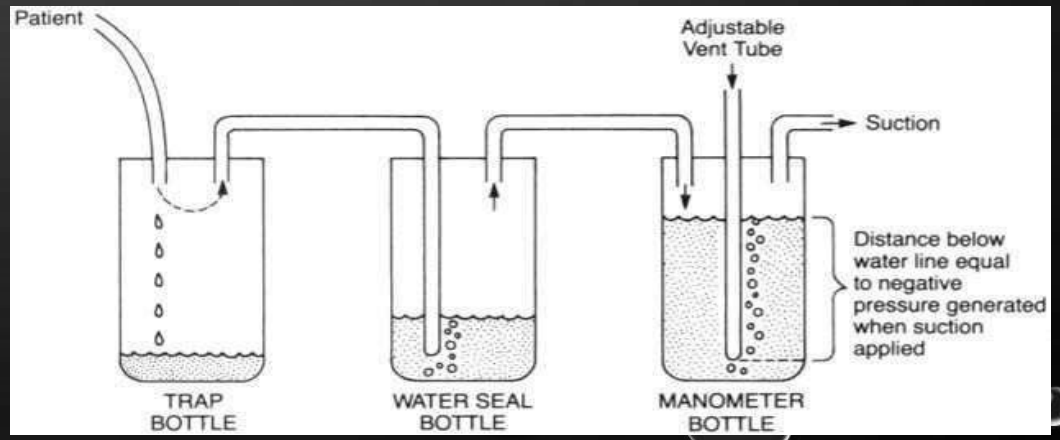
- Tension-pneumothorax can be life-threatening.
- The immediate treatment is insertion of a chest tube.
- Chest tubes are generally inserted using local anesthesia.
- The chest tube is left in place until the lung leak seals on its own; this usually occurs within two to five days.

THORACOSTOMY (CHEST TUBE)

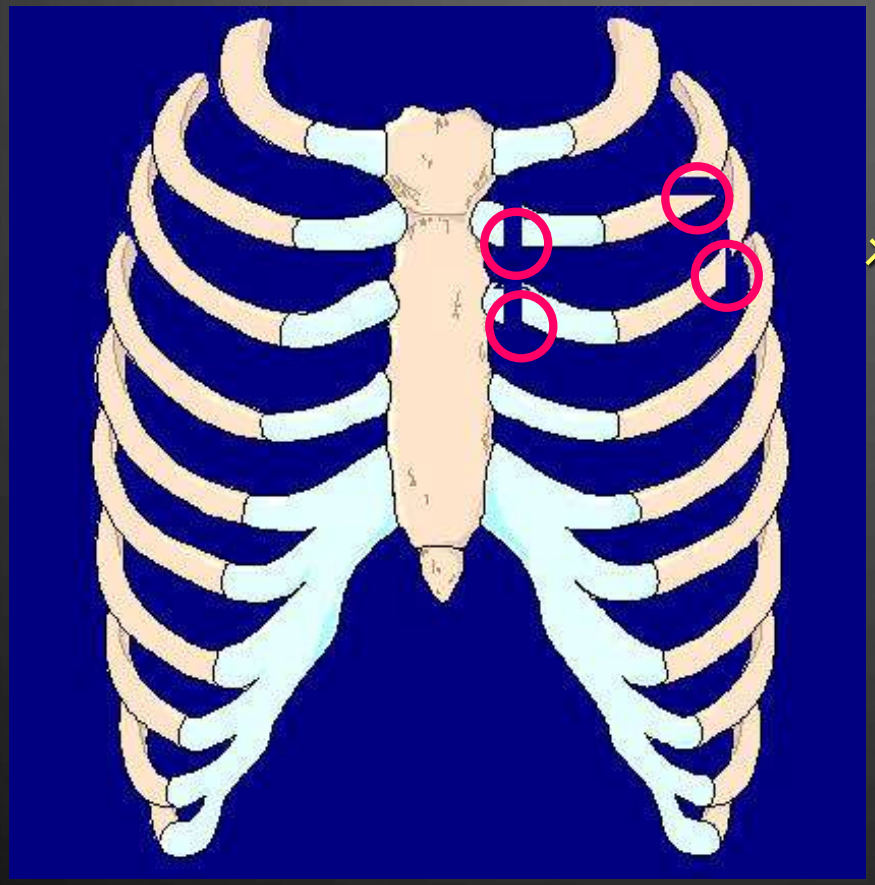






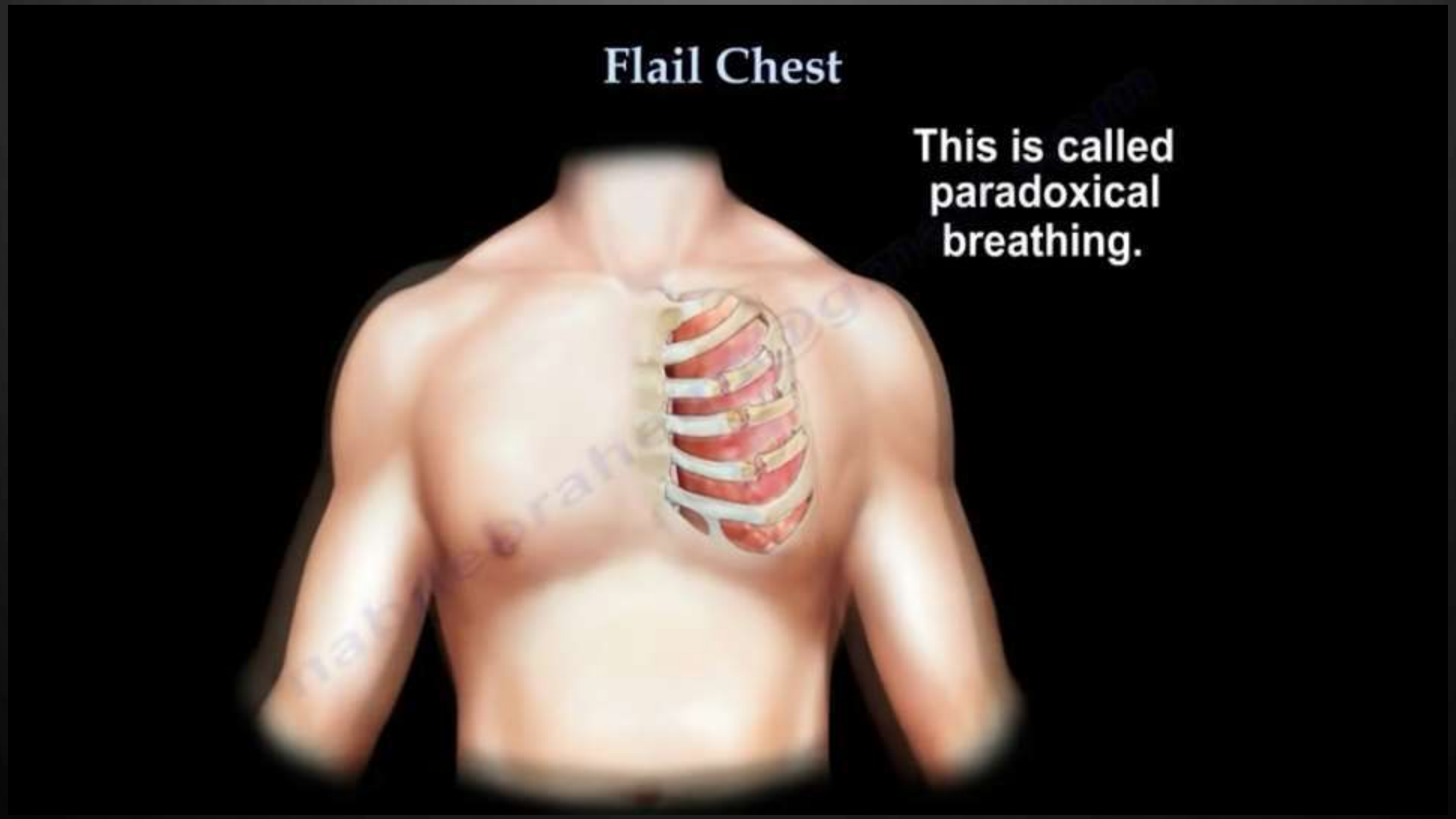


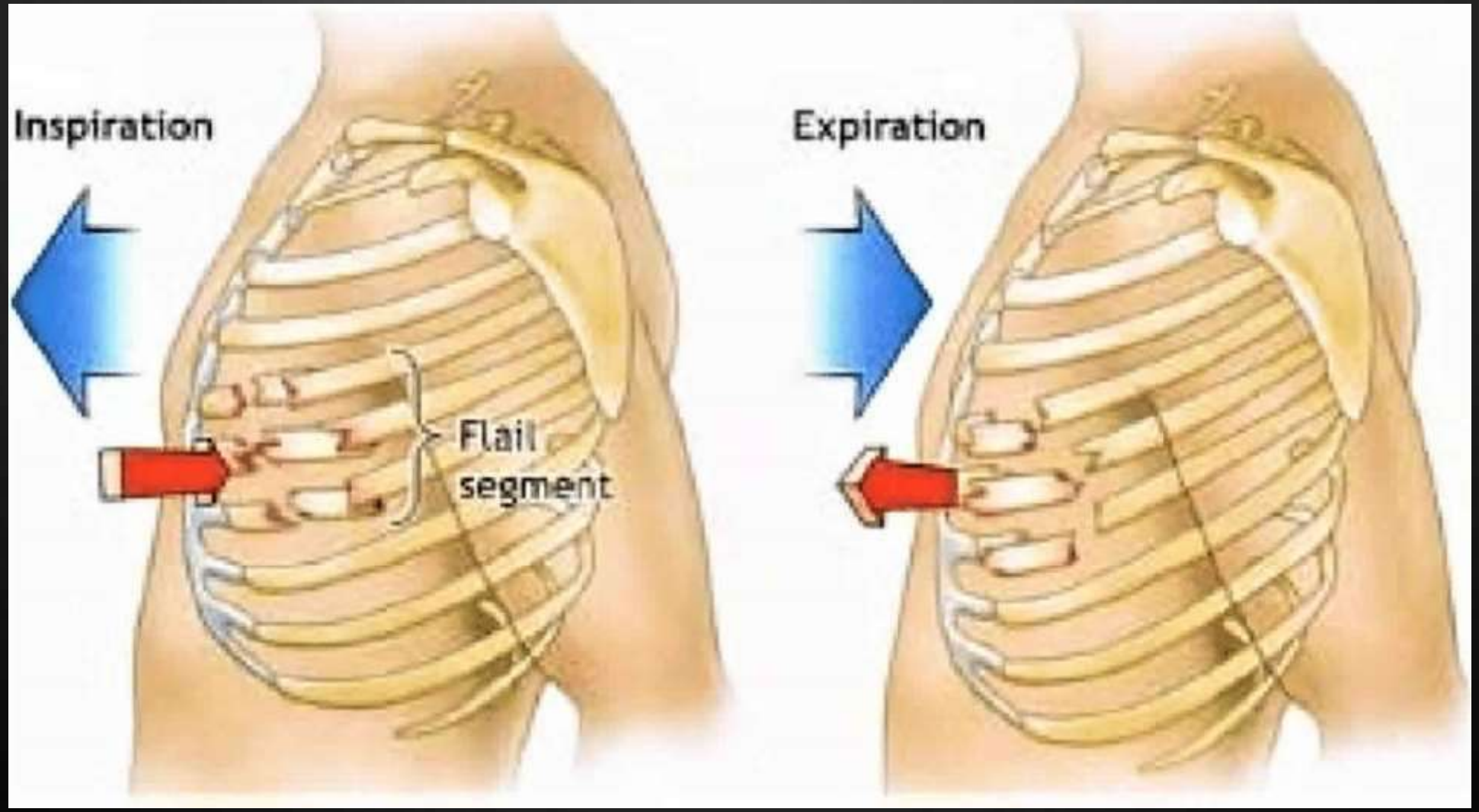
FLAIL CHEST



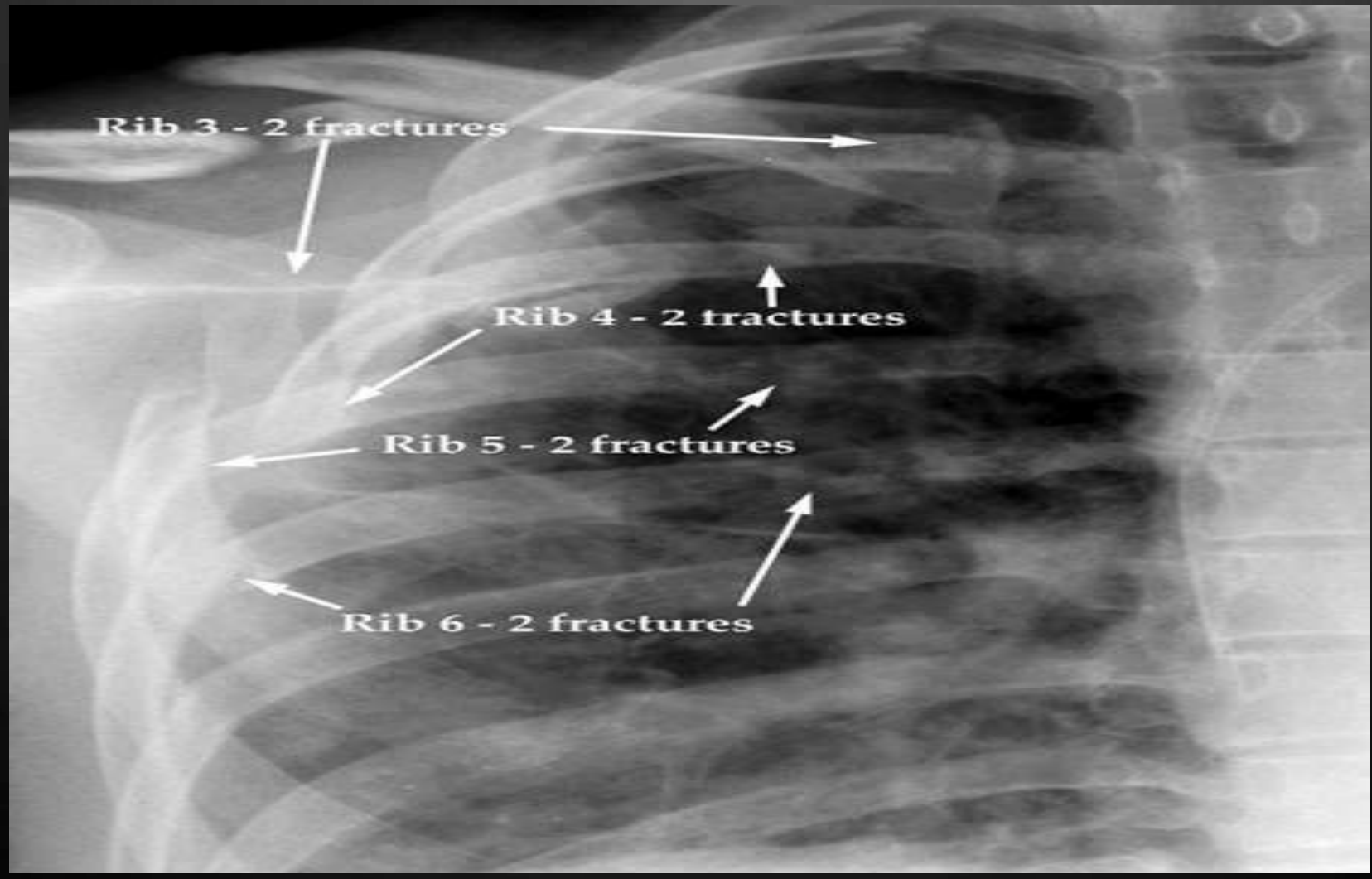
- The breaking of 2 or more ribs in 2 or more places

FLAIL CHEST




Inspiration
Flail segment
Expiration

FLAIL CHEST


Rib 3 - 2 fractures
Rib 4 - 2 fractures
Rib 5 - 2 fractures
Rib 6 - 2 fractures

FLAIL CHEST IS A TRUE EMERGENCY

THANK YOU

STRESS ULCER

Ahmed Maki Radeef (M.Sc. Anesthesia and ICU Technology)

INTRODUCTION

- Stress ulcerations are common in intensive care unit (ICU) patients, some of which can cause hemorrhage. As a consequence, many critically ill patients require prophylaxis for primary prevention of bleeding from stress ulceration or treatment for stress ulcer-related bleeding.

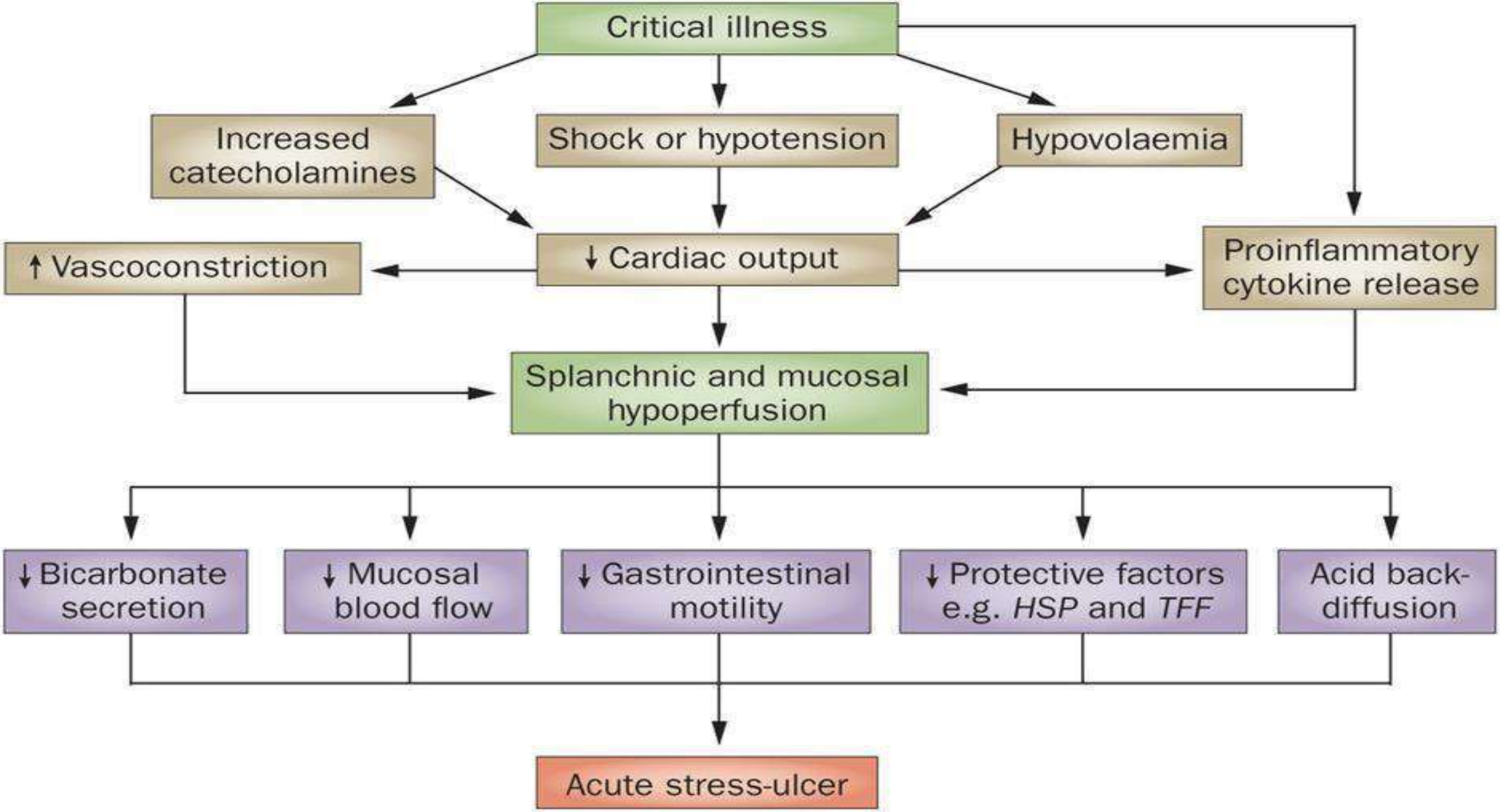
DEFINITION

Stress ulceration is defined as ulceration of the upper gastrointestinal (GI) tract (esophagus, stomach, duodenum) that occurs **due to** hospitalization. Hemorrhage from stress ulceration is secondary GI bleeding (ie, nosocomial GI bleeding), which is distinct from primary GI bleeding (ie, GI bleeding that **results in** hospitalization). Primary prevention of GI bleeding from stress ulcers is known as stress ulcer prophylaxis (SUP).

PATHOPHYSIOLOGY

In most critically ill patients, the gastric mucosal blood flow is impaired. Reasons include systemic hemodynamic changes (hypotension and/or vasopressor therapy) and/or local alterations, *e.g.*, reduced splanchnic blood flow because of positive end-expiratory pressure in mechanical ventilated patients. In addition to the ischemic tissue damage itself, hypoperfusion leads to a reduced production of several protective mechanisms that exist in a healthy stomach. The latter include various components such as mucus, phospholipids, bicarbonate, trefoil factor family peptides and heat-shock proteins. For example, gastric ischemia/reperfusion in an experimental

Moreover, the production of the vasodilator nitric oxide is reduced, the level of endothelin-1, a strong vasoconstrictor, is significantly increased. This shift can further harm the mucosa. While these mechanisms can cause mucosal damage, they are often insufficient by themselves to cause major ulcerations and gastric bleeding. A crucial component for overt damage is the presence of gastric acid. This provides the rationale for the use of acid-suppressive drugs such as PPI or H2RA for pharmacological prophylaxis.


Critical illness
Increased catecholamines
Shock or hypotension
Hypovolaemia
↑ Vascoconstriction
↓ Cardiac output
Proinflammatory cytokine release
Splanchnic and mucosal hypoperfusion
↓ Bicarbonate secretion
↓ Mucosal blood flow
↓ Gastrointestinal motility
↓ Protective factors e.g. *HSP* and *TFF*
Acid back-diffusion
Acute stress-ulcer

RISK FACTORS FOR STRESS ULCERRELATED BLEEDING

Multiple investigations have been conducted to identify patients at risk for stress ulcer-related bleeding. A large, prospective multicenter was able to identify two main risk factors: mechanical ventilation and coagulopathy. Other high risk :head/spinal cord trauma-gcs<10, acute kidney failure, acute liver failure, hypotension/sepsis/septic shock, ICU stay> 1 week, high dose corticosteroids and major surgery

PHARMACOLOGICAL PROPHYLAXIS

If a stress ulcer prophylaxis is necessary, different options are available: Options include the acid-suppressing drugs, PPI and H2RA, or the mucosa-protective agent sucralfate. Sucralfate is a reasonable option and reduces the risk of stress ulcer-related bleeding. However, a large trial revealed its inferiority to H2RA, so that an acid suppressive medication is preferred for SUP.

SUCRALFATE

- It exerts its effects by coating and protecting the gastric mucosa, without altering gastric acid secretion or significantly buffering acid.
- Administered orally or via NGT at a dose of 1 gram four times per day.

- Use of sucralfate should be avoided in patients with compromised renal function to avoid aluminum accumulation and poisoning

PPI

- Block acid secretion by **irreversibly** binding to and inhibiting the hydrogen-potassium ATPase pump that resides on the luminal surface of the parietal cell membrane.
- omeprazole, lansoprazole, rabeprazole, pantoprazole, esomeprazole.
- PO/NG/İV
- Rapid onset of action, linear kinetics, longer duration of action, and lack of observed tolerance.
- Not renally eliminated.

H2 RECEPTOR ANTAGONISTS (H2RA)

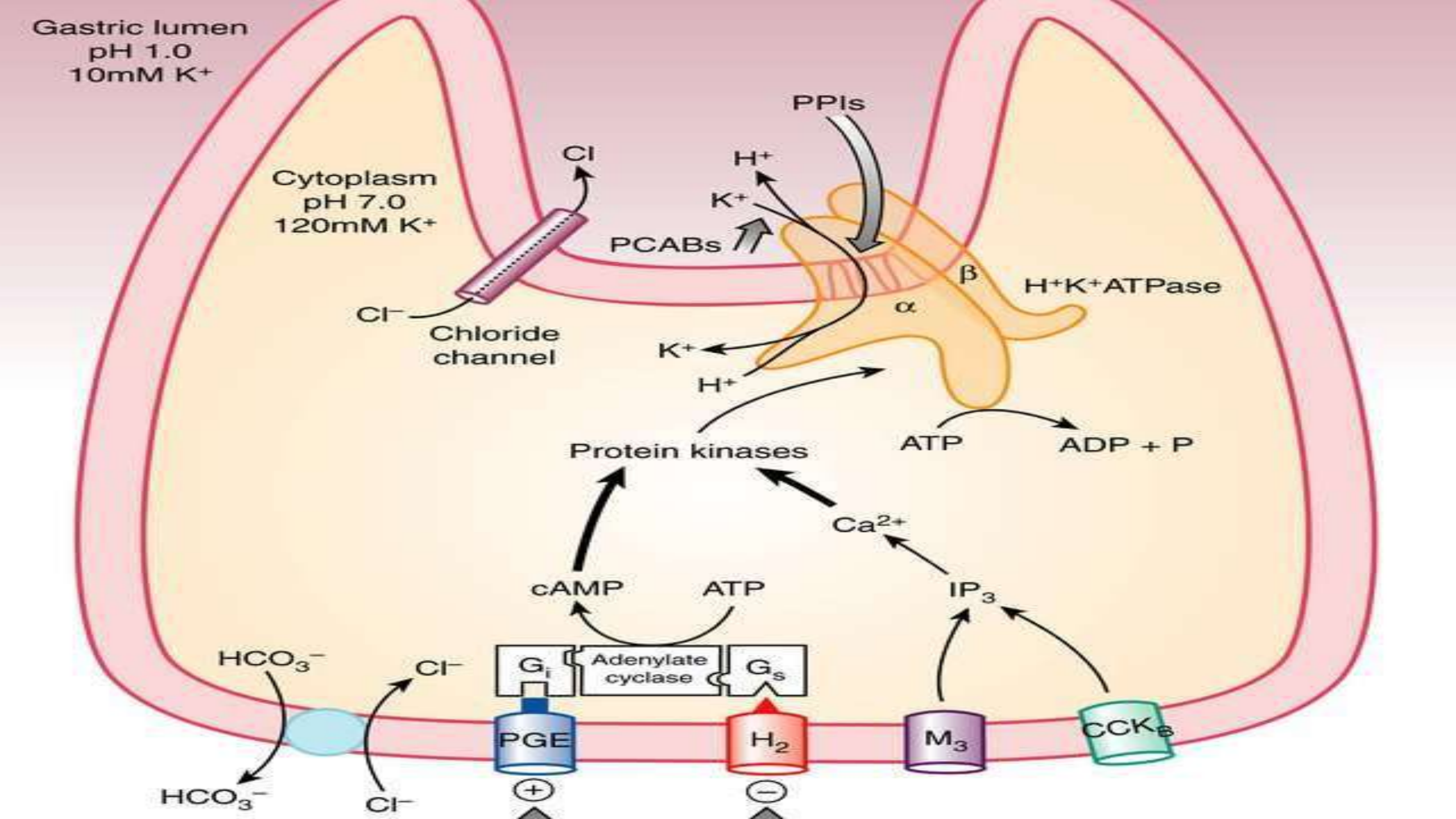
- Act by decreasing gastric acid secretion through **reversible, competitive inhibition** of histamine stimulated acid secretion and are effective in reducing basal acid production.
- Acid suppression with H2RA is incomplete.
- Cimetidine, Ranitidine, Famotidine, Nizatidine.

ANTIACID

- Antacids neutralize gastric acid and protect the gastric mucosa.
- Administered every one to two hours at a dose of 30 to 60 mL either orally or via nasogastric tube.
- Side effects include hypermagnesemia, hypercalcemia, hypophosphatemia, constipation, and diarrhea.

PROSTANOID

- Misoprostol , inhibit gastric acid secretion by selectively reducing the ability of the parietal cell to generate cyclic AMP in response to histamine.
- Exert a cytoprotective effect by enhancing mucosal defense mechanisms.
- Found ineffective in stress ulcer prophylaxis


Gastric lumen
pH 1.0
10mM K+
Cytoplasm
pH 7.0
120mM K+
Cl
Cl–
Chloride channel
PPIs
H+
K+
PCABs
β
α
H+K+ATPase
K+
H+
Protein kinases
ATP
ADP + P
Ca2+
IP3
cAMP
ATP
Gi
Adenylate cyclase
Gs
HCO3–
Cl–
HCO3–
Cl–
PGE
H2
M3
CCKB
+
–

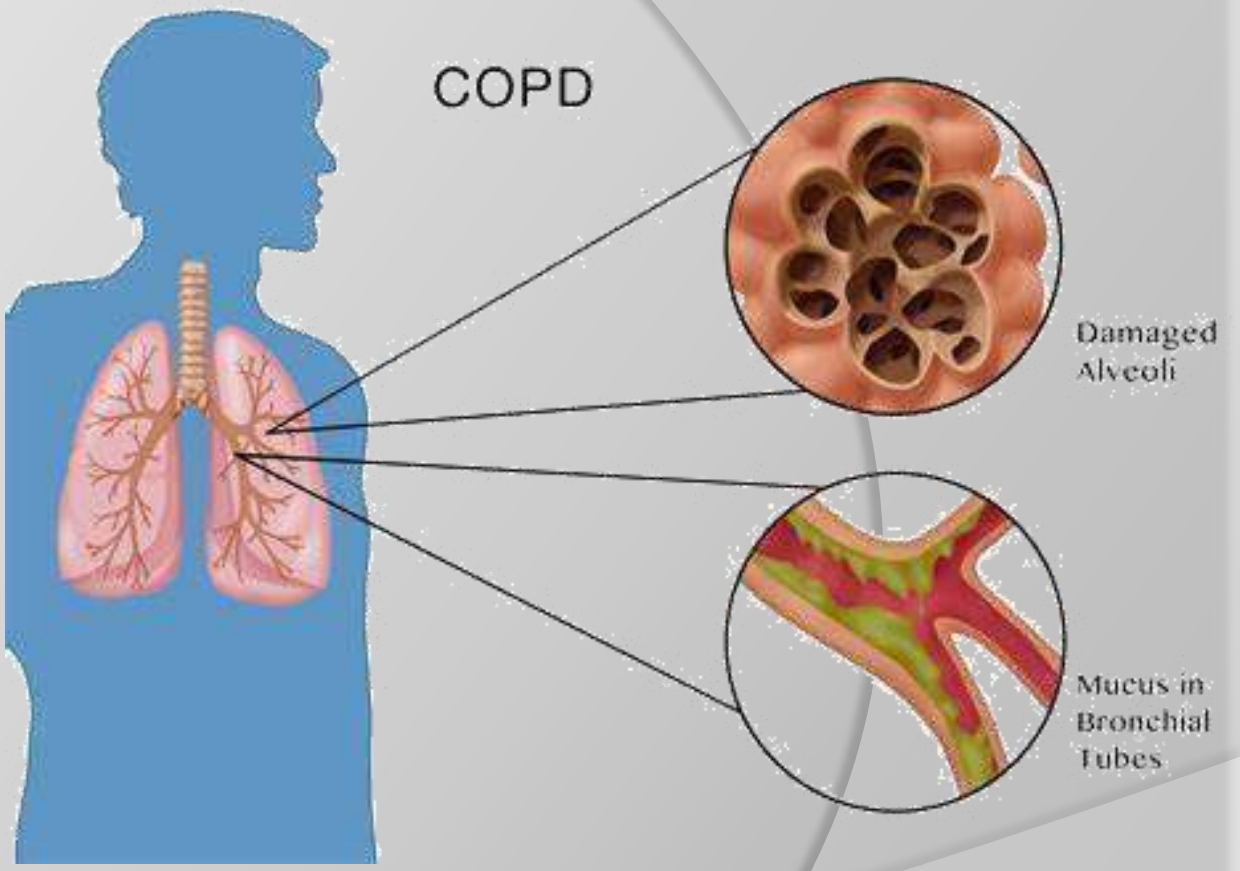
NUTRITION

Enteral Feeding

- Enteral feedings initiated within 12 hours of trauma were as effective as H2RA.
- The risk of clinically important gastric bleeding was reduced in patients receiving enteral feedings.

THANK YOU

Chronic Obstructive Pulmonary Disease



Ahmed Maki Radeef (M.Sc. Anesthesia Technology)

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by airflow limitation that is not fully reversible. COPD includes emphysema; chronic bronchitis; and small airways disease.

COPD is present only if chronic airflow obstruction occurs; chronic bronchitis without chronic airflow obstruction is not included within COPD.

COPD is the fourth leading cause of death and affects >10 million persons in the United States. Estimates suggest that COPD will rise from the sixth to the third most common cause of death worldwide by 2020.

Oxygen Supplemental

Oxygen Supplemental O2 should be supplied to keep arterial saturations ≥90%. Hypoxemic respiratory drive plays a small role in patients with COPD. Studies have demonstrated that in patients with both acute and chronic hypercarbia, the administration of supplemental O2 does not reduce minute ventilation. It does, in some patients, result in modest increases in arterial PCO2, chiefly by altering ventilation-perfusion relationships within the lung. This should not deter practitioners from providing the oxygen needed to correct hypoxemia.

COPD and Hypoxic Drive: Mechanism

It is common dogma on the wards that oxygen therapy for chronic CO2 retainers should be targeted between 88-92% during a COPD exacerbation.

The mechanism often quoted is the “hypoxic drive to breath“. The idea is that COPD patients tend to have chronically elevated levels of carbon dioxide due to the nature of their illness. As such, administering oxygen to these patients with COPD can be dangerous.

COPD and Hypoxic Drive: Mechanism

Their chronically elevated carbon dioxide levels result in loss of the hypercapnic-mediated respiratory drive and they rely solely on their "hypoxic" drive to breath. The patient's chemo-receptors are already tolerant of high levels of carbon dioxide.

However, also contributory to the desaturation seen with higher oxygen levels is the Haldane effect. The Haldane effect states that deoxygenated hemoglobin has a higher affinity for CO2 because it is a better proton acceptor than oxygenated hemoglobin.

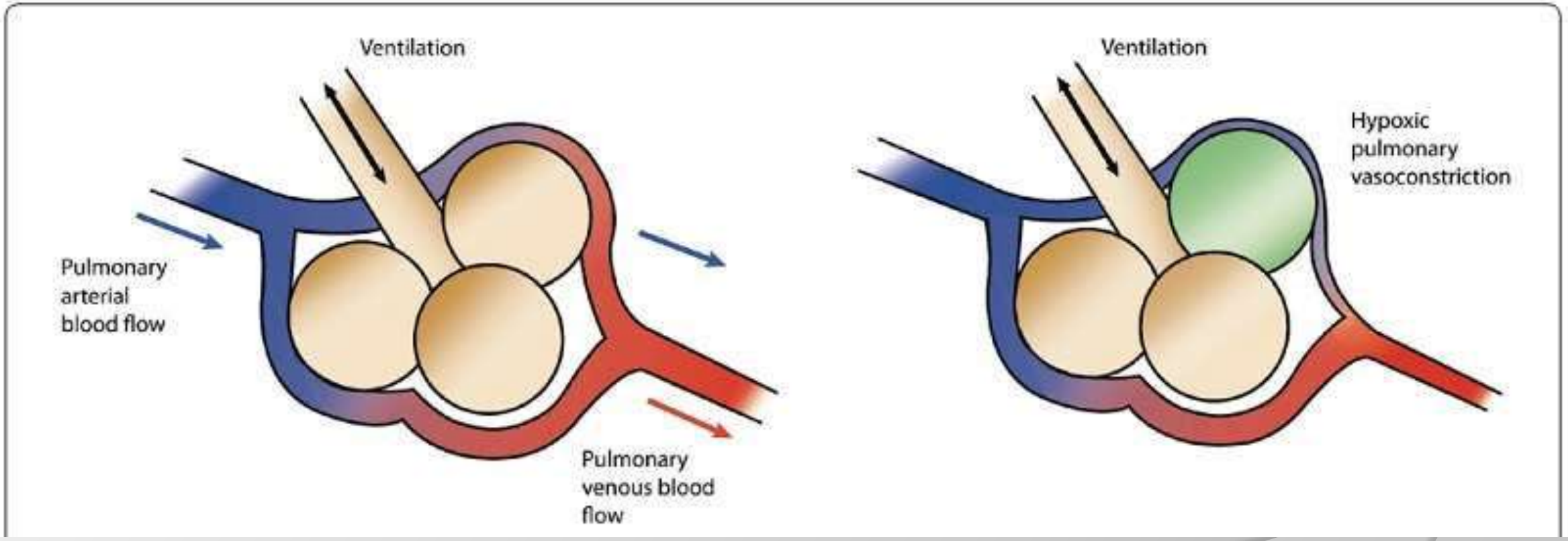
COPD and Hypoxic Drive: Mechanism

Therefore, increasing oxygen concentration in the blood by giving patients supplemental oxygen means carbon dioxide molecules will be displaced in favour of the oxygen, thereby reducing alveolar expulsion.

When alveolar oxygen tension is reduced (i.e bronchoconstriction, mucus plugging), it induces vasoconstriction of pulmonary capillaries supporting the affected alveoli. This is meant to counteract possible shunting and normalize the V/Q ratio, a mechanism called hypoxic pulmonary vasoconstriction. The strongest determinant for hypoxic pulmonary vasoconstriction is the alveolar partial pressure of oxygen.

COPD and Hypoxic Drive: Mechanism

Therefore, providing a high fraction of inspired O2 (FiO2) will increase O2 tension in alveoli with a low level of ventilation (i.e scarred alveoli in setting of COPD), inhibiting hypoxic pulmonary vasoconstriction. As a result, alveoli with relatively impaired ventilation (which would be vasoconstriction normally) are no longer, leading to an increase in V/Q mismatch.


Ventilation
Pulmonary arterial blood flow
Pulmonary venous blood flow
Ventilation
Hypoxic pulmonary vasoconstriction

COPD and Hypoxic Drive: Mechanism

According to Donald F. Egan's, CO_2 is normally the drive to breath. But, if a patient with COPD is having so much trouble breathing that there is no way possible that he can speed up his breathing further to blow off that excess CO_2 "regardless of patient effort," CO_2 no longer is the drive to breathe, and PO_2 becomes the drive to breathe.

Mechanical Ventilatory Support

The initiation of noninvasive positive-pressure ventilation (NIPPV) in patients with respiratory failure, defined as $PaCO_2$ >45 mmHg, results in a significant reduction in mortality rate, need for intubation, complications of therapy, and hospital length of stay. Contraindications to NIPPV include cardiovascular instability, impaired mental status or inability to cooperate, copious secretions or the inability to clear secretions, craniofacial abnormalities or trauma precluding effective fitting of mask, extreme obesity, or significant burns.

Mechanical Ventilatory Support

Invasive (conventional) mechanical ventilation via an endotracheal tube is indicated for patients with severe respiratory distress despite initial therapy, life-threatening hypoxemia, severe hypercarbia and/or acidosis, markedly impaired mental status, respiratory arrest, hemodynamic instability, or other complications. The goal of mechanical ventilation is to correct the aforementioned conditions.

Mechanical Ventilatory Support

Factors to consider during mechanical ventilatory support include the need to provide sufficient expiratory time in patients with severe airflow obstruction and the presence of auto-PEEP (positive end-expiratory pressure), which can result in patients having to generate significant respiratory effort to trigger a breath during a demand mode of ventilation. The mortality rate of patients requiring mechanical ventilatory support is 17–30% for that particular hospitalization. For patients age >65 admitted to the intensive care unit for treatment, the mortality rate doubles over the next year to 60%, regardless of whether mechanical ventilation was required.

Bronchodilators

Typically, patients are treated with an inhaled β agonist, often with the addition of an anticholinergic agent. These may be administered separately or together, and the frequency of administration depends on the severity of the exacerbation. Patients are often treated initially with nebulized therapy, as such treatment is often easier to administer in older patients or to those in respiratory distress.

This approach has significant economic benefits and also allows an easier transition to outpatient care. The addition of methylxanthines (such as theophylline) to this regimen can be considered, although convincing proof of its efficacy is lacking. If added, serum levels should be monitored in an attempt to minimize toxicity.

Glucocorticoids

Among patients admitted to the hospital, the use of glucocorticoids has been demonstrated to reduce the length of stay, hasten recovery, and reduce the chance of subsequent exacerbation or relapse for a period of up to 6 months. One study demonstrated that 2 weeks of glucocorticoid therapy produced benefit indistinguishable from 8 weeks of therapy.

The GOLD guidelines recommend 30–40 mg of oral prednisolone or its equivalent for a period of 10–14 days. Hyperglycemia, particularly in patients with preexisting diagnosis of diabetes, is the most frequently reported acute complication of glucocorticoid treatment.


Thank You
For Your
Attention

محاضرة (١)

عناية مركزة عملي

د. علاء غانم الكواز

PACU
(Post anesthetic care unit)

Also called post anesthetic recovery unit or recovery room

Definition: it is a special unit located close to operating theatre, provides monitoring, closeobservation and care to patient after anesthesia and surgery. It should have 1.5- 2 beds per operating room used beds and stafted with 1:2 nurse to patient ratio for stable patients, 1:1 ratio for unstable patients.

Usually patient stay for 1 hr and it can be hold for 6hr or more according to patient condition.

PACU requirements includes:

1. Contains monitoring equipments which include ECG, oximeter, capnograph, BP monitoring & temperature.
2. Ventilation equipments which include oxygen source, face masks, Ambu bag endotracheal tubes, suction device and ventilator .
3. Portable defibrillator.
4. Intravenous fluids: crystalloid and colloid with I.V cannula and giving sets.
5. Essential and emergency drugs.

Aim of PACU

1. Full recovery with full return of consciousness.
2. Return of muscle power eg. protruding of tongue, grasping of hand, elevation of head for 5 seconds.
3. Return of protective airway reflexes.
4. Respiratory system stability: normal respiration with normal color.

5. CVS stability- normal BP, HR and rythm.
6. Normal body temperature; no hyperthermia or hypothermia.
7. Control of nausea & vomiting.
8. Control of bleeding.
9. Control of pain.

Rules for PACU

1. Patient should transported from operation room to PACU by member of anesthesia team who know about patient operation and condition.
2. Patient should be received by anesthetic member in PACU with verbal report about his condition.
3. Patient should be evaluated continuously by the nurse.
4. The decision of discharge from PACU is the responsibility of anesthesia doctor.

Criteria of discharg from PACU

1. Conciausness level: Patient fully awake and should respond to verbal stimulation, oriented and be able to answer question appropriately.
2. Normal vital signs: with CVS and respiratory system stability.
3. Good muscle power and normal reflexes.
4. Normal fluid balance and body temperature.
5. Pain control, with no nausea or vomiting.
6. No bleeding at wound site (dressing and drains).
7. For spinal and epidural anesthesia, feeling his legs with ability to move the knee joint.

محاضرة (١)

عناية مركزة عملي

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محاضرة (١) عناية نظري

د. علاء غانم الكواز

Principles of intensive care unit (ICU)

(Critical care unit / Intensive therapy unit / Intensive treatment unit ITU)

Definition of intensive care unit :

Intensive care units (ICUs) are special ward in the hospital that provide management to critically ill patients(risky patients), that their condition is unstable, life threatening or potentially threatening.

The ICU usually staffed with well trained nurses & medical professionals & contains special advanced monitoring equipments & life support medications.

Patients may be referred directly from an emergency department or from a ward if they rapidly deteriorate, or immediately after surgery if the surgery is very invasive and the patient is at high risk of complications.

HOW INTENSIVE CARE UNITS WERE BORN?

Harvey Cushing was the most brilliant brain surgeon of his generation. His patients adored him, describing him as caring and kind, but he kept his staff in a state of terror. He was intolerant of mistakes and could be cold, harsh and bullying, but he was forgiven, because his results spoke for him.

• Before Cushing, 8 out of 10 brain surgery patients died. In his hospital, the surgeon reduced mortality to just 8%.

• In a time before antibiotics, and with the risk of bacterial infection killing anyone going under the knife. Cushing operated under the strictest cleanliness, he wore gloves and a mask, doing whatever he could to sterilize the wound and reduce the chance of disease,and continued the care after he had finished his operations, the period when patients were at greatest risk of dying.

Cushing carried over his meticulousness during surgery to the post-operative care of the patients

• He often tended to patient’s wounds himself, ensuring they were kept free of infection. He introduced strict observation systems and record keeping, the first widespread use of x-rays and blood pressure

monitoring. Each individual patient was the focus of care by a team of specialist staff.

- Cushing's whole ward was more like an intensive care unit than other surgeons,“The nurses and junior surgical staff knew that if the bed sheets were not tucked in properly, the dressing wasn't clean or the patient was complaining, they were going to be in big trouble.”

The polio epidemic in Copenhagen resulted in 316 patients developing respiratory muscle paralysis and/or bulbar palsy, with subsequent respiratory failure and pooling of secretions. The Blegham Hospital, the hospital in Copenhagen for communicable diseases, had only one tank respirator and six cuirass respirators at the time. This was completely inadequate to support the hundreds of polio patients with respiratory failure and bulbar involvement .the mortality rate from polio was 85–90% and, as the epidemic progressed, the situation looked desperate.

Professor Lassen, chief physician at the Blegdam Hospital, had a strong desire to provide treatment for all polio victims, despite insufficient respirators, and therefore consulted with Dr Bjorn Ibsen, a Copenhagen anaesthetist. Professor Lassen hoped that positive pressure ventilation, as used in modern anaesthesia at that time, might be a solution. Two days later, a 12-year-old girl with polio and resultant respiratory failure and bulbar palsy had a tracheostomy formed just below the larynx: a rubber cuffed tracheostomy tube was inserted and positive pressure ventilation successfully delivered manually with a rubber bag. Tracheostomies had been performed in Copenhagen 4 years before this, but with little beneficial effect on outcome.

• Dr Ibsen had the idea of caring for all such patients in a dedicated ward, where each patient could have their own nurse. Thus, in December 1953, the specialty of intensive care was born.

Intensive care has undergone enormous change since the establishment of the specialty more than 65 years ago, and further changes will undoubtedly be seen in coming years.

Definition of critical care

It is a term used to describe the care of patients who are extremely ill/ or whose clinical condition is unstable or life threatining.

Intensive care medicine, also called critical care medicine, is a medical specialty that deals with seriously or critically ill patients who have, or at risk of, or are recovering from conditions that may be life-threatening. It includes providing life support, invasive monitoring techniques, resuscitation, and end-of-life care Patients

Aim of critical care:

1. Saving the life after primary insult by providing optimal therapy to critically ill patient, and treating the immediate life-threatening condition of the patient.
2. Maintain vital organ system functioning to improve the condition of the patients until the underlying injury or illness can be treated.
3. Using advanced therapeutic, monitoring and diagnostic technology.

* Intervals of ICU medical measures(managements) :

One important key of success in any ICU is depending on good understanding of the functions of ICU services. There are 2 intervals of ICU medical measures :

1. **Period of supportive measures :**
   during early acute period of primary pathology till the point when definitive diagnosis is reached, supportive measures will help to delay the collapse of body's vital functions.
2. **Period of therapeutic measures :**
   period from the point of primary diagnosis with putting the plan of specific therapeutic measures throughout the duration of management until recovery.

Difference of ICU ward from usual medical ward in the hospital

1. ICUs take care of patients with severe injuries or life threatening illnesses , which require life support equipments, close observation & monitoring.

2. The ICUs contains highly trained nurses, Anesthetists, physicians, respiratory therapists who are specialized in dealing with critically ill patients.
3. ICUs also characterized by high staff- patient ratio (eg. 1:2 or 1:1).
4. It contents advanced medical equipments and resources that is not routinely available in other hospital ward.

High dependency unit

It is a medical ward that provides a level of care intermediate between intensive care unit & general ward care. most acute hospitals have a transitional high dependency unit (HDU) for patients who require close observation, treatment and nursing care that cannot be provided in a general ward, but whose care is not at a critical stage to warrant an (**ICU).**

Functions of ICU

It is to provides supportive medical care for critically ill patients which include:

1. Respiratory care
2. Circulatory care
3. Fluid & Electrolytes balance
4. Acid- base balance
5. Metabolic status monitoring and regulations.
6. Nutritional care, (Enteral Feeding, parenteral feeding).

7. Physiotherapy & rehabilitation.

8. Psychotherapy.

Types of ICU

A. General types:

1. **Medical intensive care unit (MICU)**
2. **Surgical intensive care unit (SICU)**
3. **Main or mother intensive care unit (Main.I.C.U)** which is combined medical & surgical & Trauma ICU.

B. Specialized type:

1. **Respiratory care unit (RCU):** providing care for patient with respiratory diseases as respiratory failure and severe respiratory infection eg severe pneumonia.
2. **Post-anesthetic care unit (PACU):** Also known as the post-operative recovery unit, or recovery room, the PACU provides immediate post-op observation and stabilization of patients following surgical operations and anesthesia usually inside or near operating theater, patient can be held for 6 hr or more.
3. **Neonatal intensive care unit (N.I.C.U):**

   This specialty unit cares for neonatal patients who have not left the hospital after birth. Common conditions cared for include prematurity and associated complications, congenital disorders such as congenital diaphragmatic hernia, or complications resulting from the birthing process.
4. **Pediatric intensive care unit (P. I. C. U):**

   Pediatric patients are treated in this intensive care unit for life threatening conditions such as asthma, influenza, diabetic ketoacidosis, or traumatic neurological injury.

5. **Coronary care unit (CCU):**

   Also known as Cardiac Intensive Care Units (CICU) or Cardiovascular Intensive Care Unit(CVICU), this ICU caters to patients specifically with congenital heart defects or life threatening cardiac conditions such as a myocardial infarction or a cardiac arrest.

6. **Neurosurgery intensive care unit (NSICU):**

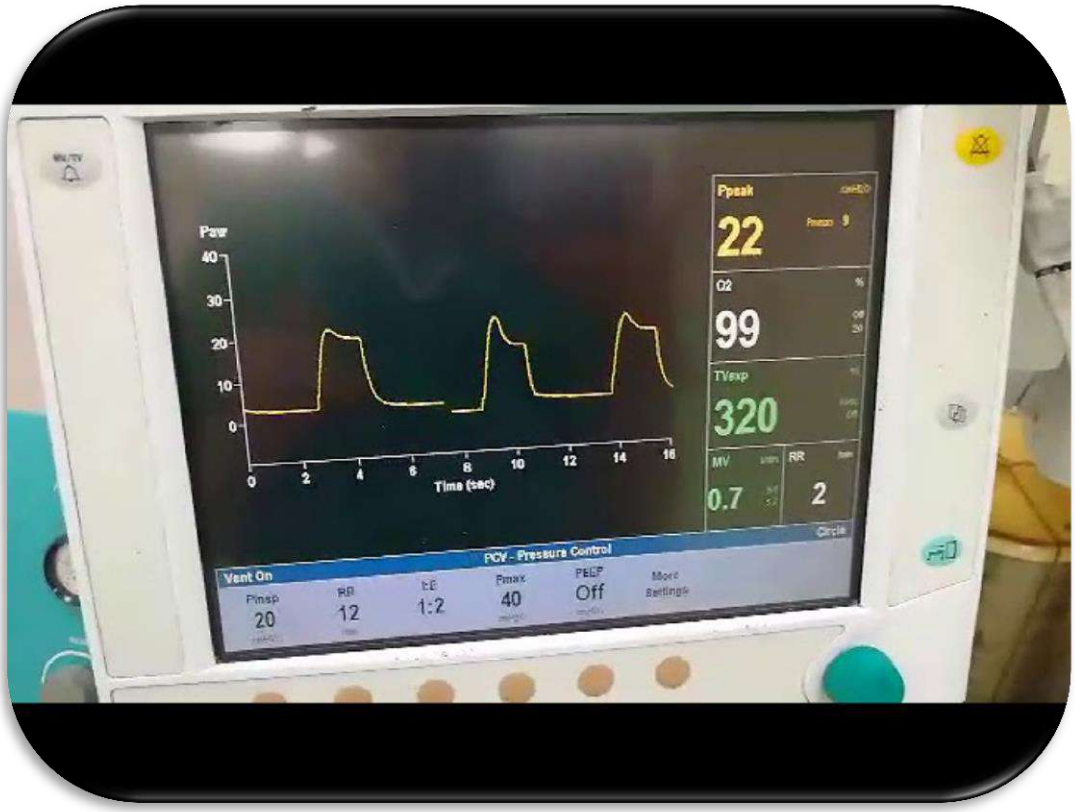
   Patients are treated for head injury ,brain aneurysms, brain tumors, stroke, and post-surgical patients who have undergone various neurological surgeries performed by experienced neurosurgeons require constant neurological exams. Nurses who work within these unitshave neurological certifications.

7. **High dependency unit (HDU):** mentioned above

محاضرة ٢
عناية مركزة عملي
علاء غانم الكواز

Ventilator setting parameters

1. FiO_2 (fraction of inspired oxygen).
2. V_T (tidal volume).
3. R R (Respiratory rate) or frequency (f) or Breathing rate.
4. V_E (minute ventilation).
5. 1: E ratio (inspiration to expiration ratio).
6. Paw (Peak airway pressure).
7. Trigger sensitivity.
8. Peak flow/ flow rate.
9. Sigh.



1. FiO_2

The percentage of oxygen concentration delivered to the patient by ventilator.

Initially we set FiO_2 on high level & then we decrease it gradually according to SPO_2 and ABGs.

FiO_2 percent is between 21-100%. Initial FiO_2 may be set on 100% until arterial blood has adequate oxygenation ,then we decrease it to 60% or less according to the oxygenation.

The FiO_2 is adjusted to maintain SPO_2 above 90% (which equal to 60mm Hg PaO_2 by ABG).

High FiO_2 protect against hypoxemia but it can lead to oxygen toxicity which may occur if FiO_2 is greater than 60% for more than 24 hr.

In premature infants high FiO_2 may lead to blindness.

Signs and symptoms of oxygen toxicity

- Dyspnea and chest pain or tightness of the chest.
- Dry cough.
- Flushed face.
- Dizziness.
- Blurred vision.
- Twitching & muscles especially of face.
- Convulsion.

2. Tidal volume (V_T)

The volume of air delivered to the patient during each breath (during inspiration or expiration).

It is usually set bet 6- 10 ml/ kg.

Large tidal volume my cause volutrauma and may cause damage the lungs.

3. Respiratory rate (frequency):

The number of breaths given by ventilator per minute which is 10- 16 breath/ m in adult.

4. Minute ventilation:

Volume of expired air in one minute.

Minute ventilation is equal to tidal volume multiply by respiratory rate: $V_E = VT \times R.R.$

5. Inspiratory to expiratory ratio (I: E ratio):

The ratio of inspiratory time to expiratory time during breath (usually its 1:2 in adult & 1: 1.5 in pediatric patient).

6. Peak airway pressure (Paw):

It is the highest airway pressure during inspiration we keep peak pressure below 30 cm H_2O. persistan peak airway pressure above 50 cm/H_2O increase the risk & barotrauma.

7. Trigger sensitivity:

Trigger sensitivity control the amount of patient effort needed to initiate a breath during inspiration

Usually set bet (-0.5) - (-2.0) cm H_2O.

Increasing the sensitivity decrease the work of patient to initiate or trigger a ventilation breath.

While decreasing the sensitivity (by increasing the negative pressure to -2) will increase the patient effort that needed to initiate inspiration.

8. Flow rate:

The speed of air delivering by the ventilator per unit of time (liter/ minute).

The higher the flow rate, the shorter the inspiration, and the faster peak airway pressure is reached.

9. Sigh:

It is a deep inspiration.

Sigh is a breath that has greater volume than tidal volume, usually 1.5-2 times the tidal volume, Sigh rate is 4-8 times/ hour.

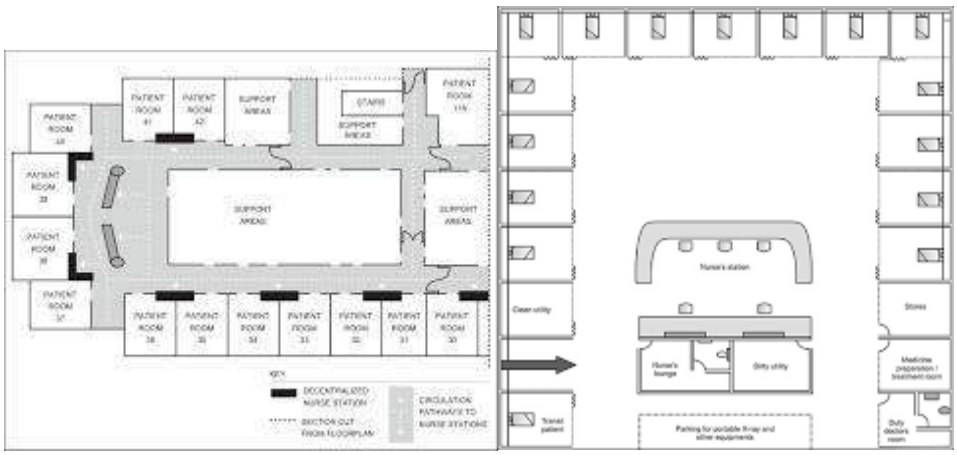
محاضرة ٢ عناية عملي

د. علاء غانم الكواز

Structure & designing of I.C.U

Design of ICU:

1. Designs should produce improvement of physical and psychological state of the patient and staff.
2. The design should allow all patients be easily observed, It can be with linear or circular placement of beds and central nursing station.



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3. It should contain open bed area in addition to isolated rooms for infections cases.

4. It should meet the need of the hospital, sometimes one ICU in not enough and we should prepare 2 or more ICU in the same hospital.
5. In general, ICU beds is 2-10% of total hospital beds.
6. The no. of beds should not less than 4 & not more than 20.
7. The optimal no. is 14, if more than 14 is needed, then 2 ICUs can be opened.

Structure of ICU:

1. More than one source of oxygen supply (both central and cylinder) is needed for each bed, 4 oxygen inlet, 3 air outlets, and 3 suction inlets.
2. More than one source of power supply (at last 16-20 power points near every bed).
3. 4 data outlet.
4. Adequate artificial light
5. Large windows with good access of sun light.

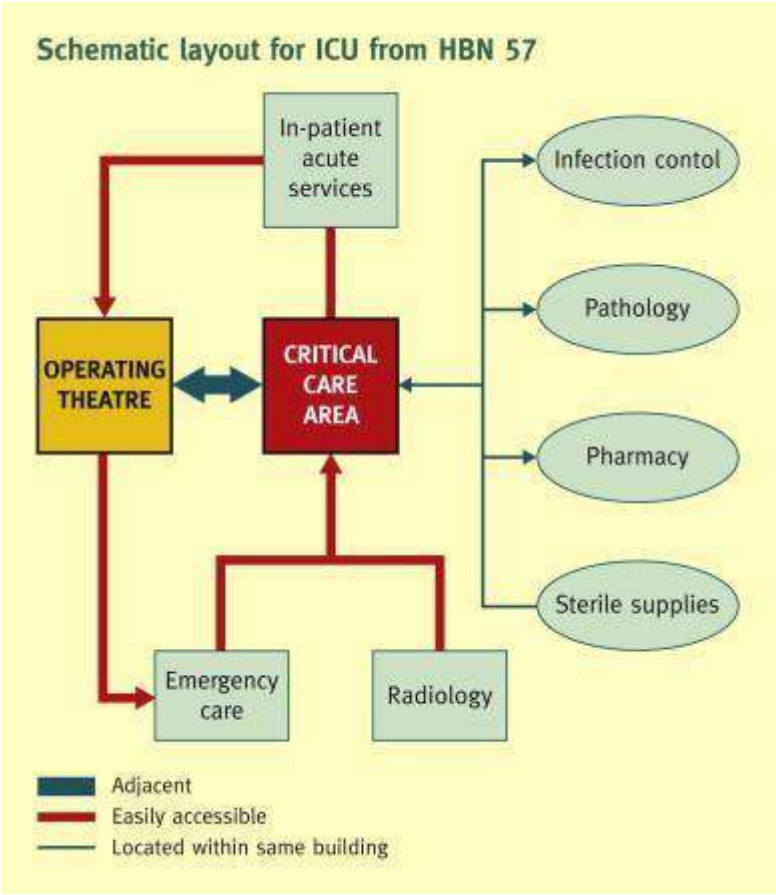
Size of ICU:

The size is depend on the type of services provided.

1. In general hospital 2% of hospital beds.
2. In sub-spaciality hospital 10% of total hospital beds.

Location of ICU:

1. Should be centrally located with easy access to operating theater, emergency, laboratory and radiology and other departments.
2. Easily to be reached.
3. Away from main hospital traffic.
4. Restricted area & restricted entry.



Entrance to ICU

1. Board corridor allow easy transport of beds and equipments.
2. Double door entrance, over size elevator and doors.
3. Changing room for shoes, Gowns & masks.

4. Separated toilet & kitchen facilities.

5. Areas for supportive and storage services.

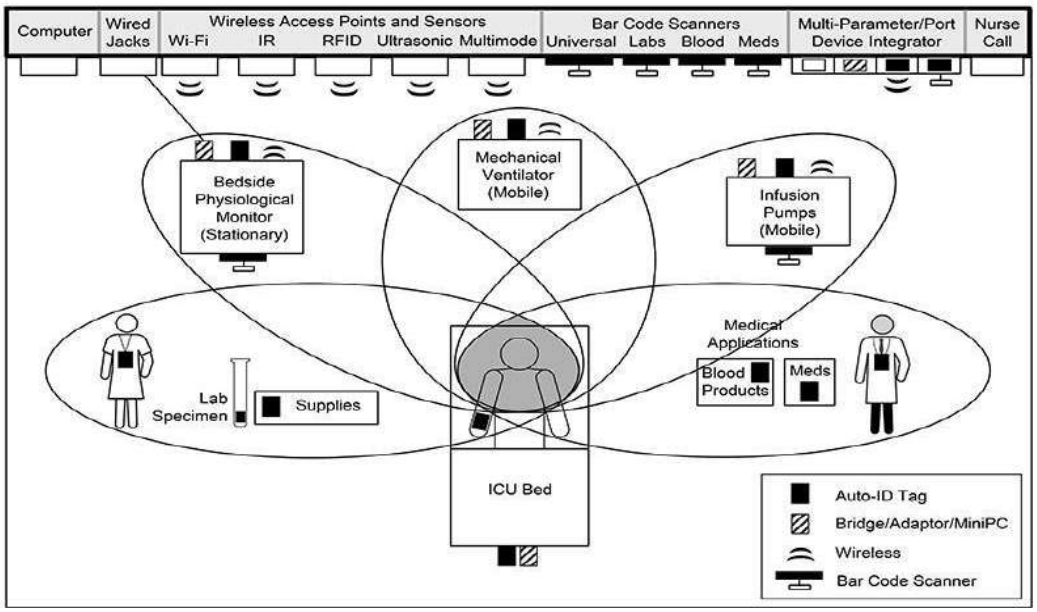
6. Emergency Exit

Bed space

Sufficient space is required around the beds for free movement of ventilator, monitors & other equipments.

1. At least 20 m^2 for each bed space
2. Total patient care area is 2.5-3 times of bed space area
3. Minimum of 1.2 m head- wall space
4. Minimum of 1.8 m leg space
5. Space between two bed is 2 m
6. In circular design minimum of 3.5 m space between center & beds.
7. The isolated room is minimum of 25 m^2 space to accommodate patient and staff and equipments with at least 2.5m traffic area beyond the bed.

Figure 3.


Computer
Wired Jacks
Wireless Access Points and Sensors
Wi-Fi
IR
RFID
Ultrasonic
Multimode
Bar Code Scanners
Universal
Labs
Blood
Meds
Multi-Parameter/Port Device Integrator
Nurse Call
Bedside Physiological Monitor (Stationary)
Mechanical Ventilator (Mobile)
Infusion Pumps (Mobile)
Medical Applications
Blood Products
Meds
Lab Specimen
Supplies
ICU Bed
Auto-ID Tag
Bridge/Adaptor/MiniPC
Wireless
Bar Code Scanner

Supportive and storage space

1. Storage area is at least 10m^2 per bed.
2. They should be away from patient area but not more than 30m , it contains intravenous fluids, giving sets, sheets and dressing, gowns.. etc.
3. Supportive spaces of dirty (25m^2) and clean (15m^2) rooms are necessary.
4. Doctor on call rooms (15m^2) with wash room and other room with food and drinks facilities.

ICU environment

1. Large windows with plenty of sunlight.

Patient exposing to sun light have been shown to decrease stress and improve sleep quality.

lack of natural light and outside view increase incidence of disorientation.

clock and calendar on each bed space is useful for patients orientation.

2. Air conditioning with temperature maintained at 25-27 ℃ and humidity of 40- 50%.
3. Positive pressure air flow from patient to outside.
4. Noise free, with noise absorbable materials, silence or soft music, and light colors.

Nurse station

1. Should be central and allow all patients to be observed.
2. Central monitoring system.
3. Complete visibility of all patients.
4. Special places for patients files & records and reference books.
5. Pharmacy and refrigerator for emergency and essential drugs
6. Easy communication, Telephone, intercom system and computers with internet connection.

Staffing

1. Doctors: anesthetist, physician, and sometimes surgeon.
2. Nursing staff: 1:1 or 1:2 patient ratio
3. others:

- Radiographer
- Physiotherapist
- Pharmacist
- Dietitians
- Psychotherapist
- Laboratory staff if available.

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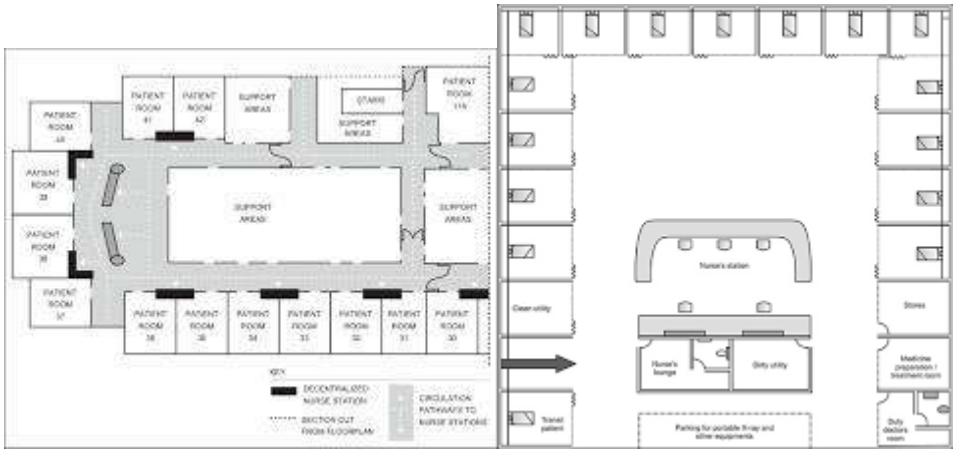

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1. More than one source of oxygen supply (both central and cylinder) is needed for each bed, 4 oxygen inlet, 3 air outlets, and 3 suction inlets.
2. More than one source of power supply (at last 16-20 power points near every bed).
3. 4 data outlet.
4. Adequate artificial light
5. Large windows with good access of sun light.

Size of ICU:

The size is depend on the type of services provided.

1. In general hospital 2% of hospital beds.
2. In sub-spaciality hospital 10% of total hospital beds.

Location of ICU:

1. Should be centrally located with easy access to operating theater, emergency, laboratory and radiology and other departments.
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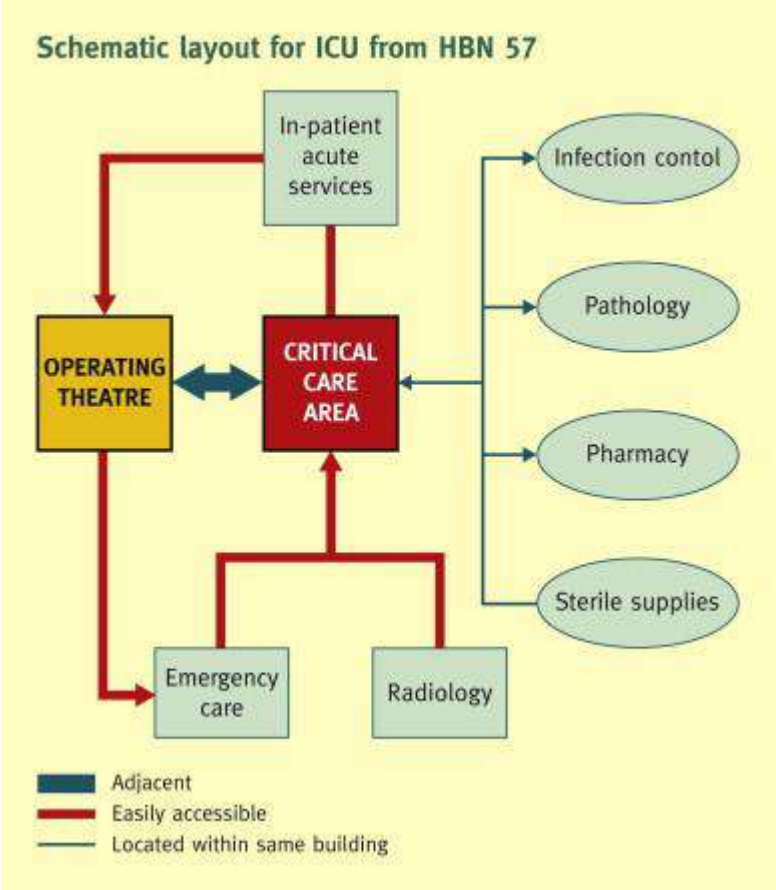


Entrance to ICU

1. Board corridor allow easy transport of beds and equipments.
2. Double door entrance, over size elevator and doors.
3. Changing room for shoes, Gowns & masks.

4. Separated toilet & kitchen facilities.

5. Areas for supportive and storage services.

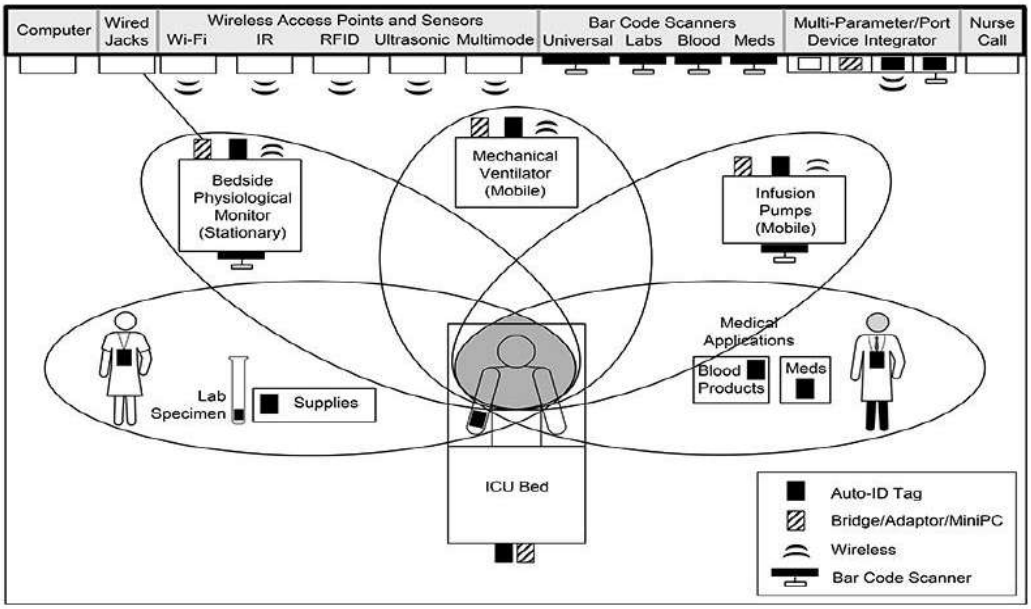
6. Emergency Exit

Bed space

Sufficient space is required around the beds for free movement of ventilator, monitors & other equipments.

1. At least 20 m^2 for each bed space
2. Total patient care area is 2.5-3 times of bed space area
3. Minimum of 1.2 m head- wall space
4. Minimum of 1.8 m leg space
5. Space between two bed is 2 m
6. In circular design minimum of 3.5 m space between center & beds.
7. The isolated room is minimum of 25 m^2 space to accommodate patient and staff and equipments with at least 2.5m traffic area beyond the bed.

Figure 3.


Computer
Wired Jacks
Wireless Access Points and Sensors
Wi-Fi
IR
RFID
Ultrasonic
Multimode
Bar Code Scanners
Universal
Labs
Blood
Meds
Multi-Parameter/Port Device Integrator
Nurse Call
Bedside Physiological Monitor (Stationary)
Mechanical Ventilator (Mobile)
Infusion Pumps (Mobile)
Medical Applications
Blood Products
Meds
Lab Specimen
Supplies
ICU Bed
Auto-ID Tag
Bridge/Adaptor/MiniPC
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Supportive and storage space

1. Storage area is at least $10m^2$ per bed.
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3. Supportive spaces of dirty ($25m^2$) and clean ($15m^2$) rooms are necessary.
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ICU environment

1. Large windows with plenty of sunlight.

Patient exposing to sun light have been shown to decrease stress and improve sleep quality.

lack of natural light and outside view increase incidence of disorientation.

clock and calendar on each bed space is useful for patients orientation.

2. Air conditioning with temperature maintained at 25-27 ℃ and humidity of 40- 50%.
3. Positive pressure air flow from patient to outside.
4. Noise free, with noise absorbable materials, silence or soft music, and light colors.

Nurse station

1. Should be central and allow all patients to be observed.
2. Central monitoring system.
3. Complete visibility of all patients.
4. Special places for patients files & records and reference books.
5. Pharmacy and refrigerator for emergency and essential drugs
6. Easy communication, Telephone, intercom system and computers with internet connection.

Staffing

1. Doctors: anesthetist, physician, and sometimes surgeon.
2. Nursing staff: 1:1 or 1:2 patient ratio
3. others:

- Radiographer
- Physiotherapist
- Pharmacist
- Dietitians
- Psychotherapist
- Laboratory staff if available.

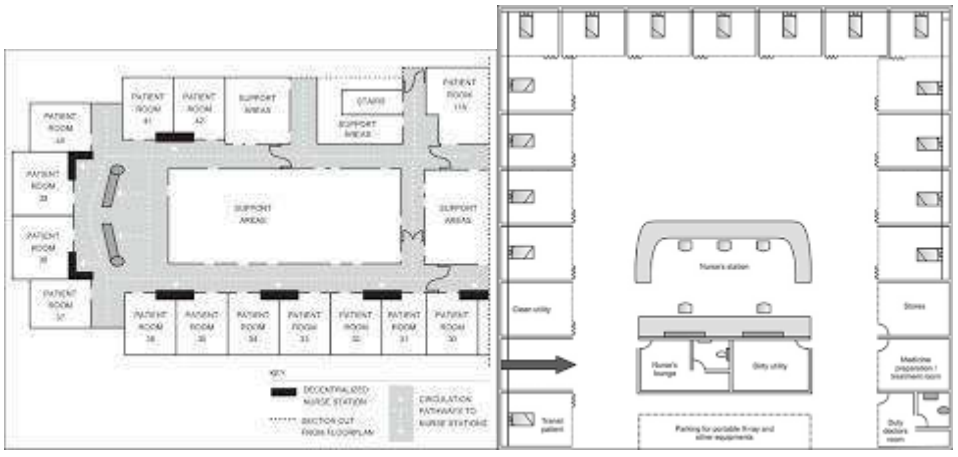
محاضرة(٢) عناية نظري

د.علاء الكواز

Structure & designing of I.C.U

Design of ICU:

1. Designs should produce improvement of physical and psychological state of the patient and staff.
2. The design should allow all patients be easily observed, It can be with linear or circular placement of beds and central nursing station.



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3. It should contain open bed area in addition to isolated rooms for infections cases.

4. It should meet the need of the hospital, sometimes one ICU in not enough and we should prepare 2 or more ICU in the same hospital.
5. In general, ICU beds is 2-10% of total hospital beds.
6. The no. of beds should not less than 4 & not more than 20.
7. The optimal no. is 14, if more than 14 is needed, then 2 ICUs can be opened.

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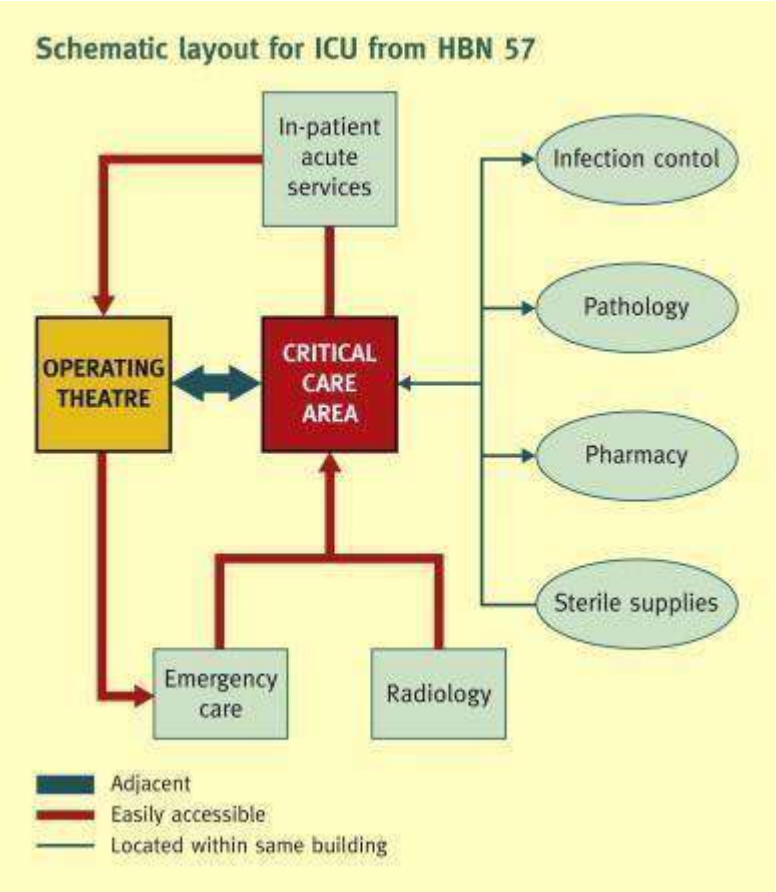

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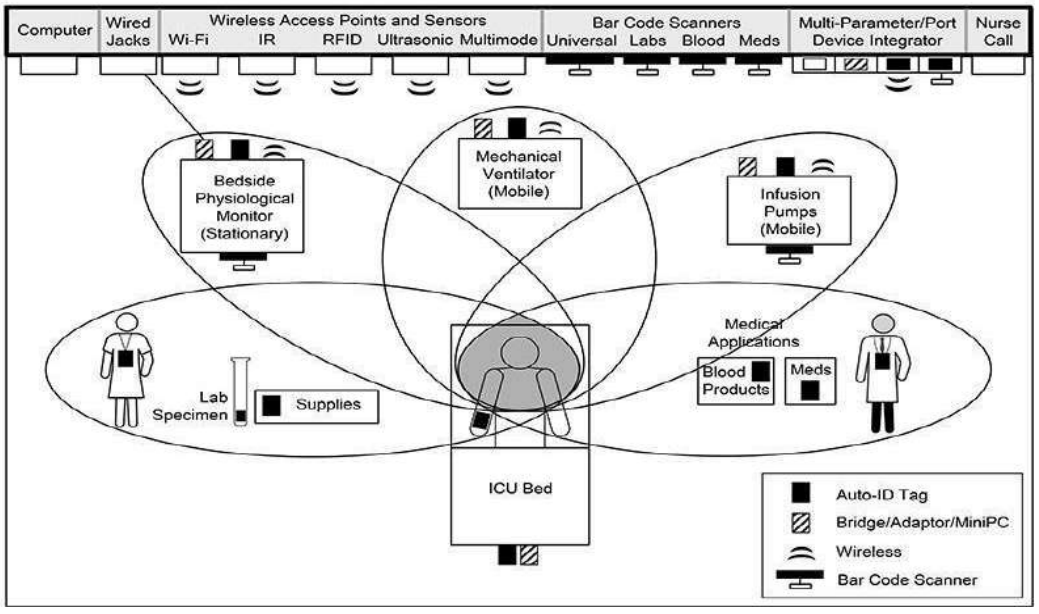

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3. Others:

- Radiographer
- Physiotherapist
- Pharmacist
- Dietitians
- Psychotherapist
- Laboratory staff if available.

محاضرة (٢) عناية مركزة

د.علاء غانم الكواز

Types of ICU

1. **General types**

a. Medical intensive care unit (MICU)

b. Surgical intensive care unit (SICU)

c. Main or mother intensive care unit (Main.I.C.U) which is combined medical & surgical & Trauma ICU.

2. **Specialized type**

a. Post-anesthetic care unit (PACU) usually inside operating theater, patient can be held for 6 hr or more.

b. Neonatal intensive care unit (N.I.C.U)

c. Pediatric intensive care unit (P. I. C. U)

d. Coronary care unit (CCU)

e. Respiratory care unit (RCU)

f. Neurosurgery intensive care unit (NSICU)

g. Trauma intensive care unit (T. I. C. U)

h. Burn intensive care unit (B. I. C. U)

Post anesthetic care unit :

It is an example of specialized types of ICU , also called post anesthetic recovery unit or recovery room

Definition: It is a special unit located close to operating theatre, provides monitoring, close observation and care to patient after anesthesia and surgery. It should have 1.5- 2 beds per operating room used beds ,and staffed with 1:2 nurse to patient ratio for stable patients, 1:1 ratio for unstable patients.

Usually patient stay for 1 hr and it can be hold for 6hr or more according to patient condition

.

PACU requirements includes:

1. Contains monitoring equipments which include ECG, oximeter, capnograph, BP monitoring & temperature.
2. Ventilation equipments which include oxygen source, face masks, Ambu bag endotracheal tubes, suction device and ventilator .
3. Portable defibrillator.
4. Intravenous fluids: crystalloid and colloid with I.V cannula and giving sets.

5.Essential and emergency drugs.

Aim of PACU

1. Full recovery with full return of consciousness.
2. Return of muscle power eg. protruding of tongue, grasping of hand, elevation of head for 5 seconds.
3. Return of protective airway reflexes.
4. Respiratory system stability: normal respiration with normal color.
5. CVS stability- normal BP, HR and rythm.
6. Normal body temperature; no hyperthermia or hypothermia.
7. Control of nausea & vomiting.
8. Control of bleeding.

9.Control of pain.

Rules for PACU

1. Patient should transported from operation room to PACU by member of anesthesia team who know about patient operation and condition.
2. Patient should be received by anesthetic member in PACU with verbal report about his condition.
3. Patient should be evaluated continuously by the nurse.
4. The decision of discharge from PACU is the responsibility of anesthesia doctor.

Criteria of discharge from PACU

1. Consciousness level: Patient fully awake and should respond to verbal stimulation, oriented and be able to answer question appropriately.
2. Normal vital signs: with CVS and respiratory system stability.
3. Good muscle power and normal reflexes.
4. Normal fluid balance and body temperature.
5. Pain control, with no nausea or vomiting.
6. No bleeding at wound site (dressing and drains).
7. For spinal and epidural anesthesia, feeling his legs with ability to move the knee joint.

Classification of critical care units

1. **Level (1):**

- Usually in small hospital.
- Provides observation, monitoring and short term ventilation.
- The medical director should be a certified intensive care specialist.
- The nurse- patient ration is 1:3

2. **Level (2):**

- Located in large hospitals.
- Should capable of providing a high standard of general intensive care , provides observation, monitoring and long term ventilation.
- The medical director and at least one other specialists should be certified intensive care specialist.
- Nurse- patient ratio is 1:2, and medical staff are available in the unit all the time.

3. **Level (3)**

- Located in major referral hospital.
- It should provide all aspect of intensive care management for indefinite period.
- Should be staffed by intensive care specialist, critical care nurses and all health professionals.
- Complex investigation & imaging is available at all times.
- Nurse-patient ratio is 1:1 &should be present all the times.

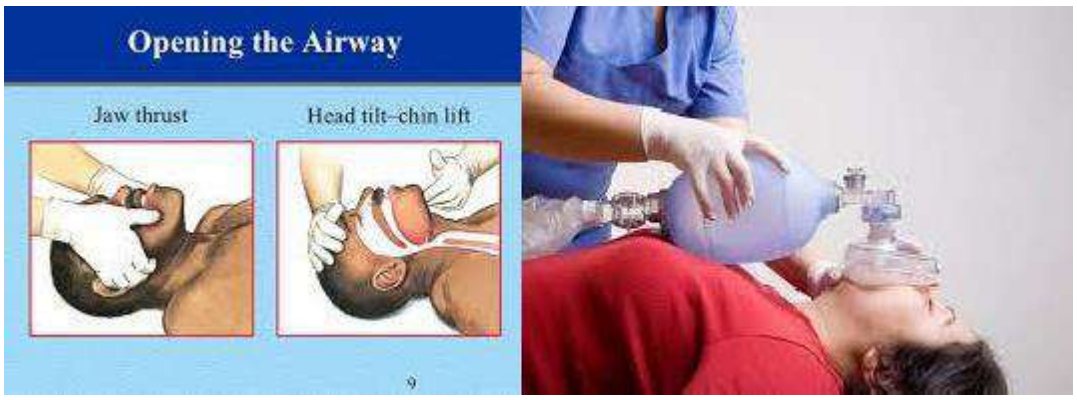
محاضرة٣ عناية عملي

د. علاء غانم الكواز

Clinical management in I. C. U

Early measures for **clinical management** of emergency cases admitted to ICU includes:

1. Uncover the patient, remove clothes and search for any hidden injures .
2. Quick review of patient with physical examination.
3. Maintain ABC(Airway Breathing Circulation), ensure clear airway, and the patient breath normally and chick for bleeding.

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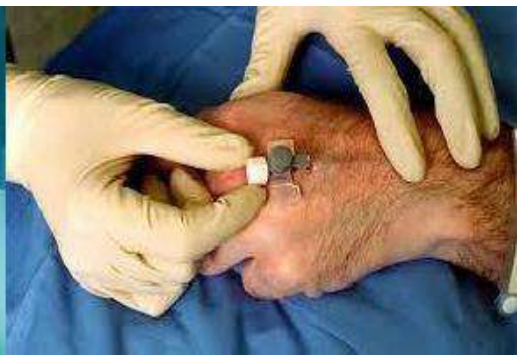
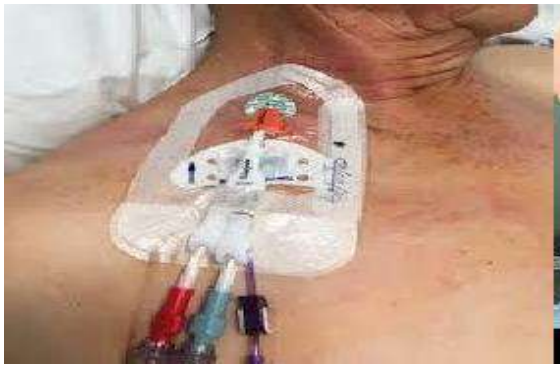




5. Take blood sample & send for investigations: blood group and cross matching, serum electrolytes, blood urea and serum creatinine, blood sugar …etc.
6. Attach bed side monitor for SPO_2 Blood pressure, heart rate & rhythm …etc.

7. Give medication: antibiotics & analgesic drugs if needed
8. Maintain fluid balance.

9. Check for urine output and temperature.

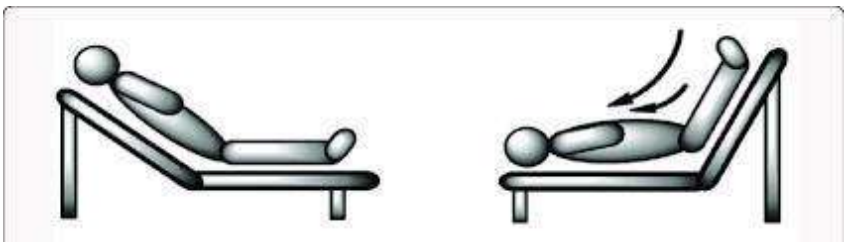
10. Documentation: record everything with timing.

Subsequent (delayed) clinical management of patient in ICU includes:

1. Head position.
2. Analgesia
3. Sedation
4. Glucose monitoring
5. DVT prophylaxes
6. Infection control
7. Peptic ulcer prophylaxes.
8. Bedsore
9. Maintenance of fluid and nutrition
10. Inotropic drugs .

1. Head position

Head up elevation by 30 degree for head injury patients ,and supine position with elevation of the legs for shocked patients.



2. Analgesia:

- Acetaminophen (paracetamol) or NSAID used for mild to moderate pain & particularly effective in muscular and skeletal pain
- Morphine or fentanyl which used for sever pain.

3. Sedation: Benzodiazepines

Diazepam: long acting sedative particularly useful in status epileptics .

Midazolam: short acting can be given as infusion in intubated patients.

4.Glucose monitoring

Checking for signs & symptoms of hypoglycemia as anxiety, pallor, palpitation, sweating, irritability with sending for random blood sugar and treatment with hypertonic glucose fluid (GW 10%) if needed.

If there is hyperglycemia treatment with insulin accordingly, hyperglycemia is particularly important in head injury because it is worsening the outcome & prognosis of primary injury.

5.DVT prophylaxis;

- Intermittent pneumatic compression devices and elastic compression stocking..
- Using of prophylactic heparin or low molecular weight heparin.

6.Infection control :

- Frequent hand washing
- Use of disinfectants
- Maintain aseptic technique for any procedure
- Superficial & deep cleaning & wash of the ward with regular culture for infection.
- Using of antibiotic drugs**.**

7. Peptic ulcer prophylaxis: H_2-blockers as Ranitidine &cimetidine, especially important in severely injured patients.

8. Bed sore:

- Disposition of patient with 2 hourly changing in position**.**
- Using of air mattresses.
- Cleaning & dressing of bed sore with each shift.
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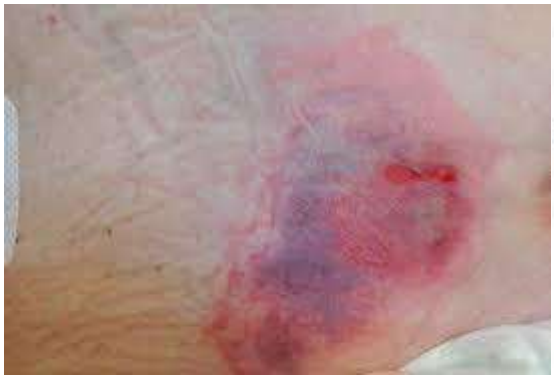

9. fluid & nutrition :

- Maintenance of fluid balance, calculating requirements with input and output, NS. 0.9%, RL and DW 5% fluids can be given.
- Oral feeding with normal diet & fluid intake.
- Enteral feeding: through nasogastric tube with high protein diet, milk & egg… ext.

- Parenteral feeding: though central line: eg Amino acids and lipid emulsion solutions.

10. inotropic drugs

To maintain normal BP. in hypotensive patient, eg. septic shock and Heart failure .

- Dopamine, Dobutamine .
- Noradrenalin, Ephedrine and phenylephrine.

محاضرة (٣) عناية عملي

د. علاء غانم الكواز

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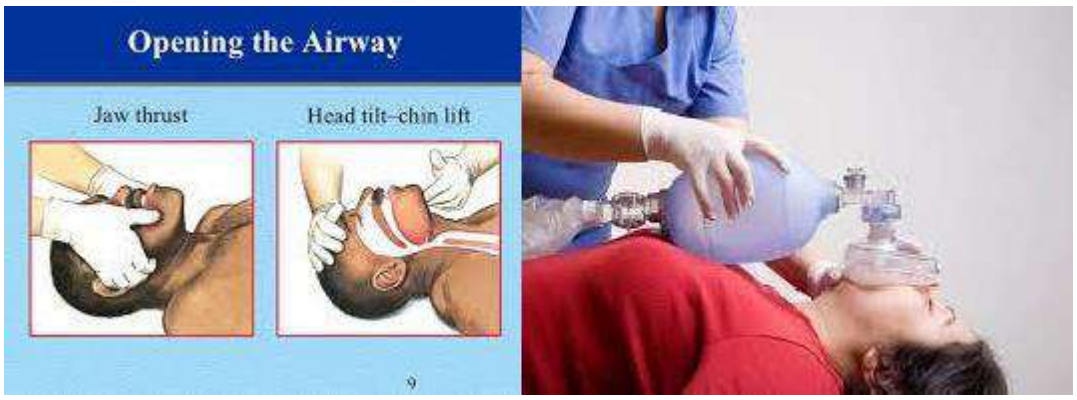


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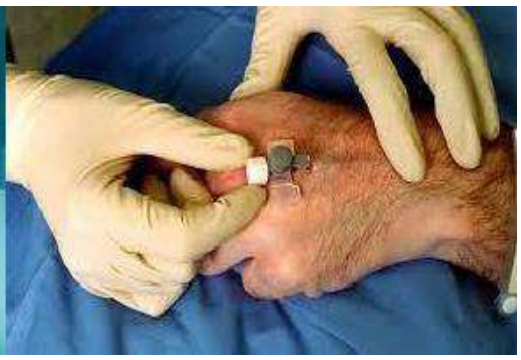
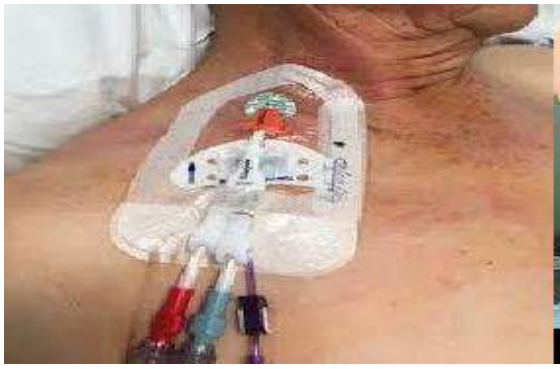


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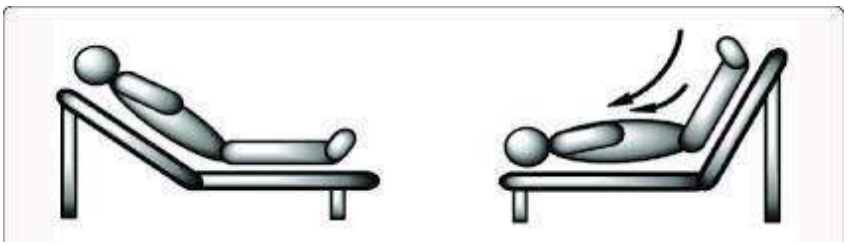

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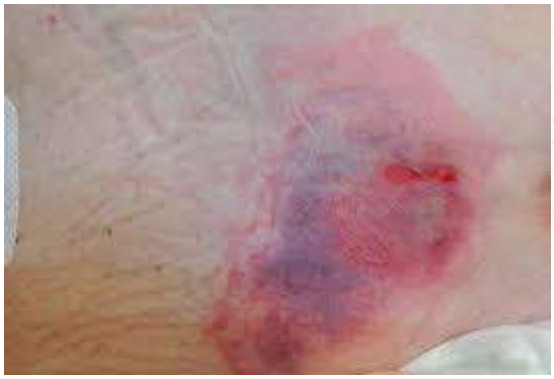

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