

EMERGENCY MEDICINE

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first five seminars + Al-bashir's hospital notes

FIRST SEMINAR

When we are dealing with any case in the ER, we start with ABC management and general assessment from the moment the patient steps in, including the gate and general appearance. Using ABC, once you insure the patient's airway, you move forward and check breathing. But before you move to the next step you need to recheck airway and breathing then start with circulation. Our aim here is to stabilise the patient.

- Airway problems include anything that prevents a patent airway, like any obstruction causes (angioedema, hematoma, foreign body aspiration etc)
- Breathing problems include anything preventing adequate oxygenation, like (pulmonary edema, tension pneumothorax, asthma exacerbation and bronchospasm)
- Circulatory problems include any type of shock (hypovolemic, cardiogenic..etc) as well as sepsis and bleeding whether it was internal or external.

☆ Door to ECG is 10 minutes.

☆ Patient's SPO2 needs to be >94%. If lower, give oxygen except to patient's with chronic hypercapnia (respiratory failure type 2 + some COPD patients)

While listing differential diagnosis for the patient's condition, we always keep the most critical diagnosis on top of our list and we do the needed to either diagnose it or rule it out (even if it was rare. we prioritise depending on how critical and life-threatening rather than whatever is common)

Systemic review when taking history in the ER involves taking a brief history of systems related to the list of differentials we established. We don't ask about all of them here unlike taking history in clinics.

- for example, a patient came to the ER with chest pain. We need to rule out acute coronary syndrome first because it is the most critical, then pneumothorax and so on.

ACUTE CORONARY SYNDROME

Anginal chest pain is retrosternal, radiating to the jaw/arm. Exacerbated by exertion and relieved by rest. If not relieved by rest or nitroglycerin then it could be an MI due to angina.

Atypical ACS occurs in three categories of patients (women, diabetic patients and the elderly).
Their most common symptom is acute onset of dyspnea.

In emergency cases if we suspect murmurs we need to take the patient to a quiet room, if not possible then we need to perform bed-side echocardiogram. Murmurs are musical audible sounds on auscultation, where the heart rate is abnormally heard.

☆ STEMI ; we need to have ST elevation in at least two contiguous leads on the 12 lead ECG. (leads that assess the same wall)
- example: ST elevation seen in V1 and V2 is considered STEMI. but ST elevation in V2 and V3 is not indicative of STEMI.

☆ for STEMI, 1 mm (1 mm) is enough for limb leads. 2 mm (2 mm) is enough for chest leads.
- we need only 1 mm for limb leads but more in chest leads because limb leads are further than chest leads to the heart so they cover a wider area of the heart, so a wider area involved is needed to elevate the ST in these leads.

☆ In NSTEMI, patients could present with normal ECG, inverted T wave or even ST depression. That's why we can NOT only rely on ECG for diagnosis. So if we suspect NSTEMI then we need to perform cardiac enzymes test and admit the patient putting him/her on a continuous cardiac monitoring (3 leads maximum).

ACUTE CORONARY SYNDROME

- ☆ IF any patient experienced a second attack of chest pain we need to do a 12 leads ECG again.
- ☆ Most sensitive cardiac enzyme is troponin I. However, chronic kidney disease can lead to elevation in troponin levels (mainly CHF)
- ☆ We could use CPK- MB enzyme, however it is not specific. It can be elevated in any heart damage, or in cases of renal failure. CPK in general could be elevated in muscle disorders, like rhabdomyolysis or even IM injections.

In any case of ACS, we need to return some kind of perfusion to prevent ischemia and necrosis. We also need to give aspirin (75 mg is enough as anti-platelets dose; > 300 mg dose is anti-inflammatory)

- ☆ If a patient has an MI and is hypotensive, do NOT give morphine and nitroglycerin as they can exacerbate hypotension.
- ☆ When managing patients with STEMI, the first thing we need to do always is to restore perfusion to prevent further damage and necrosis. If 90 mins have already passed, give clopidogrel (plavix) and aspirin.
 - if CATH isn't available/ possible then also give medications.

MI is better handled by the elderly as they have collaterals that will decrease their mortality rates unlike the young.

AORTIC DISSECTION

Typical presentation is someone coming to the ER complaining of sudden severe chest pain radiating to the back following heavy weight lifting.

☆ Other symptoms depend on the site of the deficit, if the ascending part is involved then we get stroke-like symptoms and syncopes. If the arch is involved, we get a radio-radial delay associated with chest pain. If the descending part is involved, we get abdominal pain/ groin pain, as well as radio-femoral delay.

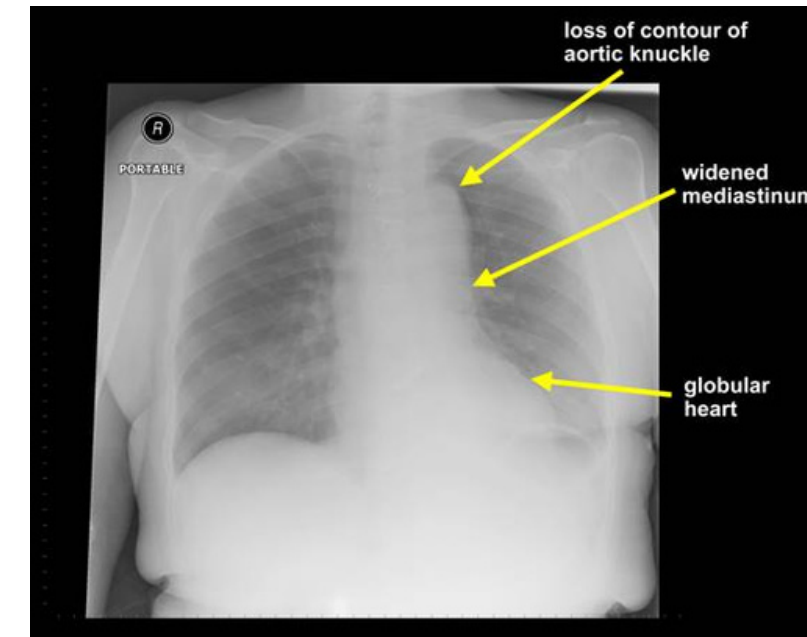
☆ stroke- mimics include hypoglycaemia, sepsis and aortic dissection involving the ascending aorta.

(This was just mentioned briefly, I added an explanation) One of the risk factors for aortic dissection is bicuspid aortic valve, which is a congenital anomaly where the aortic valve has two leaflets instead of three, which makes this valve ineffective at stopping blood from leaking back into the heart. This leakage is called aortic regurgitation. The aortic valve may also become stiff and not open up, which is called aortic stenosis, which causes the heart to pump harder than usual to get blood through the valve. The aorta may become enlarged with this condition and this increases the risk of aortic dissection.

AORTIC DISSECTION

X-ray findings that we can see in cases of AD are:

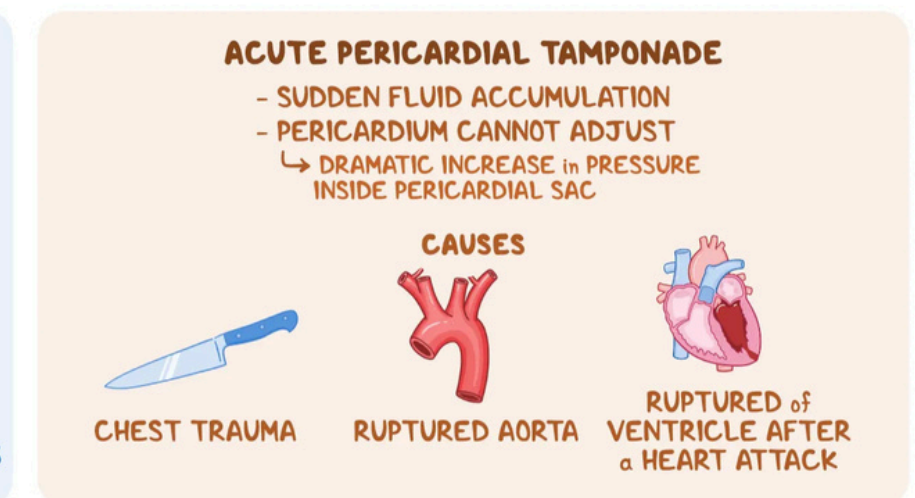
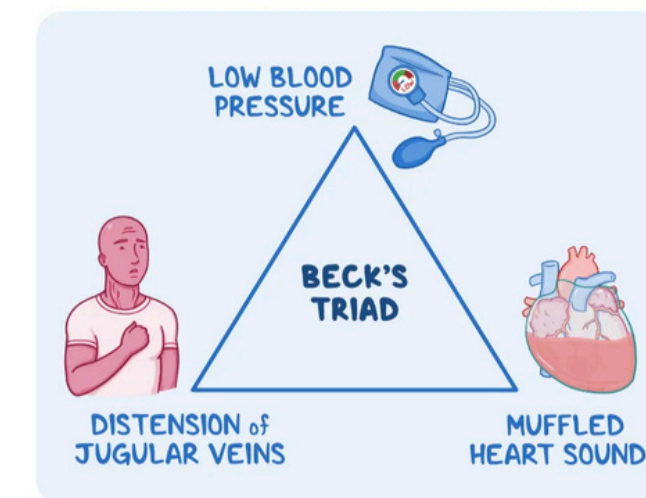
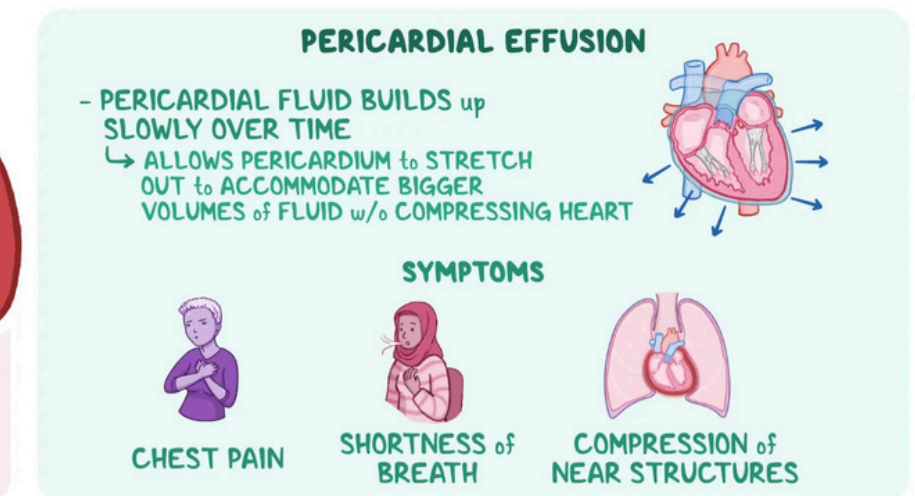
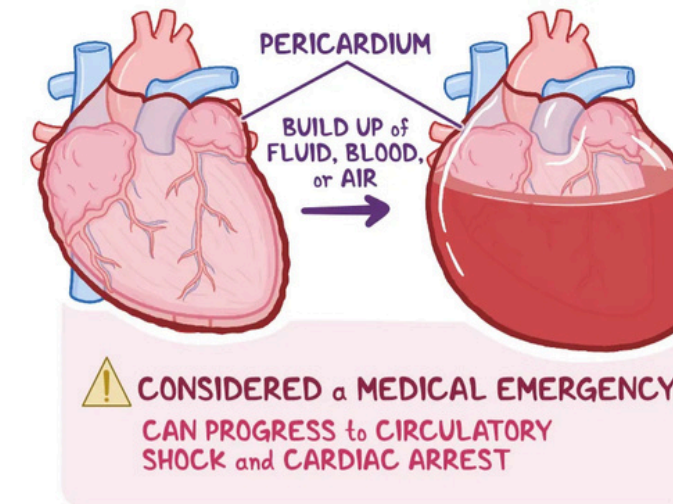
- loss of the heart knuckle (knob)
- Widening of the mediastinum
- Calcium sign
- Plural effusion and sometimes displacement of the trachea/esophagus



One of the most important complications of aortic dissection is cardiac tamponade.

Beck's triad is very important.

- Muffled heart sound occurs because of the weak sound transmission.
- Hypotension occurs due to the fluid restricting the heart expansion (no preload) which interferes with its contractility too (no afterload)
- JVD is due to the retention of the blood back to SVC and up the jugular vein as the heart is unable to pump the blood in its chambers.



PULMONARY EMBOLISM/ DVT

Risk factors are components of Virchow's triad (picture aside)

Mostly it involves lower limbs. It occurs in upper limbs mainly in cancer patients.

In lower limbs, we will find edema. There are two types of edema, pitting and nonpitting edema.

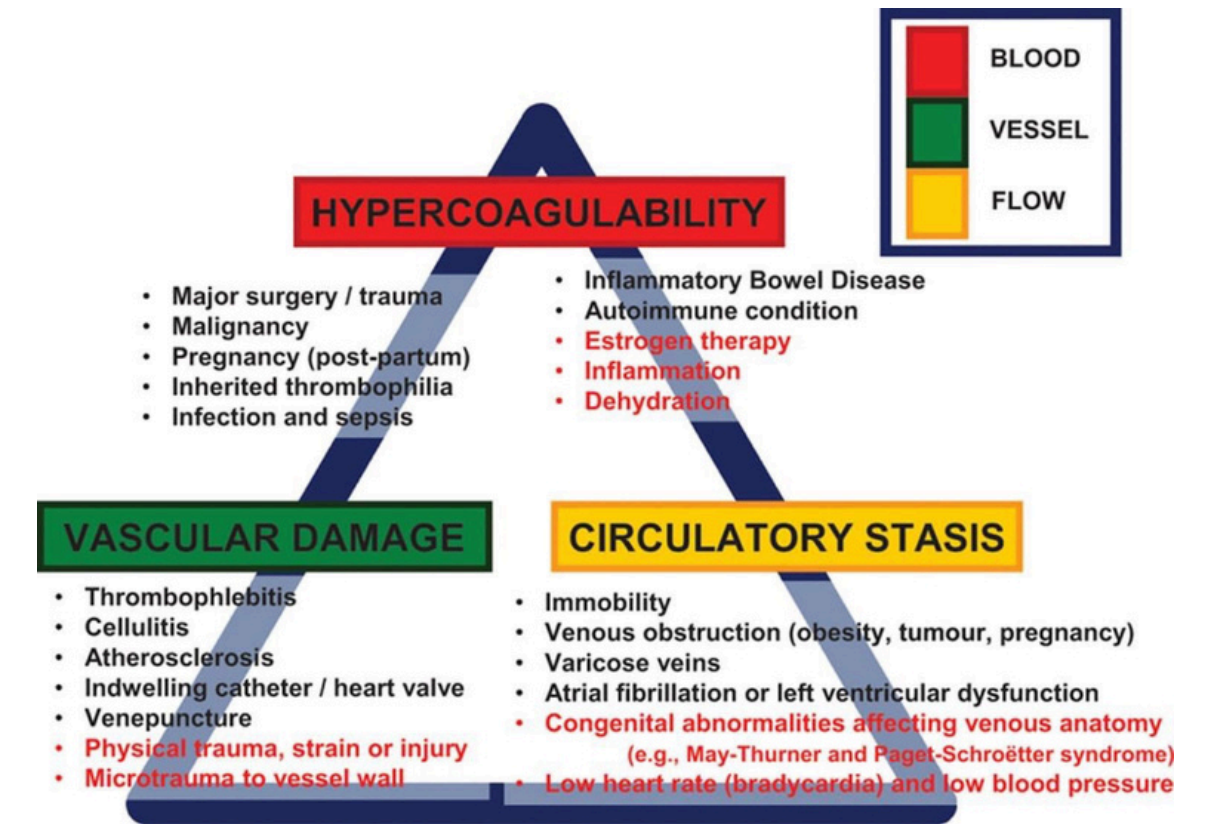
- most common cause of nonpitting edema is myxoedema (hypothyroidism) shin of tibia
- Most common cause of pitting edema is heart failure and fluid accumulation due to excess fluids.

D-dimer is used to determine whether there is a dvt/pe or not (indicative of a clot) however if it was positive then the diagnosis is not confirmed, we still need further testing. (it could be normally elevated in pregnant women and cancer patients.)

☆ Most common ECG finding in PE patients is sinus tachycardia (abnormal change) // normal change is finding the ECG normal. A rare but unique and classic pattern known as McGinn-White sign "S1Q3T3" can be seen. (a deep S wave in lead 1, deep Q wave in lead 3 and an inverted T wave in lead 3)

X- ray findings in PE patients include 7 signs, sometimes we find none so xray is not the best investigation to do. We need to examine popliteal as well as femoral pulses.

Treatment is to give two-drugs therapy (warfarin and heparin) they should be started together as warfarin has a slow onset of action.



PNEUMOTHORAX

It could be either spontaneous or due to a cause. Mostly spontaneous in thin tall men, wise to a rupture of a small emphysematous bullae.

Iatrogenic pneumothorax could be due to therapeutic/ diagnostic procedures like biopsy taking, central vein cannula introduction as well as aspiration.

Tension pneumothorax is either closed or open. Closed in blunt injuries, like due to a seatbelt in car accidents or airbag, and open in gunshot and stabbing wounds. Triad of tension pneumothorax includes: absent breath sounds, jugular vein distension and hypotension.

Physical examination and related findings are very important to diagnose tension pneumothorax. We could find tracheal deviation, hyperresonance on percussion as well as absent/reduced breath sounds on one side.

SECOND SEMINAR

pulmonary edema

In x-ray images, we can spot pulmonary edema as we can highlight fluid accumulation there. We need to see the obliterated costophrenic angle, if both angles are obliterated then we say that bilateral plural effusion then identify on which side it is more prominent.

Anything that causes dilation of the atrium, or increased volume overload will result in an increase of B-type natriuretic peptide which mainly enhances sodium excretion. (Water follows sodium ions which depletes fluid volume.

Cardiac wheeze can be seen in patients with left sided heart failure. This sound occurs during expiration and it blunts other sounds. It occurs as the patient uses a lot of power to breathe.

Tripod position is seen in asthmatic exacerbation , COPD patients, patients with HF (pulmonary edema) and epiglottitis.

☆ Morphine causes reduction in preload and afterload + causes vasodilation + has an anxiolytic effect.
Nitroglycerin decreases both afterload and preload too.

☆ If we suspect right ventricular MI (inferior MI) preferably do NOT give morphine and nitroglycerin as it can exacerbate hypotension.

☆ Treatment of patients with decompensated HF treatment is giving LASIX IV as well as nitroglycerin IV. As infusion (slowly) to reduce the risk of life-threatening hypotension.

☆ In cardiogenic shock, give vasopressors as they have a positive inotropic effect. As well as anti-platelets and dobutamine injection.

SECOND SEMINAR

Asthma/ COPD

Shortness of breath has many etiologies, like asthma and COPD which are different from each other by their nature and pathophysiology.

Asthma is a chronic inflammatory disease - type1 hypersensitivity reaction - which's exacerbated at night/early morning due to elevated cortisol levels and inflammatory mediators during these hours. Plus the weather is colder which provokes bronchospasm. What triggers it often is exposure to the allergen. Asthmatic patients often have high blood PH or normal, and low CO_2 in cases of hyperventilation. In severe asthmatic attack with bronchial obstruction, if CO_2 level is normal then the patient is having an imminent respiratory failure. If CO_2 level > 45 then the patient is entering a respiratory arrest. In asthmatic patients, we measure FEV1 to identify the severity of asthma.

- status asthmatics then give ketamine.
- Silent chest is an indication of a life-threatening asthmatic attack.
- - in severe recurrent attacks episodes of asthma (multiple previous admissions to the ICU) ,, give biological agents against IL-5. (Reslizumab/benralizumab)

☆ Reason why we don't give magnesium sulfate to patients with imminent/ respiratory failure is that IV magnesium needs 10 mins to result in bronchodilation, thus delaying intubation which isn't ideal in life-threatening conditions.

- Moderate asthma, give an inhaler with a spacer, let the patient breathe through it for 10-15mins. Or a nebuliser (up to 4) and it's better than the inhaler.
- if it is safe for the patient to take oral medications, give. If not then only treat with IV agents.

☆ ALWAYS keep an eye on asthmatic patients for any signs of deterioration. If observed then take needed steps to stabilise the patient.

SECOND SEMINAR

Asthma/ COPD

Differences between asthma and COPD :

B2 agonist is the first line to treat asthma whereas the first line for treating COPD is inhaled corticosteroids. They are also different in that asthma exacerbation occurs after exercising when the bronchodilation effect produced by sympathetic system activation wears off. While COPD patients have exacerbations during exercise.

COPD:

☆ Normally, what drives our ventilation is CO₂ concentrations. But in COPD patients, they are adapted to high levels of CO₂ so their main ventilation driver is O₂. Elevating spo₂ in COPD patients higher than 88%-92% results in respiratory centre depression which causes the patient to fall into CO₂ narcosis, this leads to decreased level of consciousness and sleepiness due to acute hypercapnia on top of chronic.

- COPD patients often have polycythemia (the affinity of haemoglobin to CO is higher than it is to O₂) . It results in ineffective RBCs and thus false anemia. This enhances erythropoietin and the production of more RBCs.

- Most common cause for COPD patients to come to the ER is exacerbations caused by respiratory infections. (Give corticosteroids, prednisolone orally 30-60mg)

SECOND SEMINAR

Asthma/ COPD

☆ We have two types of respiratory failure, type one is associated with hypoxemia without hypercapnia. Type two is associated with hypercapnia.

Treatment is different, we can use C-pap to treat heart failure and type 1 respiratory failure, as well as in patients with obstructive sleep apnea. It works by providing continuous constant pressure of oxygen that the patient breathes spontaneously. Bi-pap is used to treat patients with hypercapnia like patients with type 2 respiratory failure. It works typically by “breathing instead of the patient”, by controlling the rate of respiration. It provides two different pressures, a high pressure to assist inspiration, and a low pressure to keep alveoli open.

☆ Arterial blood gases monitoring is very important in these situations. (COPD and type 2 respiratory failure).

☆ We can perform x-ray to rule out complications.

THIRD SEMINAR

Trauma is basically any type of shock. It needs to be dealt with either with BLS or ACLS.

ACLS requires a professional person to do. It's different than BLS in that it involves advanced airway management (intubation) and the use of medications.

Usually, we perform BLS until we reach the hospital then the staff can start ACLS.

☆ 5H and 5T are for the reversible causes of a cardiac arrest:

- 5Hs (hypo/hyperkalemia, heart disease/hypoxia, H ions/ acidosis, hypothermia, hypovolemia)
- 5Ts (tension pneumothorax, thrombosis-PE/MI, toxins, cardiac tamponade, trauma)

When assessing the patient's airway, we use look listen and feel technique.

- look for chest movement.
- listen for respiratory sounds.
- feel the pulse (carotid) // breath (against your cheek)

☆ Start with chest compressions. One cycle contains (30compressions and 2breaths)

☆ do NOT perform head tilt chin lift in patients with suspected cervical spine injury.

We don't perform jaw thrust in all patients because it's a very painful stimuli.

- While performing chest compression, make sure the palm of the hand is placed on the sternum (of the nondominant hand) and place the dominant hand above. We work 5 cycles in 2 minutes.

☆ Effective CPR — push HARD (2-2.5 inches/ 5-7cm) = 2/3 of the chest wall,,

FAST (100-120 compressions per minute) and allow chest RECOIL.

Chest recoil is important to insure the refilling of the ventricles to maintain a cardiac output.

- this isn't done in patients with ventricular fibrillation (use EDD)

THIRD SEMINAR

☆ in paediatrics, the most common cause of a cardiac arrest in infants is hypoxia. Each cycle in paediatrics' CPR contains 15/2 then another 15/2. So 30 compression with two breaths after every 15 compression as they need more oxygen.

- Importance of recovery position is to prevent aspiration, prevent movement and further more trauma to the patient.

Defibrillator is used in ACLS instead of AED. We need to check the rhythm and decide whether we need to give a shock or not. We have shockable and non-shockable rhythms.

- shockable rhythms include ventricular tachycardia (bizarre) irregularly irregular + pulseless ventricular tachycardia (wide QRS) regular.
- Non-shockable rhythms include pulseless electrical activity and asystole (can't back up from it)

We start CPR until ECG monitor is attached, then we check the rhythm. The first cycle prior to monitor attachment and identification of the rhythm isn't considered the first cycle.

- If non-shockable, give epinephrine within the first cycle after you checked the rhythm and start counting. Give 1mg IV push.
- If shockable, start giving epinephrine after the second cycle of compressions. Give amiodarone IV only in the 3rd (300mg) and 5th cycle (150mg). In cases of amiodarone allergy or if it's not available, give lidocaine.

☆ do NOT give amiodarone if it is a non-shockable rhythm.

☆ IV epinephrine is given in shockable and non-shockable rhythms every other cycle. One cycle give, do not give in the second. And so on.

THIRD SEMINAR

If there is a reversible cause for this cardiac arrest, you need to fix it.

Recovery from a cardiac arrest is called ‘return’.

Keep performing CPR for up to 30 mins. After 30mins, permanent brain damage would have already occurred (could indicate brain death). So check pupil dilation. The least is to perform 8 cycles before stopping.

☆ in cases of hyperkalemia, the first thing we need to do is to give calcium gluconate/ calcium chloride (gluconate is better) as it stabilizes the cardiac membrane (decreasing its sensitivity to potassium ions).

- Then, we should give salbutamol (inhalational) because it is the fastest to reduce potassium level (takes around 20mins) For hyperkalemia, give 20 mg but if the patient is a known case of ischemic heart disease give 10mg. (Asthma and COPD are given 5mg for symptomatic management)
- Give dextrose along with insulin to result in shifting of potassium inside cells.

The most common cause of hyperkalemia is renal failure so if no medication works then the patient needs to undergo dialysis.

☆ Advanced airway is when intubation is needed. When breathing becomes independent of chest compressions. After introducing the tube after the first cycle of CPR, and connect the tube to the bag mask ventilator. Once you do that, you need to give a breath every 5-6 seconds -not paired with compressions-.

☆ If a shock is needed, resume CPR until the device charges up, ensure clearance then give the shock. Immediately resume CPR again.

Atrial fibrillation is not as life-threatening as ventricular fibrillation. The atrium contributes to only 15-20% of the blood pumped into the ventricle as most of it passes passively due to pressure difference. Ventricles do all the work by contracting strongly (isometric/ isovolemic) until the valves open then the blood passes the great vessels. In ventricular fibrillation, ventricles can't contract thus no cardiac output, no perfusion and thus it is deadly.

FOURTH SEMINAR

First thing we do to assess airway is ask the patient since if (phonation) occurs, we can assure both the airway + breathing.

☆ Most common cause of non-allergic angioedema is ACE inhibitors (Enalapril)

If the patient is using accessory respiratory muscles (sternomastoid, trapezes, scalene and serratus anterior) then it gives an idea about respiratory problems.

☆ Flail chest — occurs when 3 ribs are broken from two points (contiguous ribs). This leads to having this separated part of the chest wall moving in during inspiration and out during expiration. (paradoxical breathing).

- unstable blood pressure is mostly pointing to hypotension.

☆ unstable patients :

- _ GCS less than 8
- _ hypotensive
- _ unconsciousness
- _ cardio-respiratory instability .

Giving blood units:

Each blood unit contains around 400ml of blood in it. Each unit elevates hemoglobin by 1g/dl assuming the patient isn't bleeding as we give blood units.

☆ Electrolytes differences after blood transfusion: in the beginning, sodium is elevated upon infusion. For potassium, after minutes to an hour following blood transfer, some blood cells undergo lysis which results in potassium ions elevation.

- GCS ranges from (0-15)
- 13-15 - normal & oriented
- less than 8 - needs intubation
- (0-3) - brain death

FOURTH SEMINAR

Secondary survey :

- Brain hemorrhage is associated with Anisocoria (unequal size of the eyes' pupils)

☆ Intracranial bleeding can be associated with many symptoms depending on where the bleeding occurs. Common signs include:

- Battle sign (hematoma behind the ear)
- Raccoon eye
- CSF leak through the ears + nose (CSF rhinorrhea and CSF otorrhea)
- Bleeding behind the tympanic membrane

Neck trauma: zones are very important (zone 2 is the most prone to injury)

☆ Hard signs for trauma: Thrill & bruit

, pulseless, intensive bleeding (large hematoma)

☆ Soft signs : the exact opposite of hard signs

☆ the most common abdominal organ to be injured - blunt injury (spleen) - penetrating injury (liver)

In cases of undifferentiated hypotension, following a trauma and we have symptoms hinting at a shock but we don't know where is the bleeding. We perform FAST in order to assess bleeding in four quadrants.

☆ in traumatic events where the pelvis is injured, like car accidents and we suspect a pelvic fracture or something. And the patient is hypotensive, we perform suprapubic FAST to identify internal bleeding. However the first and most important step to managing this case is to apply a pelvic binder, which compresses the pelvic to stop bleeding and stabilise it in cases of fractures or blunt trauma.

FIFTH SEMINAR

Abdominal pain;
AAA

- post CATH results in femoral pseudo-aneurysm
 - most common site for aortic aneurysm is infra-renal. it's important to know the size to decide the management.
 - Patients with connective tissue disorders have increased risk of having triple A as well as aortic dissection.
 - screening for AAA is important for elderly individuals who have a smoking history. (using ultrasound which is very sensitive to screen and detect triple A, but it is insensitive to its complications/rapture)

- Grey turner sign and Cullen sign are signs of retroperitoneal hemorrhage. could also be seen in acute hemorrhagic pancreatitis or adrenal gland hemorrhage. (so they're not sensitive nor specific to triple A)

- when the patient comes with the triad, he needs to be administered to OR immediately because the patient is unstable. (same goes for acute surgical abdomen - in cases of board-like rigidity of the abdomen)
 - Another management to triple A is to use an EVAR, it is less invasive and similar to CATH. but its uses are limited due to how expensive it is.
 - surgical repair is also indicated when the expansion of the AAA exceeds 0.5cm every 6 months// 1cm per year.

FIFTH SEMINAR

Abdominal pain; cholecystitis

- Murphy's sign is positive when the patient ceases the inspiration when you palpate his RUQ area due to pain.
- Blood tests are done to rule out other disorders when someone comes with epigastric/RUQ pain. (Could also increase our suspicion)
- □ Ultrasound we would see the wall thickened >4 mm + double wall sign due to peri-cholecystic fluid (white black white) + and we can see the stone (its shadow) which is called wall acoustic shadow (wall echo shadow)
- Positive predictive value of Murphys sign is high but the negative predictive value is not high.
- □ Risk factors for biliary colic and cholecystitis (5F : female, fat, fertile, forty)
- □ When we have a huge stone at the neck of the gallbladder, which compressed the hepatic duct and results in jaundice. it is called Mirizzi syndrome

FIFTH SEMINAR

Abdominal pain;
biliary colic

- Biliary colic is two types (with stones ((calculus biliary colic) or without) often it starts as biliary colic then progresses to acute cholecystitis and so in.
- □ in this case examinations shows no signs of inflammation + negative Murphy sign. but we could see impacted stone on ultrasound (see the wall acoustic shadow of it)
- often comes after the person has had a heavy meal which caused the gallbladder to contract pushing a (cholesterol based stone up which would get lodged at the narrowest point of the bladder (the neck) which causes pain. most often it would spontaneously resolve as the stone falls back down into the gallbladder. When the stone stays there and doesn't get down, and the pain stays for longer than 6 hours and inflammation around the stones occurs, signs of inflammation will become visible on ultrasound. and the patient could also develop fever.
- □ when the patient has Charcot's triad (jaundice, RUQ pain, fever) along with altered mental status/ confusion + shock which is then called reynold's pentad.

FIFTH SEMINAR

Abdominal pain;
biliary colic

- the most common cause worldwide is alcohol. but here in our arab world it's biliary pancreatitis. second most common is due to hypertriglyceridemia
- I GET SMASHED (stands for the most common causes of acute pancreatitis)
- □ Handle bar injury is a high risk factor for acute pancreatitis (especially in pediatrics)
- Kidney function test, liver function test, along with CBC are required. kidney function test in order to check hydration and electrolytes. whereas liver function is important since if we have biliary pancreatitis, we will find high alkaline phosphatase and gamma GT (sometimes with obstructive jaundice)
- □ Lipase is more specific for acute pancreatitis. normal is 100-110 but in cases of acute pancreatitis it should be at least 3 times higher than normal.
- □ Gold standard is CT with IV contrast (swelling and signs of inflammation along with complications)

FIFTH SEMINAR

Abdominal pain

Acute appendicitis:

the most important indicative sign is the shifting pain.

□ the gold standard is CT with IV contrast. but we have many other options. but ultrasound is enough and good especially for pediatric age group.

- Dunphy sign could be done by asking the patient to cough, which causes irritation of the peritoneum.
- Markle sign is done by asking the patient to stand on their toes then stand normally again (like if they jumped) and that would provoke pain.
- Psoas sign if positive indicated that the appendix is close to the psoas muscle. While obturator sign indicates that the appendix is more pelvic.

Intestinal obstruction:

Colicky abdominal pain is caused by peristaltic movement, so if it is absent then no colicky pain.

Absolute constipation (Obstipation) no stool and no gas, whereas in constipation we don't have stool but we have gas.

- □ In mechanical, we have hyper sound but then it became absent. The opposite occurs with functional.
- □ The higher the obstruction the more the vomiting, the less the distention.

FIFTH SEMINAR

Abdominal pain; Ischemia

- Acute mesenteric ischemia could occur due to a thrombus that
- Chronic is diagnosed by exclusion. and it often occurs due to atherosclerosis that involves multiple arteries
- Vasospasm is a cause of acute ischemia and it occurs in patients taking cocaine or norepinephrine for a long period of time
- Venous obstruction like in cases of hepatomegaly
- □ The most common site for an embolism is the superior mesenteric artery embolism. and the most important site for ischemia
- □ Severe abdominal pain out of proportion to the physical examination is the typical presentation of a patient with mesenteric ischemia.
- □ Lactate is high in patients with mesenteric ischemia due to lack of perfusion so cells shift to anaerobic like any patient with sepsis .
- □ MI, Hemorrhage, Sepsis and dehydration increase lactate.
- WBCs are higher than 20000 in cases of mesenteric ischemia.
- Death rate without treatment is almost 100%, but with treatment it reaches 25%. it is very hard to diagnose and treat and often it is always delayed
- Treatment includes NBO, analgesia, antibiotics IV and IV fluids as well as anti-coagulant (heparin) unless we are gonna administrate the patient to OR to prevent massive bleeding.
- gold standard CT angio/ CT with IV contrast
- Chronic mesenteric ischemia it is called intestinal angina (when the patient eats, the demand increases for blood and thus results in pain)
- Intestinal angina and mesenteric ischemia can be caused by heart failure as well as hypovolemia

FIFTH SEMINAR

Abdominal pain

Gynaecological cases:

management in cases of ectopic pregnancy actually starts with stabilizing the patient with ABC, then do laparoscopy which is both diagnostic and therapeutic. if the patient was stable, we could use another treatment (terminating pregnancy with methotrexate) ,, but if the patient is unstable we can perform a salpingectomy (removal of the tube and the pregnancy) vs salpingotomy (incision and removal of the pregnancy from the tube then stitching)

- ectopic pregnancy starts with abdominal pain then bleeding, whereas miscarriage starts with bleeding then abdominal pain.
- In cases of ovarian torsion, the female comes to the clinic with severe vomiting (more than once) along with abdominal pain.
- Treatment of ovarian torsion starts with ABC, give analgesia (NSAIDS + opioids), then if stable perform a laparoscopy to uncoil the twisted ovary. if there's a cyst or mass we should resect it as it increases the risk of twisting again. If the ovary is necrotic, remove the ovary too.

AL-BASHIR'S

HOSPITAL NOTES

TUESDAY NOTES

- ☆ CABC in trauma :- C in the beginning is for stopping catastrophic bleeding
 - ☆ CABC in CPR :- C in the beginning stands for compression
 - ☆ A is for a patent airway and c spine protection (mainly c3-c4 to protect the phrenic nerve)
 - ☆ B is for breathing and adequate ventilation.
 - ☆ C is for circulation and CPR if needed.
 - ☆ D is for disability and neurological case. (assess GCS)
 - ☆ E is for exposure and environment.
-
- ☆ We need to perform CPR in 4 important presentations of patients:
 - Absent pulse
 - Absent breathing
 - Unconscious person
 - Abnormal breathing (respiratory arrest in which the respiratory rate is less than 8 breathes per minute) here we need a definite intubation right after the first cycle of chest compression.

Applying a tourniquet will compress skin first, veins (preventing return of blood) muscle, then arteries.

-In case we don't compress arteries, blood will pass to the arm, but does not return so it will shift to leave through the site of the wound and bleeding will be exacerbated.

TUESDAY NOTES

Contraindications for nasal airway are mid-face injury + basal skull fractures

Contraindications for oral airways are a patent gag reflex + a conscious patient.

- Rhinorrhea is different than otorrhea in that blood will create a surface tension and then it will concentrate. If CSF is present in the mixture with blood (rhinorrhea) it will expand avoiding the concentrated blood creating a halo sign.

- Hypoxic patient often has cyanosis & agitation
- Patients with CO₂ retention have cyanosis + sleepiness

☆ Sounds of the Airway (wheeze, stridor, crackles (fluids))

- Stridor: high pitch sound during inspiration. Causes include (angioedema & foreign body aspiration) involves upper airway.
- wheeze: expiratory sound in COPD and asthma patients due to inability to open small airways as they collapse during expiration. So air starts getting trapped inside the alveoli and results in hyper-inflated lungs. Involves lower airway.
- Crackles: sound heard due to fluids surrounding the alveoli. It could be due to pneumonia (associated with fever) or due to pleural effusion subsequent to heart failure.

Tachypnea: treat with oxygen (patient is hyperventilating due to lack of oxygen)

Bradypnea: treat with ambu bag (they have insufficient/ ineffective spontaneous breathing so they need positive pressure ventilation) — a breath every 6 seconds

☆ Pulse oximetry isn't accurate as it measures the amount of bonded oxygen to haemoglobin and not the partial pressure. (so polycythemia could give 100% oxygen saturation while the patient is hypoxic) That's why ABG is better although it is painful.

TUESDAY NOTES

- ☆ Stress hormones (CCGG) : cortisol, catecholamines, growth hormone and glucagon.
 - Insulin is the only hormone that works independently of oxygen presence.

Hypovolemic shock: Bleeding is either on the floor (external) or in four places more (internal bleeding - pelvis, long bones, chest and the abdomen). Patients appear pale, hypotensive, capillary refill time is >3 seconds (indicating hypoperfusion). So we need to perform FAST to pinpoint the internal bleeding site, if there is any the immediately send the patient to OR. We need to also check pulses and we could use it to estimate the BP if we don't have any equipment to measure it:

- popliteal is not normally felt.
- If the brachial pulse is intact, then systolic BP is >90
- If the femoral pulse is intact but not brachial, then systolic BP is between 70-80
- If the femoral and brachial and absent, but carotid intact then systolic BP is 60

Give fluids immediately if the patient is hypotensive in order to secure the blood pressure. In stage 1 and 2 of hypovolemic shock, give isotonic solution. Stage 3 give blood units.

☆ For each 1 L blood loss, give 2-3 L of isotonic solution. (to insure 1 L is going to the intravascular compartment, and the rest will shift to other compartments according to the normal distribution of fluids between different compartments) - we know $\frac{2}{3}$ of fluids are found in the extravascular compartment so if we only gave 1 L isotonic solution for every 1 L blood, only around 300-375ml will end up in blood vessels-.

TUESDAY NOTES

☆ First CBC in ER is a baseline, we need to perform it after giving fluids to the patient and restore a good volume. When patients bleed, they lose volume of blood not concentration so measuring HB will be incorrect until we restore the volume. Then it will be diluted and give us the correct measures. According to the results we get after restoring the volume, we decide if the patient needs blood or not. Give 10-20 ml per kg depending on the patient's status and co-morbidities.

☆ Signs of clinical brain death: 8 signs

- fixed dilation of the pupils.
- dull deep dark eyes.
- absent corneal reflex.
- absent laryngeal reflex .
- absent cough reflex .
- no reaction to pain/ no motor function
- EEG indicative of no brain activity
- no respiratory drive.

☆ Important pupil changes in emergencies:

- Anisocoria (unilateral pupil dilatation) is indicative of brain haemorrhage.
- Pinpoint pupils + shallow breathing is associated with opioid overdose.
- Pinpoint pupils + bad smell + SLUDGE symptoms (salivation, lacrimation, urinary incontinence, defecation, gastric cramps and emesis) are indicative of organophosphate poisoning. The patient would have poor response and is sweaty which can be seen in hypoglycaemic patient too (but the difference is that their skin is cold)

TUESDAY NOTES

☆ ECG notes:

The most important lead to check is lead 2 as it goes along with the electrical activity of the heart. But this doesn't mean we could ignore other leads.

- AVR is always negative (inverted in comparison to other leads). So if it was positive, then there is a problem.

When you are handed an ECG, check patient's information. Then check if the rhythm is regular or not. You do that by observing the p-wave, if it is present then we have a sinus rhythm. If the p-wave is absent, then we have abnormalities. (AF- then no sinus rhythm, or SVT where the p-wave goes missing within the QRS complex)

We can observe many abnormalities like ST elevation/depression, T inversion, peaked T wave like in hyperkalemia...

We need to perform cardiac enzyme tests in addition to ECG to diagnose an MI.

1. Myoglobin is the fastest to peak as it appears within 2 hours when it reaches its peak, before completely being washed out after 4 hours.
2. Troponin I is the best as it is the most sensitive and specific to the heart. It appears after 6 hours of an MI, peaks after 12 hours and disappears within a week.

TUESDAY NOTES

Treatment for MI is to give dual anti-platelets therapy. (Aspirin + clopidogrel)

- aspirin give 325 mg crushed/ sublingual; reduces mortality rate up to 25-40%
- clopidogrel give 4 tablets orally, each tablet contains 75mg.

Contraindications for aspirin: active peptic ulcer, allergy and asthma (give coated aspirin)

Give MONA to treat an MI but with caution with opioids and nitroglycerin.

We don't give high oxygen therapy because we are afraid of oxygen toxicity which is far worse than CO toxicity. It results in infusion injury and the production of reactive oxygen radicals which leads to cellular damage.

Methadone is more potent than morphine but we prefer morphine due to its vasodilation effect.

- inferior MI (contraindicates nitroglycerin and morphine as the patient is preload dependent). FILL THE TANK.

In cases where we need to give an electric shock, we could either give synchronised (which catches the R wave and gives a shock according to it) used in SVT. or unsynchronised (gives the shock blindly and not specifically, always giving maximum and it is used in CPR often)

WEDNESDAY NOTES

For injuries, we have killing chest injuries:

1. Tension pneumothorax
2. Hemo/pneumothorax
3. Open chest injury— place a tube distal to the site of injury + 3 sided occlusive dressing
4. Flail chest.
5. Cardiac tamponade — either blood (trauma or AD)/ fluid (heart failure and malignancy). First do an aspiration (10cc is enough) to restore cardiac output then an open surgery to get rid of clots.

AAA: pulsatile mass, use ultrasound.

PE we need CT angio as definitive investigation but first insure that the patient is not allergic and check his/her kidney function. We rely on well's criteria to determine how likely the complaint is due to DVT/PE. score less than 3, exclude PE. between 4-6, suspected. Higher than 6, most likely.

D-dimer isn't specific but if it was negative we can exclude PE. If positive, more investigation like CT if not performed before.

☆ if the patient has allergies, suspected internal bleeding or abnormal kidney function then do not perform CT angio.

Treatment of PE is to give heparin (80mg per kg) and warfarin (18mg per kg).

WEDNESDAY NOTES

- Regarding abdominal pain:

Most common complain in elderly, paediatric and pregnant women is perforation.

DM patients could either present with DKA or hyperosmolar status depending on which type of DM they have.

- DKA is observed in DM type 1 patients, mostly they're known cases of this disease or paediatric patients. Their glucose is often > 250 . They present with ketonemia/ketonuria AND acidosis. If one of these isn't present then it is not a DKA. Give fluids 6-8 L push.
- Hyperosmolar on the other hand occurs in patients with DM type 2. Their glucose level is > 600 . Their level of consciousness is reduced. They're more dehydrated than patients with DKA and need at least 12 L of fluids to compensate. Need to check potassium levels, if high, treat withOUT insulin.

If potassium level is between (3-5) mg, give insulin along with fluids. 0.1ml per kg infusion by an insulin pump. Give antibiotics as high glucose increases the risk of infections.

Most common causes of hyperosmolar is stress, starvation or a missed insulin dose.

In order to identify whether the patient has an acute renal failure or not we look at HB. if it is high, then that's an acute onset of renal failure. But if it was low, then it indicated a chronic case.

WEDNESDAY NOTES

If appendicitis is suspected, the gold standard is to do a CT angio, if the contrast passes then exclude appendicitis. If it doesn't then there's an inflamed appendix. The patient often tends to remain rigid and avoid any movement as it can cause irritation and provokes his/her pain.

We use alvarado score (elevated WBC + rebound tenderness + Fever + vomiting + loss of appetite)

Sometimes the patient can present with only constipation and anal pain.

- A patient with renal colic will move a lot, trying to find a comfortable position and they will appear restless.
- In female patients, abdominal pain could be due to ectopic pregnancy, ovarian torsion, a cyst or appendicitis. So clinical presentation is very important. We need to investigate these gynaecological causes to prevent adhesions that can lead to infertility.
- In young children(less than 3 years old) critical cause of abdominal pain is intussusception, which presents with a target sign on ultrasound imaging and current jelly stool. Treatment is air or fluid enema. If abdominal pain has been there for longer than 8 hours then we have a risk of necrosis so we need surgery.
- In elderly patients, critical cause of abdominal pain is mesenteric ischemia. Especially post-operatively. Or in patients with atrial fibrillation and a history of PE.

WEDNESDAY NOTES

In a patient with a ruptured oesophagus which could result from violent vomiting, we look for signs of boerhaave triad: a crushing heart sound during systole, subcutaneous emphysema and chest pain.

Treatment of hypoglycaemia is called the rule of 5.

Treatment of hyperkalemia is the rule of 10. (not working/acidosis then dialysis)

Causes of no urine output could either be:

- pre-renal (dehydration, hypovolemic shock, renal artery stenosis/closure)
- Intra-renal (kidney cancer, renal failure, polycystic kidney disease)
- Post-renal (stones and cancers)

We need to introduce a Foley catheter and slowly drain the urine to avoid the rupture of the bladder. After the first foley and drainage we can examine the new formed urine afterward and take its appearance into consideration.

Causes of constipation could be indicated by ABD:

- A for adhesions (post operative mainly)
- B for bulging (hernia and strangulation)
- C for cancer

If abscess is suspected we should not do a surgical incision or we would cause sepsis. The patient often has fever, presents with onset of 3-5 days of abdominal pain and the mass itself could appear yellowish on the skin.

In cases of hernia strangulation, we need to reduce the hernia before 6 hours has passed or necrosis has occurred and we need a surgery.