EMERGENCY ROOM MEDICINE

NOTES

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HIGH-YIELD NOTES

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Table Of Contents:

What's included: Ready-to-study summaries of a broad range of emergency medicine topics presented in succinct, intuitive and richly illustrated downloadable PDF documents. Once downloaded, you may choose to either print and bind them, or make annotations digitally on your iPad or tablet PC.

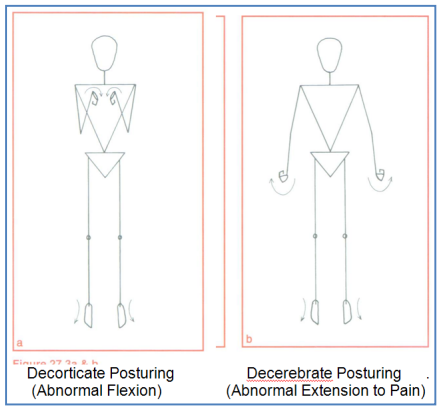
Free bonus: Emergency Medicine - Diagnosis and Management- 6th Ed

File List:

ASSESSMENT OF LOC:

- **Glasgow Coma Scale (GCS) - KNOW THIS:**
  - **Eyes opening in response to:**
    - 4. Spontaneous
    - 3. Voice
    - 2. Pain
    - 1. Not
    - (Score out of 4)
  - **Speech:**
    - 5. Oriented
    - 4. Confused Speech
    - 3. Inappropriate Speech
    - 2. Incomprehensible/Incoherent
    - 1. None
    - (score out of 5)
  - **Motor**
    - Can either -Obeying commands/localising painful stimulus (directly respond to painful stimulus)/ withdraws from painful stimulus/flexing (Very bad)/Extension (very very Bad)/None
    - (Score out of 6)
  - **(Less than GCS10 = Deeply unconscious; Less than GCS8 = Coma)**
  - **(Max Score = 15 = Awake, Alert, Responsive = Conscious)**
  - **(Min Score = 3 = Dead)**

Components:		Score:	
Eye Opening:	Spontaneously	4	
	To verbal command	3	
	To pain	2	
	Nil	1	
Verbal Response:	Orientated	5	
	Confused	4	
	Inappropriate words	3	
	Incomprehensible sounds	2	
	Nil	1	
Motor response:	Obeys command	6	
	Localises pain	5	
	Withdraws to pain	4	
	Abnormal flexion	3	(*decorticate posturing*)
	Extension to pain	2	(*decerebrate posturing*)
	Nil	1	



(*Remember *4 Eyes, Jackson 5, 6 Cylinder Engine*)

- **AVPU**
  - **Conscious**
    - **A**wake
    - *responding to* **V**oice
  - **Unconscious**
    - Only Responsive to **P**ain
    - **U**nresponsive

Table II: Example of AVPU Assessment Tool

A=Alert	Patient is alert and conscious
V=Verbal	Patient responds to verbal stimulus
P=Pain	Patient responds to painful stimulus
U=Unresponsive	Patient is unresponsive to any form of stimulus

PRIMARY SURVEY (ABSOLUTELY CRITICAL IN CASES OF LOC):

- **ABCDEFG:**
  - **Airway:**
    - **Commonest cause of Unconsciousness is Airway Obstruction**
    - **Clear The Airway:**
      - Left Lateral Position/Suction/Guedell Airway/Jaw Thrust
    - **(Generally GCS<8 require definitive airway management – Endotracheal tube/Intubation →Ventilation)**
  - **Breathing:**
    - Essential to ensure adequate ventilation
    - (without adequate ventilation, pt will suffer a secondary hypoxic brain injury)
    - **Give Oxygen**
    - **Maintain Sats & Normocarbia**
      - If hypoventilation → Hypercarbia → Cerebral Vasodilation → Increases Intracranial Pressure → Decreases Cerebral Perfusion.
  - **Circulation:**
    - Must ensure adequate circulation (IV fluids)
    - Cerebral perfusion pressure = Blood Pressure – Intracranial Pressure
      - As ICP goes up, must ensure at least a normal BP in order to maintain cerebral circulation
    - In head trauma, 5 minutes of Hypotension → 25% increase in mortality.
  - **Disability (Ie. Level of Consciousness):**
    - Assess level of consciousness & Document it.
    - Monitor for changes
    - Assess Pupillary Size/Reactivity
  - **Exposure:**
    - Examine the Whole Patient (Including the Back)
  - **DON"T EVER FORGET GLUCOSE**

- **CASES**
  - Give each of the following cases a AVPU rating and GCS.
  - **Case 1**
    - A 16 year old female is brought into your ED by ambulance after falling from a third floor balcony. Her eyes are closed despite painful stimulus and she's making groaning noises. She pulls her hands and feet away when painful stimulus applied to them.

    - AVPU……..
    - GCS……..

  - **Case 2**
    - A 24 year old man who is cheerfully intoxicated with alcohol is brought to your ED by Police. His speech is slurred he doesn't know what day or month it is, but is very happy to assist you with your physical examination of him.

    - AVPU……..
    - GCS……..

  - **Case 3**
    - A febrile 82 year old is brought into your ED by relatives. She is looking around the ED but every time you ask a question she tells you to "@#$% off" and this response never varies. She tries to hit you when you ask her to move her arms or legs.

    - AVPU……..
    - GCS……

  - **Case 4**
    - A six month old is brought your ED by his parents after suffering a generalised seizure. His eyes are open but he makes no response to any stimulus.

    - AVPU……..
    - GCS…… .
    - How useful do you think the AVPU and GCS scales are in this age group? Why?

Emergency Medicine Notes
Principles of Emergency Medicine; Triage

Small Stuff:

Key words + Definitions:

- **Emergency:**
  - "A medical condition requiring *Immediate Treatment*"
- **Triage:**
  - "The Process of Sorting Patients based on *Urgency*."
- **Resuscitation:**
  - To revive somebody from unconsciousness or apparent death
- **The Primary survey – ABCDE:**
  - Airway
  - Breathing
  - Circulation
  - Disability
  - Expose
- **Retrieval Medicine**
  - Pre-Hospital care performed in an emergency retrieval vehicle (Helicopter/Ambulance/Etc)
- **National Triage Scale:**
  - The standardized triage guidelines for Australian Hospitals.

Big Stuff:

Major Concepts:

- **What Constitutes an Emergency?:**
  - "A medical condition requiring *Immediate Treatment*"
  - (Not necessarily life-threatening)

- **Framework Dealing with an Emergency:**
  - **1. Triage:**
    - "The Process of Sorting Patients based on *Urgency*."
    - **Describe the principles of triage and priorities in care:**
      - Triage establishes *'Priorities of Care'* among groups of patients.
      - Triage done by asking: "This patient should receive medical care from a doctor in NO MORE THAN …?... Minutes."
      - Patient Assessment (Via the *Primary Survey*) is necessary to answer the above question.
    - **National Triage Scale:**
      - The standardized Triage Guide in Australia.

The scale has 5 categories and is also colour coded. It is described with examples of each category in the box below.

NTS Number	NTS Name	Maximum Waiting Time	Colour Code	Example
1	Resuscitation	0 min	Red	Cardiac Arrest Major Trauma
2	Emergency	10 min	Orange	Acute Severe Asthma
3	Urgent	30 min	Green	Acute Abdominal Pain
4	Semi Urgent	60 min	Blue	Sprained Ankle
5	Non Urgent	120 min	White	Repeat Script

- **2. 30-Second Patient Assessment in an Emergency: *"The Primary Survey"* - "ABCDE":**
  - **A**irway (With c.spine control)
  - **B**reathing
  - **C**irculation
  - **D**isability
  - **E**xpose

  } **Achieved by: "*Tell me your name*"**
  "*How are you today?*"
  "*Where does it hurt?*"

"Tell me your name":

- **Pt: "*I'm John Smith*" - You Know That:**
  - 1. Airway is Patent
  - 2. Pt's Breathing is Adequate.
  - 3. Consciousness = Adequate Cerebral Perfusion = BP is >Sys 80mmHg.
- **If Unresponsive:**
  - There is a Problem with One/More of the ABC.

***"How are you today?"*:**

- **Pt: "*Awful! This pain is dreadful*":**
  - You now know the *Problem* = Pain.

***"Where does it Hurt?"*:**

- **Pt: "*In my belly. Just here*":**
  - You now know the *Location* = Abdomen.

- **At the Same Time You are *LOOKING & FEELING*:**

LOOKING	***FEELING***
• APPEARANCE General, rash, pallor, cyanosis, jaundice • NECK Neck veins, thyroid • CHEST Respiratory distress, rate	• PULSE Rate, rhythm, volume • SKIN Temperature, sweating

You Now Have an Approximation of the First 3 Vital Signs:
- Respiratory Rate. (High/Normal/Low)
- Pulse (Tachy/Brady; Strong/Bounding/Thready)
- Temperature (Hot-ish/Cold-ish)

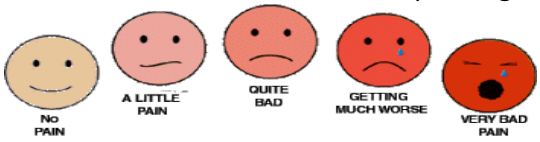
You Also Have an Idea of O_2 Saturation:
- If Central Cyanosis = Low O_2 Saturation.
- If No Cyanosis = O_2 Sats are Ok (for now)

Other Observations:
- Normal Skin Colour = Unlikely Anaemia/Jaundice/Cyanosis/Shock.
- Normal Pulse = Cardiac Output is OK; No major Arrhythmias
- Warm, Dry Extremities = Adequate Peripheral Perfusion; Not Hypothermic
- Regular, Symmetrical Chest Movement = Unlikely Pneumothorax/Acidosis.
- Soft Neck with NO JVP = No RHF, No Goitre, No Neck Trauma.

NB: ***As a Problem is Identified/Arises, It is IMMEDIATELY Dealt With*:**
- If Blocked Airway → Clear It
- If Not Breathing → Ventilate
- If Pulse is Weak/Thready → Get IV Access

- If in Pain → Give Analgesia

- **3. Assess the Patient's Pain Status:**
  - ***Analgesia should be the NEXT PRIORITY *After Prolonging Life*:**
    - Is it needed?..If So:
      - What?
      - Which Route?
      - What Dose?
  - **Pain Assessment:**
    - **Site of Pain:**
      - Location
      - Radiation?
    - **Circumstances @ Pain Onset:**
      - Trauma?
      - Ie. What caused it.
    - **Character of Pain:**
      - Pain Description – (Sharp, throbbing, aching, dull, burning)
    - **Intensity of Pain:**
      - @ Rest
      - On Movement
      - Duration
      - Continuous/Intermittent
      - Aggravating Factors
    - **Somatic Pain?**
      - Sharp, hot or stinging
      - Well localised
      - Local Tenderness
    - **Visceral Pain?**
      - Dull, cramping pain
      - Poorly localised
      - Local Tenderness or Referred Tenderness
      - Related symptoms – (Nausea, Sweating & CV Changes)
    - **Treatment?**
      - Current & Previous Meds.
  - **Pain Scales – (Because Pain is a *Subjective* Experience):**
    - **Eg. Categorical Scales:**
      - Verbal Descriptors (mild/moderate/severe/excruciating/agonising)
      - Numeric (0-10)
    - **Eg. Visual Analogue Scale:**
      - 5 Emoticon faces with corresponding scores.



    - **NB: Different Pain Scales are used for different people:**
      - Eg. Deaf people are more suited to picture-based pain scale.
      - Eg. Blind people are best suited to verbal/numeric scales.
      - Eg. Babies/Foreigners are best suited to picture-based scales.
  - **Importance of Analgesia:**
    - Relieves pain
    - Improves Patient Communication
    - Improves Patient Cooperation
    - Can improve symptoms (eg. Pain-related tachycardia)
  - **Advantages and Disadvantages of various Forms of Analgesia:**
    - **Oral Analgesia (Paracetamol/Aspirin) –** Cheap & Easy; But Weak
    - **Parenteral Analgesia (IM/IV opiates)** – Strong; But More Expensive/Complicated

        - **Regional Blocks (LA injection around a nerve) –** Only Good for Isolated Injuries
    - **4. Early Management:**
        - **Prevention of Morbidity & Complications**
        - **Minimises Suffering**

- **Importance of Communication:**
    - ***Communication is Critical for a Coordinated Effort & Positive Patient Outcome:**
        - Between Doctors & Nurses
        - Between Doctors & Patient
        - Between Doctors & Family

- **Impact of Emergencies:**
    - On Patients
    - On Families
    - On Communities
    - On Health Workers

GLS Stuff:

Triage & Assessment Questions:

NTS Number	NTS Name	Maximum Waiting Time	Colour Code	Example
1	Resuscitation	0 min	Red	Cardiac Arrest Major Trauma
2	Emergency	10 min	Orange	Acute Severe Asthma
3	Urgent	30 min	Green	Acute Abdominal Pain
4	Semi Urgent	60 min	Blue	Sprained Ankle
5	Non Urgent	120 min	White	Repeat Script

- **1. Assign each of the following patients to a Triage Category:**
  - A) Jim is 26. He is a chef at the local pub. He has cut his forearm while filleting fish. He cannot bend or feel most of his fingers and has a heavily blood stained bandage wrapped around his arm. He has no other injuries and all his observations are normal.
  - B) Jane is 40. She has been vomiting for 3 days and is a known diabetic. Her blood sugar level is "HI", pulse is 15, respiratory rate 35, blood pressure 75/40 mmHg. She is semiconscious, groaning but does localise to pain and open her eyes.
  - C) Jeffery is 52. He is a local property owner. He has tripped in a pothole and has twisted his ankle. He is still able to walk. His observations are all normal.
  - D) Jerry is 50. He has had a cough for 3 days and now seems quietly confused muttering to himself about purple kangaroos. His respiratory rate is 40, oxygen sats 82%, pulse 120 and blood pressure 100/65.
  - E) Jenny is 48. She has suddenly collapsed in the middle of a heated debate. She has no pulse and no spontaneous respirations. A bystander is doing CPR.

Patient 1 is a triage category 3
Patient 2 is a triage category 2
Patient 3 is a triage category 4
Patient 4 is a triage category 2
Patient 5 is a triage category 1

- **2. Assign each of the following patients to a Triage Category:**

PATIENT	DESCRIPTION	TRIAGE CATEGORY	REASON
1	67 yo male - VF Arrest	1	Cardiac Arrest
2	23 yo female - Repeat script for OCP	5	Repeat Script
3	38 yo male - Inversion injury to ankle. Swollen, sore, unable walk.	4	Sprained Ankle
4	12 yo male - Acute asthma. RR 40, sats 85% on air, silent chest	2	Acute Severe Asthma
5	26 yo female - 8 weeks pregnant with lower abdominal pain and vaginal bleeding. Observations all normal.	3	Acute Abdominal Pain
6	2 yo female - Febrile convulsion. Seizure stopped. Now awake.	3	Febrile Convulsion implies an Active Infection → ?Meningitis
7	18 yo female - MVA high speed. Unconscious, P120, BP 75/40	1	Acute Trauma
8	73 yo male - Rash for 2 months.	5	Not Urgent
9	43 yo female - Vomiting and diarrhoea 2 days. Now unable to keep fluids down.	3	Needs IV
10	33 yo male - Recent admission with depressive illness. Wants to talk to someone as recurrence of suicidal thoughts. Has made no attempts.	?	?

Emergency Medicine Notes
Cardiovascular Emergencies & Shock

Objectives:

- To be able to describe the physiological basis for fluid resuscitation and differences between various IV fluids

WHAT IS A CARDIOVASCULAR EMERGENCY ?

Consider these cases:

- **CASE 1**
  - 19 year old female in an MVA
  - Pale and sweaty with a distended abdomen (Indicates haemorrhage in Abdomen)
  - P is 140 and BP is 65 / 40
  - Was this a CVS Emergency ? YES
  - Why? She's in shock (Tachycardic & Hypotensive)
- **CASE 2**
  - Robert is an 81 year old retired nurse
  - He has presented complaining of palpitations and feeling weak and light headed
  - His pulse is 20 and difficult to feel (:. Hypotensive)
  - Was this a CVS Emergency? YES
  - Why? Bradycardic & Probably Hypotensive → Probably a form of Heart block. Requires Pacing
- **CASE 3**
  - Mike is a 54 year old mechanic
  - He has developed a crushing pain in his central chest and left arm while walking
  - The pain has lasted 2 hours and is getting worse
  - He feels nauseated and breathless
  - Was this a cardiovascular emergency? YES
  - Why? Possible Myocardial Ischaemia &/Or Infarct.

What Constitutes CARDIOVASCULAR EMERGENCIES:

- **1. Disturbance of BP:**
  - Shock
  - Hypertensive Crisis
- **2. Disturbance of Pulse:**
  - Brady Arrhythmias
  - Tachy Arrhythmias
- **3. Disturbance of Function and Form**
  - Pump Failure
  - Cardiac Tamponade
  - Valve dysfunction
  - Aortic Dissection
  - Myocardial Ischaemia

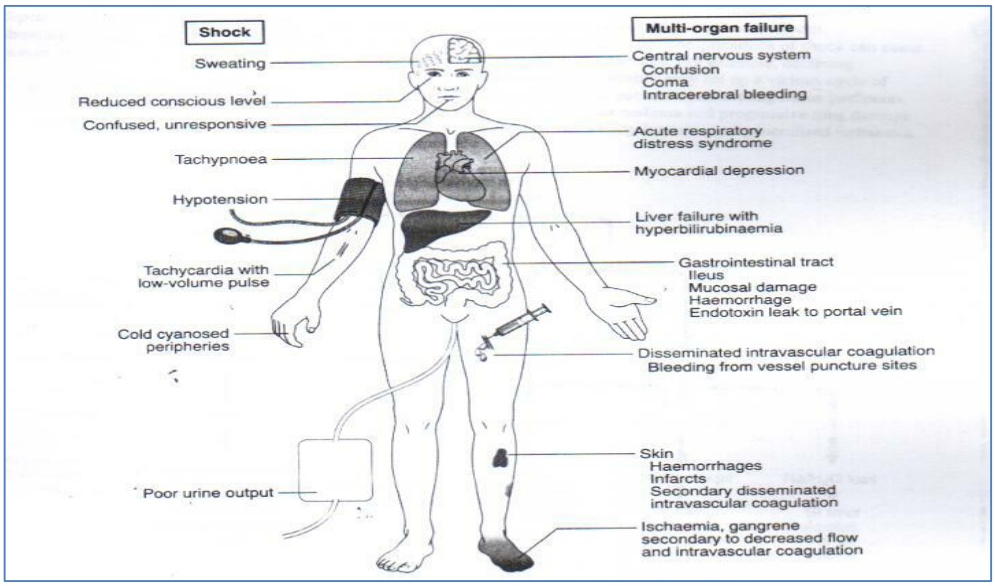
CV EMERGENCIES: 1 – BP DISTURBANCES
(Shock / Hypertensive Crisis)

SHOCK:

- **Medical Definition:**
  - EXAM Definition: Indequate Perfusion of *Vital* Organs (Heart/Brain/Kidneys)
  - Other definitions:
    - "A rude unhinging of the machinery of life"
    - A state of inadequate tissue perfusion.
  - NB: There are no numbers in definition. Shock is *relative*.
    - (Eg. A BP of 90 may indicate shock in a person with normal BP of 160)

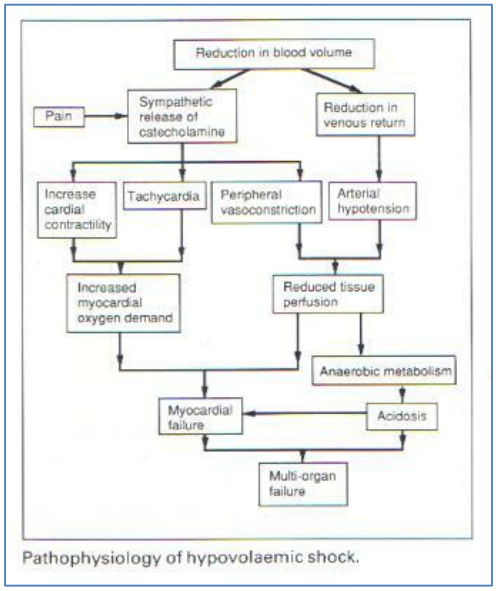
CLINICAL EFFECTS OF SHOCK:

- **Inadequate Tissue Perfusion →**
  - CNS Features such as impaired consciousness, Confusion, unresponsiveness, Coma;
  - Renal features such as ↓Urine Output;
    - (Ie. Hypoxia of Brain & Decreased Filtration in Kidney)
- (:. CNS & Renal Function can be used to measure *End Organ Perfusion*.)

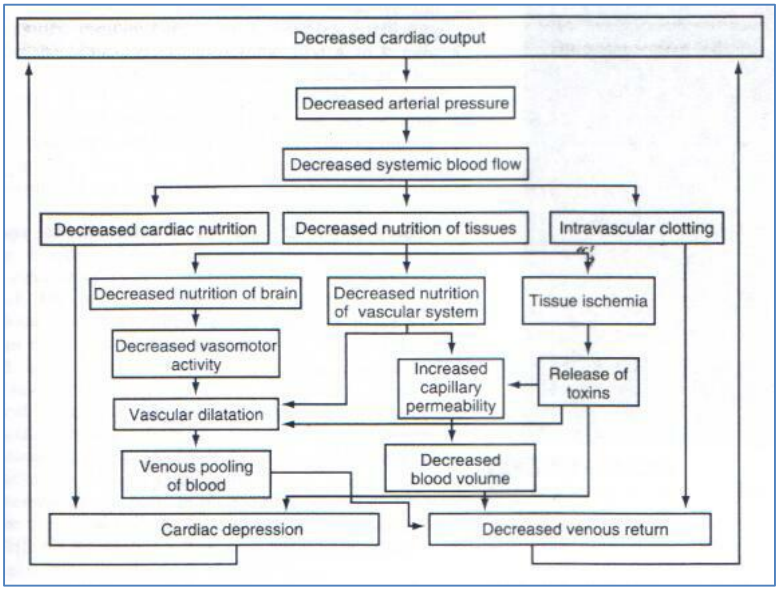


WHY DOES SHOCK OCCUR ?

- **From failure of any of the components of the cardiovascular system → State of Decreased Flow:**
  - Pump
  - Circuit – Loss of Vasomotor Tone
  - Fluid – Eg. Hemorrhage/Fluid Loss/Dehydration.



Pathophysiology of hypovolaemic shock.



Hypovolaemic Shock Vs. Cardiogenic Shock

TYPES OF SHOCK

- **LOW CARDIAC OUTPUT SHOCKs**
  - **CARDIOGENIC** – (Pump Failure – Not pumping enough)
    - Inability of the heart to pump adequately
    - Possible Causes:
      - AMI (May survive if quick treatment)
      - Valvular Problems (Eg. From RHD; Probably not fit for surgery & probably die)
      - Cardiomyopathy (Global myocardial weakness – the heart just cannot pump)
        - Require a heart transplant/mech.heart for survival.
      - Myocarditis
    - e.g. M.I., valve dysfunction, arrhythmias, myocarditis, Cardiomyopathy.
  - **HYPOVOLAEMIC** – (Not Enough Blood volume)
    - **Haemorrhagic:**
      - Blood Loss
      - Internal/External Haemorhage
    - **Fluid Loss:**
      - Eg. Loss of Gastrointestinal Fluids (Vom/Dia)
      - Eg. Metabolic (eg. Diabetic ketoacidosis) can lose lots of fluid in their Urine.
      - Eg. Large Seeping Burns
      - Eg. Heat Stroke/Sweating
  - **DISTRIBUTIVE** – increased size of circuit or capacitance due to altered vascular tone.
    - **Septic Shock:**
      - Gram Negative Sepsis: Immune Response to liberated Endotoxin (LPS) → Cascade of Cytokine Effects → ↑Cap. Permeability, Vasodilation → Hypotension → Shock.
      - May have Fever or be Hypothermic
      - Warm & Well Perfused (can → Cold, clammy & shut down)
      - Decreased Peripheral Resistance (can → ↓↓Diastolic Pressure → Compensatory Tachycardia & ↓Myocardial perfusion → myocardial Ischaemia.)
      - Causes:
        - Eg. Bowel Perforation
        - Eg. Meningococcus (Neisseria Meningitidis) can progress to septic shock.
        - Eg. Melioidosis (A gram neg that lives in the soil)
      - (incl. Increased Capillary Permeability)
      - → ↓Peripheral Resistance
      - → ↓Cardiac Perfusion
    - **Anaphylactic Shock:**
      - Acute Allergic Reaction (IgE-Mediated Mast-Cell Degranulation)
        - → Release of Vasoactive Mediators (incl. Histamine) → Loss of Vasomotor Tone → Hypotension.
        - Urticarial Rash
        - Severe Bronchospasm
        - Rapidly Aggressive Systemic Oedema (Esp. Face & Airway)
      - (Mortality of up to 50%)
      - 2 Allergic Syndromes:
        - True Anaphylaxis:
          - Prior exposure → Cross Bridging of IgE → mast Cell Degranulation
        - Anaphylactoid (Clinically indistinguishable):
          - No Prior Exposure
  - **OBSTRUCTIVE** – blockage to outflow
    - e.g. massive PE, cardiac tamponade, tension pneumothorax

COMPENSATORY MECHANISMS:

- **All compensation relates directly or indirectly to the formula:**
  - (CO = SV x HR)
- **(IMMEDIATE) Sympathetic Stimulation:**
  - Baroreceptors (in blood vessels) and CNS ischaemic response → Release of Catecholamines:
    - β_1 Receptors on Heart→
      - ↑HR (Chronotropic) → ↑CO
      - ↑Contractility (Inotropic) → ↑SV→ ↑CO
    - α_1 Receptors in Vessels→
      - →Arteriolar Vasoconstriction →↑Total Peripheral Resistance →↑BP.
      - →Venous Constriction →↓Capacitance and ↑Venous Return → ↑CO & BP.
  - (with sparing of cardiac and cerebral circulations)

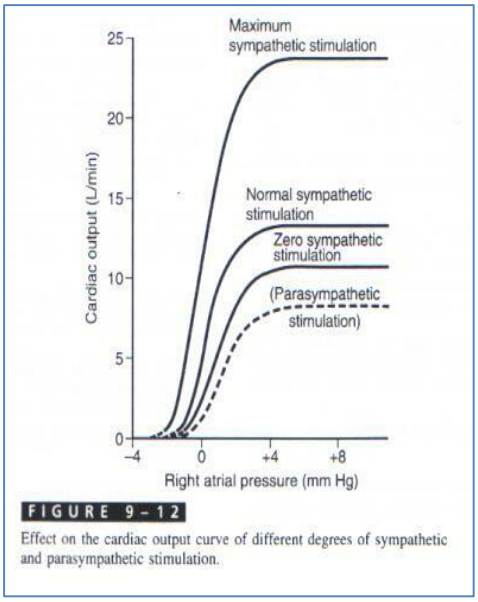
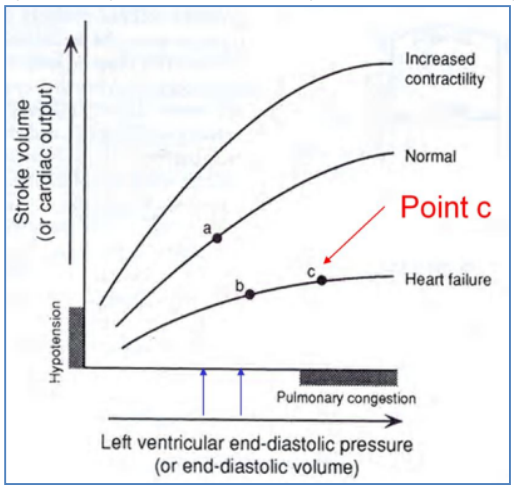


FIGURE 9-12

Effect on the cardiac output curve of different degrees of sympathetic and parasympathetic stimulation.

- **(DELAYED) Angiotensin and Vasopressin (ADH):**
  - Constrict Peripheral Arteries →↑Total Peripheral Resistance →↑BP
  - Minimises Urine Output → ↑Circulating Fluid Volume → ↑BP
- **Other**
  - Thirst Stimulated
  - EPO Produced
  - Increased salt appetite
- **Frank Starling Mechanism:**
  - ***"↑Preload → ↑Stroke Volume"***
    - Incomplete Chamber Emptying →↑PRELOAD → ↑Cardiac Output BY ↑SV
  - (*↑Preload → helps stretch myocardial fibres → Enhance cardiac function up till the point where there is no longer any overlap between the myocardial muscle fibres.*)



- ***The Concept of "CARDIAC RESERVE"*:**
  - ***Cardiac Reserve** = Maximal % that CO can Increase Above Normal.*
  - ***Normal Cardiac Reserve** = 300-400%*
  - (*The higher the Cardiac Reserve the lower the chance of shock after losing 'x' amount of blood*)

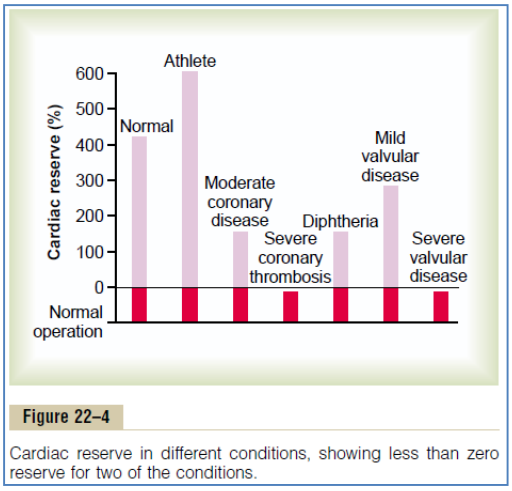


Figure 22–4

Cardiac reserve in different conditions, showing less than zero reserve for two of the conditions.

DETERMINANTS OF CARDIAC OUTPUT

- **Preload, Contractility, Afterload**

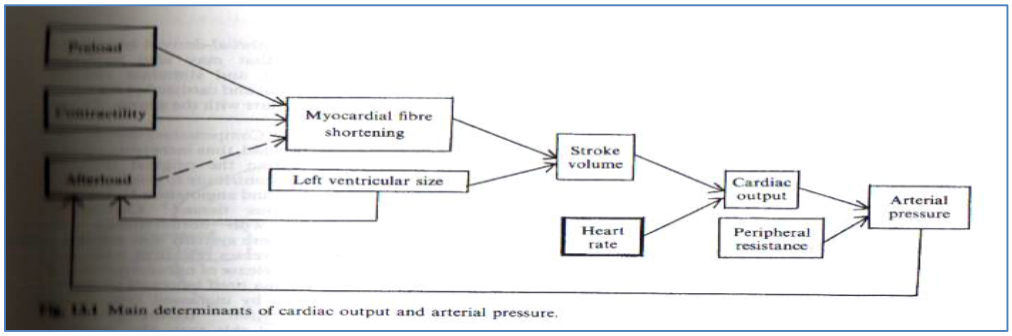


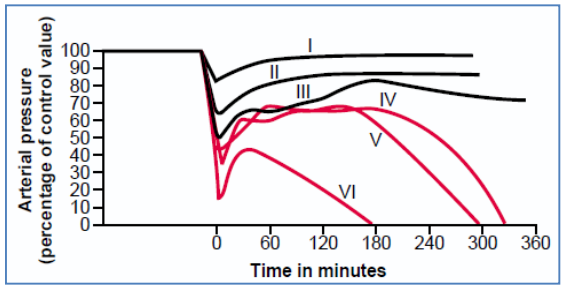
Fig. 13.1 Main determinants of cardiac output and arterial pressure.

HOW DO WE RECOGNISE AND ASSESS SHOCK ?

- **An Obvious cause. (Eg. Bleeding, seeping burns)**
- **Signs and Symptoms of Inadequate Tissue Perfusion. (Eg. Cold, sweaty)**
  - Decreased Renal Perfusion → ↓Urine Output
  - Decreased Cerebral Perfusion → Confusion, Anxiety, Agitation
  - Decreased Myocardial Perfusion → Ischaemia, Hypotension
    - NB: Since the coronaries come off the base of the aorta, if a patient is in shock with signs of myocardial ischaemia (Ie. ST-Depression), they are in SERIOUS TROUBLE.
- **Evidence of Compensation. (Eg. Tachycardia)**
- **Hypotension (Despite Compensation)**

STAGES OF SHOCK

- **1. Nonprogressive (Compensated) Shock:**
  - Beginnings of shock
  - Adequately Compensated → No Hypotension or Hypoperfusion
- **2. Progressive Shock:**
  - No longer able to compensate → Showing signs of Hypotension & Hypoperfusion
  - Still reversible with treatment
- **3. Irreversible Shock:**
  - No treatment is sufficient
  - Pt WILL Die
  - Vicious Cycle



Time course of arterial pressure in dogs after different degrees of acute hemorrhage. Each curve represents average results from six dogs.

GRADING SHOCK:

- **Grade 1:**
  - <15% Blood Loss (750mL)
  - Mild resting Tachycardia (+ Vasoconstriction)
  - *Normal BP Maintained*
- **Grade 2:**
  - 15-30% Blood Loss (750-1500mL)
  - Moderate Resting Tachycardia (+ Vasoconstriction)
  - Extended Cap.Refil time
  - *Normal BP Maintained*
- **Grade 3:**
  - 30-40% Blood Loss (1500-2000mL)
  - Severe Resting Tachycardia (+ Vasoconstriction)
  - *Hypotension*
  - (Compensatory Mechanisms Beginning to Fail)
    - Eg. Low Urine Output
- **Grade 4:**
  - 40-50% Blood Loss (2000-2500mL)
  - *Severe Hypotension*
  - End Organ Failure & Death

- **NB: Notice how Arterial Pressure is the *LAST* thing to Fall following Haemorrhage?**
  - This is because it is maintained by Vasomotor-Constriction for a while.
  - (IE. Shock can be present before any change in BP)
  - (NB: Without treatment, people suffering from haemorrhagic shock will 'bleed out' → No CO)

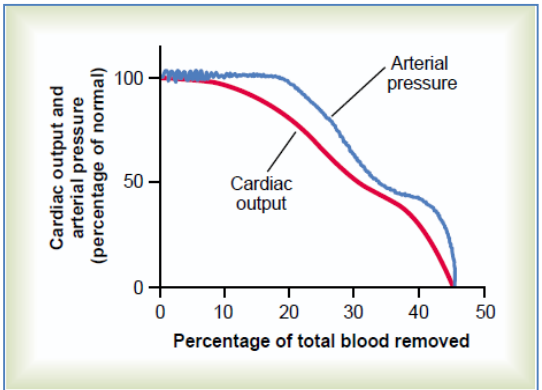


Figure 24–1

Effect of hemorrhage on cardiac output and arterial pressure.

- **Assessment of Blood Loss: (EXAM: Memorise this Table)**

Class	% Volume	Symptoms	Signs (P,BP)	Urine output	Treatment
I	<15% <750 ml	Mild	Normal	Normal	Cup of Tea Biscuit
II	15-30% < 1.5l	Anxious	P 100-120 (→Tachy) BP normal	Decreased	2L IV fluid
III	30-40% < 2l	Agitated lethargic	P 120 BP 80/60 (BP Falling)	Minimal	2L IV Fluid & 2 Units of Blood
IV	>40% > 2l	Drowsy Unconscious Crash	P 140 Very low (40)	None	IV Fluid Blood Surgery

- **NB: *The Aim of Fluid Resuscitation is to bring the patient up to Class I***

- **Good to know where the fluids that you give actually go:**
    - **Eg. Average 70kg man**:
        - 60% water :. Total body water (42L)
        - 2/3 Intracellular Fluid (28L)
        - 1/3 Extracellular Fluid (14L)
        - 5L blood [1/3 RBC (1.5L); 2/3 Plasma (3.5L)]
- **So What happens to the Different IV Fluids?:**
    - **Glucose is actively taken into cells**
        - (volume of distribution is large, as none effectively remains in blood)
        - Therefore Glucose IS NOT suitable for pressure fluid resuscitation.
    - **Crystalloids (ie. Saline) – is kept in ECF by Na/K-ATPase**
        - :. Volume of distribution = 14, and of that, 25% remains in circulation (3.5L)
    - **Colloid, kept in blood by capillary membrane (Albumin, Gelatine, etc)**
        - Volume of Distribution is 3.5L, and ALL of that remains in Circulation.
    - (500mL of Colloid = 2L of Crystalloid) – (NB: So long as you give the right amounts,
- **Blood:**
    - **The best fluid to replace blood loss is Blood**
    - But, either saline/hartmanns or colloid are still ok.
    - NB: Blood has risks (immunogenic/infections/etc)
    - NOT Glucose

TREATING SHOCK:

- **Hypovolaemic Shock:**
    - Recognise severity of the loss (Ie. Estimate how much has been lost – good 1st step to treatment)
    - Replace Loss (Typcially crystalloid Fluids)
    - Stop ongoing Loss (May require surgery)
- **Septic Shock:**
    - IV Fluid Resuscitation
    - Appropriate Antibiotics (Broad Spectrum Initially; then Specific Therapy following blood cultures)
    - Inotropic Support → Improve myocardial perfusion.
    - Vasopressors (eg. Noradrenaline or Vasopressin) (to ↑PVR → ↑Diastolic Pressure → ↑Myocardial perfusion)
    - May require surgery (eg. Bowel Perforation)
- **Anaphylactic Shock:**
    - ABC Primary Assessment
    - Adrenaline (+ Steroids – but too slow to be of use)
        - A:
            - May need early airway management (Eg. ET Intubation)
            - Nebulised Adrenaline (effective against airway oedema & Bronchospasm)
        - B:
            - Nebulised Adrenaline
            - Or IV Adrenaline
        - C:
            - IV Fluids
            - Adrenaline (IM / IV) (IV is better than IM/SC as it is absorbed faster)
- **Cardiogenic Shock:**
    - Angioplasty/Reperfusion
    - Inotropes
    - ?Transplant?
- **Mechanical:**
    - **Tamponade** (often due to trauma/Infection):
        - Pericardiocentesis
    - **Tension Pneumothorax:**
        - Thoracocentesis
    - **Pulmonary Embolus:**
        - Thrombolysis

GLS – SHOCK CASES:

Case 1 - Bart:

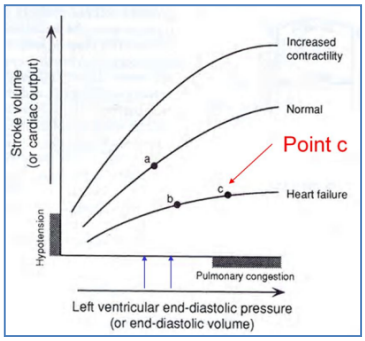
- He is pale and sweaty, has a distended abdomen and obvious bilateral femoral fractures. His pulse is 140 and his blood pressure is 75/40.

- **What signs of shock are evident?**
  - Pale and Sweaty
  - Tachycardic
  - Hypotensive
- **What Type of Shock is This?**
  - → Hypovolaemic (Haemorrhagic) Shock:
    - Seems to be bleeding into abdomen → Hypovolaemia → ↓CO →Hypotension + Compensatory Tachycardia
- **Could Bart be shocked without a change in BP?**
  - Yes. Young, healthy people are able to compensate for up to 1500mL of blood loss by Tachycardia & Vasopression, but then deteriorate rapidly afterwards.
- **Is this consistent with our definition of shock ?**
  - No - Our definition stipulates a loss of blood pressure.
  - (*Clinically important - Need to remember that relying on blood pressure changes alone to diagnose shock means that we will not recognise shock until a patient has lost 30 - 40 % of their blood volume (class 3)*)
- **Initial Treatment:**
  - Fluid Replacement – (For Hypovolaemia)

Case 2 – Homer:

- Suddenly collapsed and clutched his chest. He is pale and sweaty. His pulse is 40 and his blood pressure is 85/60. He is feeling short of breath. You note that his JVP is raised. Moe thinks that Homer has had a heart attack.

- **What signs of shock are evident?**
  - Pale & Sweaty
  - Hypotensive
  - Bradycardic → Suggests Cardiogenic Shock
- **What Type of Shock is This?**
  - → Cardiogenic Shock:
    - Myocardial Infarction → Heart Failure (↓CO) & Bradycardia→ ↓BP.
- ***Homer's ECG has shown an anterior myocardial infarction*. Why might this have caused him to be shocked ?**
  - Myocardial Infarction → Disrupted heart Contraction & Conduction → ↓HR (in this case), and ↓CO
- **If Homer has a heart that is not pumping properly (decreased contractility) which direction will his Starling curve move?**
  - His starling curve will shift Downwards (Ie. Stroke Volume & CO will be Less @ any given End-Diastolic Volume)



- **Initial Treatment:**
  - Inotropes – (For the Bradycardia)

Case 3 - Marge:

- Marge has bought a special new brand of extra strong hairspray. Begins to feel very itchy and notices small bumps coming up on her head. She collapses. She is conscious but confused. Skin is bright red & covered in raised lumps. Her pulse is 120 and her blood pressure is 90/60.

- **What signs of shock are evident?**
  - Tachycardic
  - Hypotensive
- **What Type of Shock is This?**
  - → Distributive (Anaphylactic) Shock:
    - Itchy, red, bumps on skin + History of new Hairspray → Allergy (Systemic release of Histamine & Other Vasoactive Mediators → Loss of Vasomotor Tone → ↓BP & Compensatory Tachycardia.
- **What has happened to her:**
  - Venous Tone? Decreased
  - Venous Capacitance? Increased
  - Venous Return? Decreased
  - Preload? Decreased
  - Stroke Volume? Decreased
  - Cardiac Output? Decreased
- **Why has she collapsed?**
  - Due to Postural Hypotension → Hypo-Perfusion of Brain → Momentary loss of consciousness. (Regained once supine)
- **Initial Treatment:**
  - Adrenaline – (For the Anaphylaxis)

Case 4 – Lisa:

- Lisa has been playing her saxophone. She collapsed gasping for breath. Her pulse is 120 and her Blood Pressure is 65/45. Neck veins are distended. No breath sounds on the left side. Tension pneumothorax.

- **What signs of shock are evident?**
  - Tachycardic
  - Hypotensive
- **What Type of Shock is This?**
  - → Obstructive Shock:
    - Spontaneous Tension Pneumothorax from Playing Saxophone → ↑Intra-Thoracic Pressure → Inhibits Cardiac Filling (Seen as raised JVP) → ↓CO → Hypotension & Compensatory Tachycardia
- **How might Lisa's tension pneumothorax cause her to be shocked?**
  - If pressure in the tension pneumothorax is high enough it may:
    - Compress (Decrease) Venous Return to the chest & heart → ↓CO → Shock
    - Shift the Mediastinum such that one/more of the Great vessels gets 'kinked' → ↓CO → Shock
- **Initial Treatment:**
  - Chest Drain – For the Pneumothorax.

Case 5 - Maggie:

- Her dummy fell in dog poo. Now very sleepy. Her skin is a mottled grey colour. Pulse of 180 and blood pressure is 60/40. Angry inflamed area on her face which has pus in the middle of it.

- **What signs of shock are evident?**
  - Tachycardic
  - Hypotensive
  - Grey, colourless skin
- **What Type of Shock is This?**
  - → Distributive (Septic) Shock:
    - Bacterial infection from dog faeces → Endo/Exo Toxin → Systemic Cytokine Release → Loss of Vasomotor Tone → ↓BP → Compensatory Tachycardia
- **How have the following been affected ?**
  - Venous tone? Decreased
  - Vessel Permeability? Increased
  - Myocardial function? Inotropic
- **Initial Treatment:**
  - Antibiotics
  - (Also check Lactic Acid Level):
    - High levels can indicate severe infection
    - & Can indicate lack of Tissue Perfusion & Production of Lactica Acid by Anaerobic Metabolic Pathways.

CV EMERGENCIES: 1 – BP DISTURBANCES
(Shock / Hypertensive Crisis)

HYPERTENSION (HT):

- **= A persistently high blood pressure >140/90**
- Untreated Hypertension→↑Risk of Stroke, Coronary Artery Disease (& MI), & Aneurysm.
- **95 % = Essential (Primary) HT:**
  - (HT with *no* Direct Medical Cause)
  - Usually Asymptomatic.
  - Most common in Older (>30) People.
- **5 % = Secondary HT:**
  - (HT caused by other Conditions – eg. Affecting Kidneys/Arteries/Heart/Endocrine)
    - Eg. Cushing's Syndrome
    - Eg. Hyperthyroidism (Thyrotoxicosis)
    - Eg. Pre-Eclampsia (HT in pregnant women)
  - Most common in Young (< 30) People.

THE EMERGENCY – Hypertensive CRISIS (Malignant/Accelerated Hypertension):

- **= HT Associated with Rapidly Progressive Organ Damage (renal failure, encephalopathy, retinopathy)**
  - "Malignant" or "Accelerated" Hypertension is rare; & can complicate HT of any Aetiology.
- **Pathophysiology Not well Understood:**
  - Failure of normal Autoregulation and an abrupt ↑Vascular Resistance are typically initial steps.
  - **Common Causes:**
    - Cessation of Antihypertensives (Rebound HT)
    - Autonomic Hyperactivity
    - Drug use (Cocaine/Amphetamines)
    - Glomerulonephritis
    - Head Trauma
    - Tumours
    - Pre-Eclampsia
  - **Is a Vicious Cycle of Homeostatic Failure → Potential *Multi-Organ* Involvement:**
    - →Cerebral Infarction
    - →Pulmonary Oedema
    - →Hypertensive Encephalopathy (Cerebral Oedema)
    - →Congestive Heart Failure
- **Symptoms Include:**
  - Headache
  - Drowsiness
  - Confusion
  - Vision disorders (Due to Papilloedema – Swelling of Optic Disc due to Cerebral Oedema)
  - Nausea
  - vomiting
- **Management:**
  - **Aim:** To *Smoothly* Reduce BP *over 24 to 36 hours* to 150 / 90
    - (Excessive reduction may → Coronary/Cerebra/Renal Ischaemia)

CV EMERGENCIES: 2 – DISTURBANCES OF PULSE
(*Tachy-Arrhythmias / Brady-Arrhythmias*)

DISTURBANCE OF PULSE

- **Working knowledge of ECG and Cardiac Cycle is Essential:**
  - **What does the P wave represent ?**
    - Atrial Depolarisation
  - **What does the QRS complex represent ?**
    - Ventricular Depolarisation
  - **What does the T wave represent ?**
    - Ventricular Repolarisation
  - **What rate is normal ?**
    - 70bpm
  - **What is this called ?**
    - Resting Heart Rate (Pulse)
  - **What rate is too fast?**
    - Tachycardia
    - >100bpm
  - **What rate is too slow ?**
    - Bradycardia
    - <60bpm

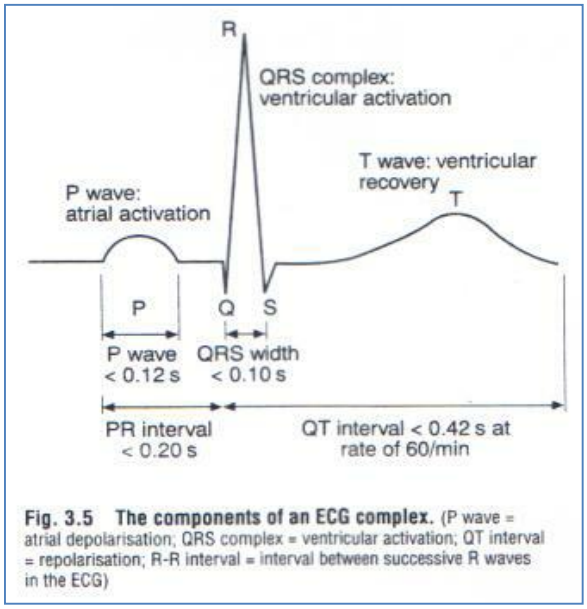
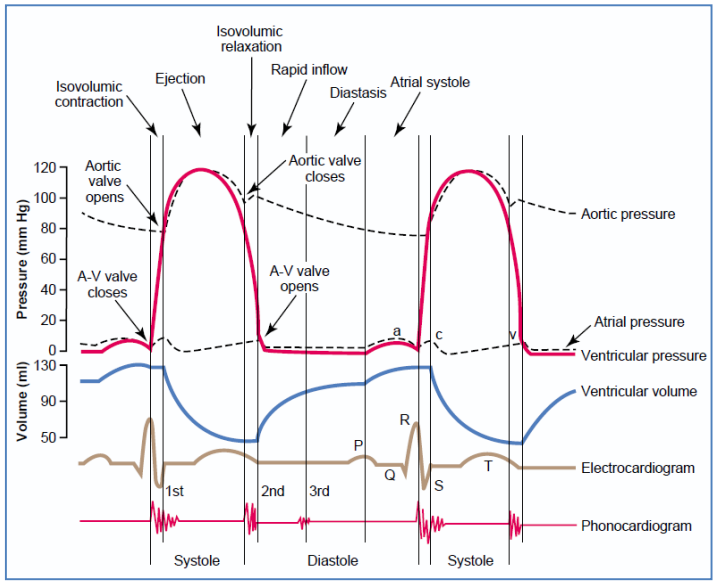


Fig. 3.5 The components of an ECG complex. (P wave = atrial depolarisation; QRS complex = ventricular activation; QT interval = repolarisation; R-R interval = interval between successive R waves in the ECG)

- **Revision of the Cardiac Cycle:**
  - **P-Wave?**
    - Atrial Systole
  - **QRS-Complex?**
    - Beginning of Ventricular Systole
  - **T-Wave?**
    - Ventricular Filling
    - Isovolumetric Relaxation
  - **S-Wave?**
    - Isovolumetric Contraction
  - **First & Second Heart Sounds:**
    - 1. Lubb (AV Valve Closure)
    - 2. Dupp (Semilunar Valve Closure)
    - **How do they relate to:**
      - **The cardiac cycle**
        - 1) Beginning of Ventricular Systole
        - 2) End of Ventricular Systole
      - **The ECG ?**
        - 1) QRS Complex
        - 2) T-Wave
  - **When blood enters the right atrium from the vena cava, Is this diastole or systole ?**
    - Atrial Diastole
    - Ventricular Diastole
  - **Which valves are open and closed?**
    - AV Valves are Open
    - Semilunar Valves are Closed
  - **How does the atria help the ventricle finish filling ?**
    - The atria contract following passive ventricular filling, actively pumping their contents into the Ventricles
    - (and how is this seen on the ECG) – P-Wave.



- **Once blood leaves the ventricles, is this diastole or systole?**
  - Atrial Systole
  - Ventricular Systole
- **Which valves are open and closed?**
  - AV Valves Closed
  - Semilunar Valves Open
- **how does the ventricle empty ?**
  - Via Active Contraction
  - (how is this seen on the ECG) – QT-Segment

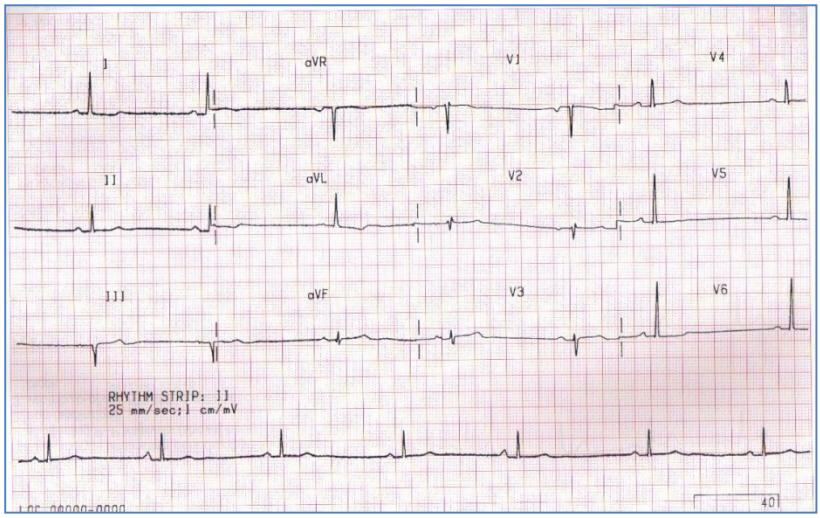
LOOKING AT ECGs:

- **Check Pt ID**
- **Check Voltage & timing**
  - 25mm/sec
  - 1large square = 0.2s (1/5sec)
  - 1small square = 0.04s
- **What is the rate?**
  - 300/number of large squares between QRS Complexes
    - Tachycardia
      - >100bpm
    - Bradycardia
      - <60bpm
- **What is the Rhythm?**
  - Sinus? (are there P-Waves before each QRS complex)
  - If Not Sinus?
    - Is it regular
    - Irregular?
    - Irregularly Irregular (AF)
    - Brady/Tachy
- **Atrial Fibrillation:**
  - Irregularly Irregular
  - P-Waves @ 300/min
- **QRS:**
  - Is there one QRS for each Pwave?
  - Long PR Interval? (1st degree heart block)
  - Missed Beats? (Second degree block)
  - No relationship? Complete heart block
- **Look for QRS Complexes:**
  - How wide – should be < 3 squares
  - If wide – It is most likely Ventricular
  - (Sometimes atrial with aberrant conduction (LBBB/RBBB)
  - IF Tachycardia, & Wide Complex → VT is most likely. (If hypotensive → Shock; if Normotensive → IV Drugs)
- **Look for TWaves:**
  - Upright or Inverted
- **Look at ST-Segment**
  - Raised, depressed or inverted
  - ST Distribution → Tells you which of the coronaries are blocked/damaged
    - Inferior ischaemia (II, III, AVF)
    - Lateral ischaemia (I, II, AVL, V5, V6)
    - Anterior ischaemia (V, leads 2-6)
  - NB: Normal ECG Doesn't exclude infarct.
  - ST Depression → Ischaemia
  - ST Elevation → Infarction
  - If LBBB or Paced, you CANNOT comment on ST-Segment

GLS - ECG CASES:

ECG 1

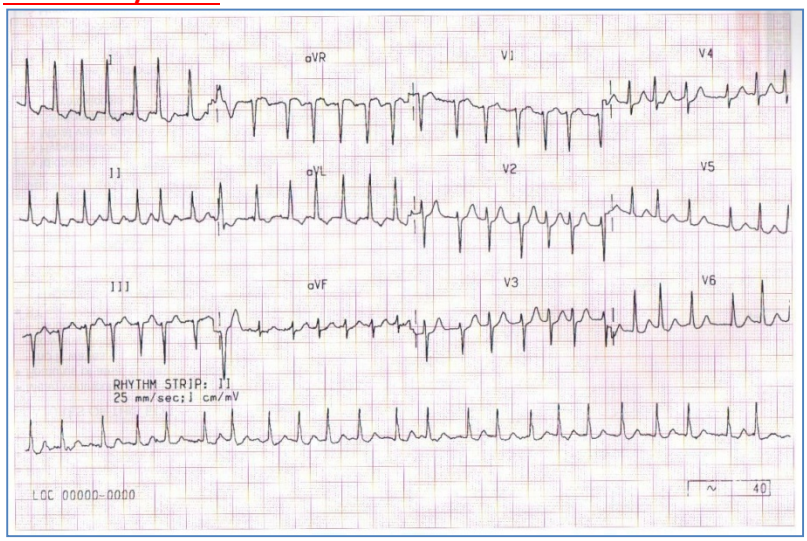
Sinus rhythm



- *P-Wave is Present*
- *Consistent 1:1 P-Wave:QRS-Complex Ratio*
- *QSR Complex is Normal*
- *Normal ECG (Sinus Rhythm)*
- CO is Normal
- (35yr old Male Triathlete)

ECG 2

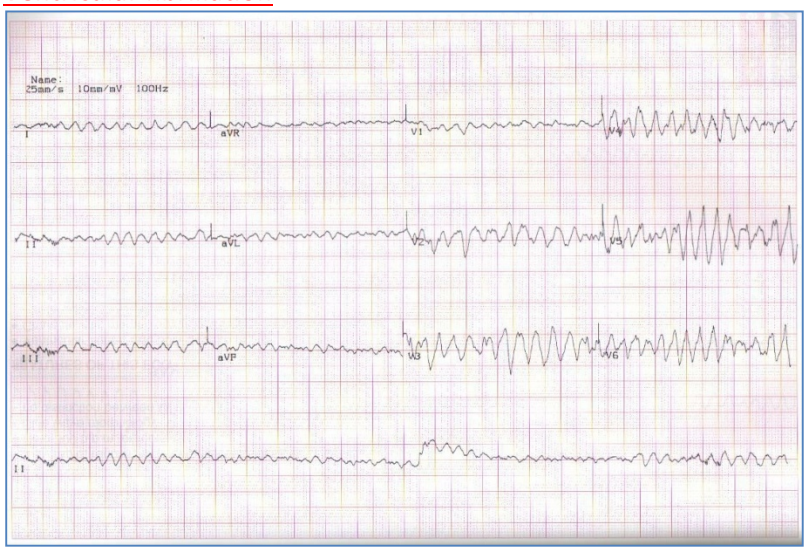
Sinus Tachycardia



- P-Wave is Present
- 1:1 P:QRS Ratio
- Sinus Tachycardia
- → ↑CO
- (Elderly Female with palpitations)

ECG 3

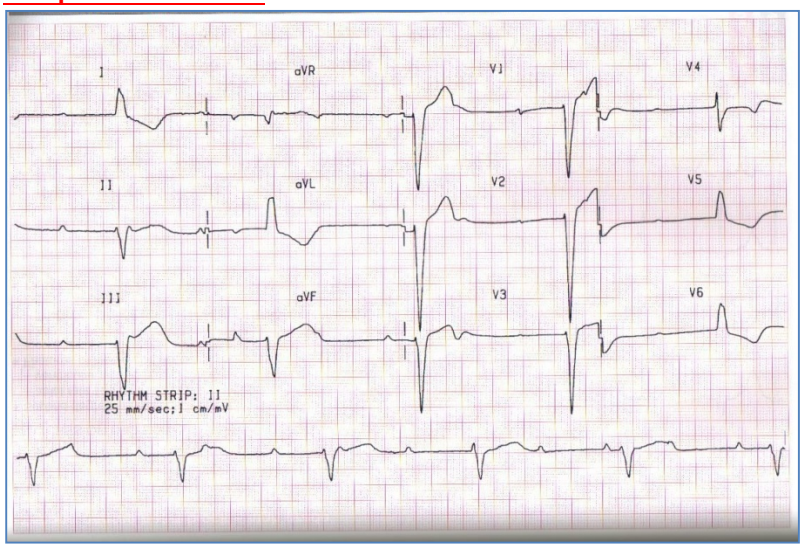
Ventricular fibrillation



- No Visible P-Wave
- No P:QRS Relationship
- No Recognisable QRS Complexes.
- Ventricular Fibrillation
- No Cardiac Output
- (61 yo male collapsed at the office, now unconscious with no pulse)

ECG 4

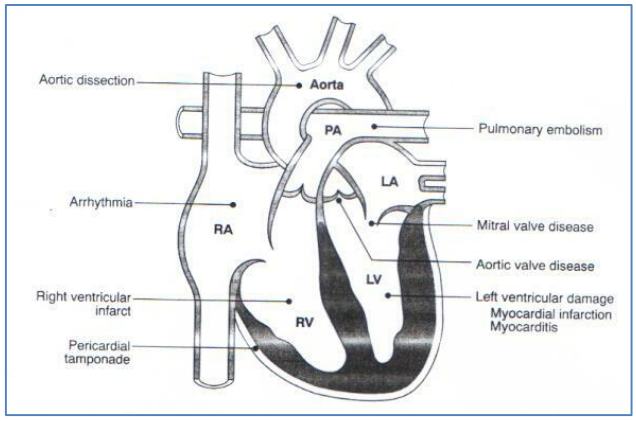
Complete heart block

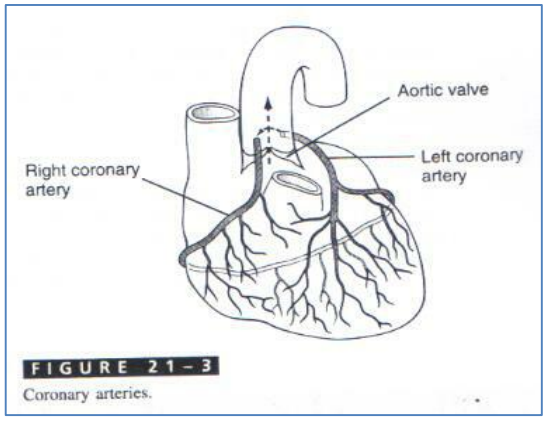


- P-Wave Present
- No P:QRS Relationship
- Inverted QRS Complexes
- ST-Depression
- Complete Heart Block (Complete conduction failure between Atria & Ventricles)
- Cardiac Output will be Reduced (due to disordered contraction of Atria & Ventricles)
- (76 yo female with recurrent dizzy episodes, collapsed twice)

CV EMERGENCIES: 3 – DISTURBANCES OF FUNCTION AND FORM
(*Myocardial Ischaemia/Heart Failure/Tamponade/Valve Dysfunction/Aortic Dissection*)

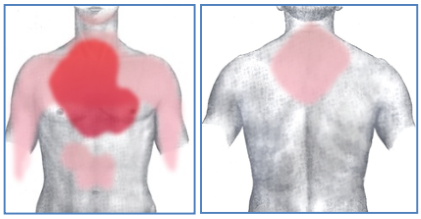
- Often associated with changes in Cardiac Output/Rhythm.
- Knowledge of anatomy of the heart needed to understand effects and treatment





MYOCARDIAL ISCHAEMIA

- **Risk factors**
  - Hypertension
  - Hyperlipidaemia
  - High cholesterol
  - Smoking
  - Obesity
  - Diabetes
  - Age
  - Systemic Inflammatory State
  - – However, in an ED setting, these are of little use.
- **Acute ischaemia**
  - **Pathogenesis** - Atherosclerosis
  - **Symptoms:**
    - Typically Crushing Chest Pain
    - Nausea, Vomiting
    - Dyspnoea
    - Radiating pain to Shoulders, Neck & Arm.



  - **Complications** → Infarction → Death of muscle.
- **Myocardial Infarction:**
  - **Pathogenesis –** Atherosclerosis (& Thrombosis)
  - **Symptoms:**
    - Arrhythmias (Palpitations)
    - Syncope
    - Crushing chest pain
    - Dyspnoea (Shortness of Breath)
    - Nausea, Vomiting
    - Sweating
    - Possible shock

- **Examination & Diagnosis:**
  - **Examination:**
    - Weak, Thready (Irregular) Pulse
    - Cool, Pale, sweaty skin (Peripheral Vasoconstriction)
    - Tachypnoea
    - If → Heart Failure:
      - → Raised JVP (Jugular Venous Pressure)
      - → Oedema (incl. Pulmonary Oedema)
  - **ECG:**
    - Arrhythmias
    - Changes in serial ECG traces.
      - Eg. ST-Elevation
      - Eg. ST-Depression
      - Eg. Pathological Q waves
  - **Imaging:**
    - Obstruction on Coronary Angiogram
  - **Cardiac Enzymes:**
    - Raised Troponin I & T Levels
    - (Also Raised Creatine Kinase-MB)
    - (NB: Rise and fall of serum cardiac biomarkers (Troponins & CKMB) indicate cardiomyopathy – Typically Infarction)
    - Therefore, Cardiac Markers have to be measured at different times.
      - 1. Immediately upon presentation
      - 2. 4-6hrs after attack (This is when cardiac troponins are released if it's an MI)
      - 3. Over the next 24hrs
- **Treatment:**
  - Nitrates (Vasodilators)
  - Thrombolytics
  - Surgery (Angioplasty/Stent)

CV EMERGENCIES: 3 – DISTURBANCES OF FUNCTION AND FORM
(*Myocardial Ischaemia/Heart Failure/Tamponade/Valve Dysfunction/Aortic Dissection*)

HEART FAILURE

- **= Where the heart cannot maintain adequate CO to perfuse the organs of the body.**
  - (or can only do so at expense of ↑ filling pressure → Frank Starling Law → ↑SV)
  - (Frank Starlings Law = ↑Preload → ↑Contractility → ↑SV → ↑CO.)

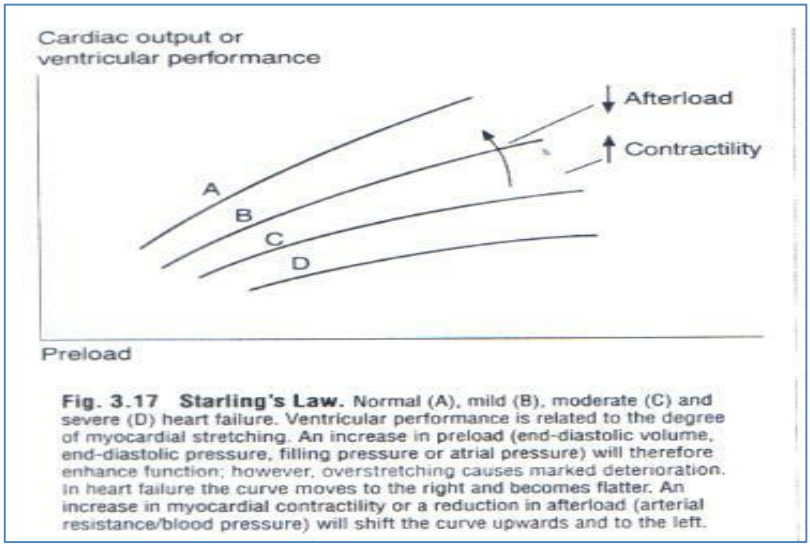


Fig. 3.17 Starling's Law. Normal (A), mild (B), moderate (C) and severe (D) heart failure. Ventricular performance is related to the degree of myocardial stretching. An increase in preload (end-diastolic volume, end-diastolic pressure, filling pressure or atrial pressure) will therefore enhance function; however, overstretching causes marked deterioration. In heart failure the curve moves to the right and becomes flatter. An increase in myocardial contractility or a reduction in afterload (arterial resistance/blood pressure) will shift the curve upwards and to the left.

- **Body's Response to HEART FAILURE**
  - **→Activation of Renin Angiotensin System →**
    - ↑Vasoconstriction → ↑PVR → ↑BP → ↑*Organ Perfusion*
    - Aldosterone Secretion → Salt & Water Retention → ↑Preload → ↑SV → ↑CO
  - **→Activation of Sympathetic System →**
    - ↑Vasoconstriction → ↑PVR → ↑BP → ↑*Organ Perfusion*
    - ↑HR → ↑CO→ ↑*Organ Perfusion*
    - ↑Contractility → ↑SV → ↑CO → ↑*Organ Perfusion*
  - Initially helps cardiac function by altering afterload or preload and ↑ myocardial contractility
  - Ultimately causes ↓CO by excess peripheral vascular resistance

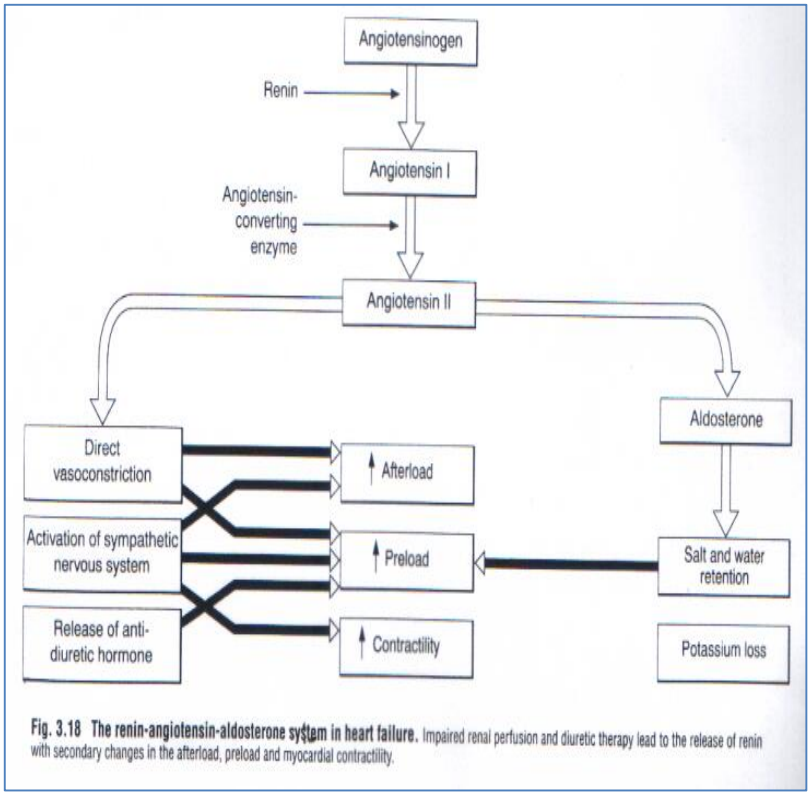


Fig. 3.18 The renin-angiotensin-aldosterone system in heart failure. Impaired renal perfusion and diuretic therapy lead to the release of renin with secondary changes in the afterload, preload and myocardial contractility.

CV EMERGENCIES: 3 – DISTURBANCES OF FUNCTION AND FORM
(*Myocardial Ischaemia/Heart Failure/Tamponade/Valve Dysfunction/Aortic Dissection*)

TAMPONADE

- **Cardiac Tamponade = "*Emergency condition where Fluid Accumulates in the Pericardium*".**
  - **AKA: "Pericardial Effusion"**
  - (NB: The Pericardium is Fibrous, & Doesn't stretch→As little as 100mL can cause tamponade)
- **Effects on CV System:**
  - Puts pressure on the heart → Prevents proper Ventricular Filling → ↓SV, CO
  - → Obstructive Shock
  - → Cardiac Arrest
- **Possible Causes:**
  - Physical trauma
  - Hypothyroidism
  - Pericarditis
  - Iatrogenic Trauma (Eg. From surgery)
  - Myocardial Rupture (Typically of an Infarcted Area following MI)
- **Treatment:**
  - Pericardiocentesis (Chest tube to drain fluid)

VALVE DYSFUNCTION

- **Eg. Aortic Valve Stenosis:**
  - **= *"Abnormal narrowing of the Aortic Valve opening."***
    - → Blood cannot be adequately pumped through the narrow orifice.
  - **Effects on CV System:**
    - → ↑L-Ventricular Afterload → LV-Hypertrophy (Initial compensation – Maladaptive)
    - → Eventually, LV-Dilation occurs due to ↑Wall Stress → Systolic Function Deteriorates.
      - → Congestive Heart Failure/Syncope/Angina
  - **Treatment:**
    - Valve Replacement
- **Eg. Mitral Regurgitation:**
  - ***= "Where the mitral valve doesn't close properly during Systole"***
    - → Regurgitation of blood from left ventricle into the left atrium.
    - **AKA: Mitral Insufficiency** or **Mitral Incompetence**
  - **Effects on CV System:**
    - → Regurgitation of blood from left ventricle into the left atrium.
    - →↓Ejection Fraction & SV → ↓CO
    - →Congestive Heart Failure (Dyspnoea, Pul.Oedema, Dyspnoea lying down)
    - → Cardiovascular Collapse → Cardiogenic Shock
  - **Treatment:**
    - Mitral Valve Replacement (If acute)
    - Vasodilators & Antihypertensives to ↓Afterload & ↑Ejection Fraction (If Chronic)

AORTIC DISSECTION

- **= *"A tear in the wall of the Aorta where Blood flows Between the Layers of the Wall"***
  - → Forces the layers of the wall apart.
  - → If Aorta Ruptures – 80% Mortality Rate
  - (An *Absolute* Medical Emergency)
- **Treatment:**
  - Emergency Surgery

ASSESSMENT OF CVS EMERGENCIES

- **AIMS:**
  - Determine Cause
  - Determine Severity
- **METHODS**
  - **1. ABC : 30 second exam**
  - **2. Stabilise**
  - **3. In Depth:**
    - Appearance
    - History
    - Examination
    - Monitoring (ECG, Sats, Vitals)
    - Investigation

APPEARANCE

- Level of consciousness
- Sweating
- Agitation
- Cyanosis or Pallor
- External blood loss
- Clutching chest, obvious bleeding or other clues to cause

HISTORY

- Nature of symptoms
- Onset of symptoms
- Progression of symptoms
- Associated symptoms
- Treatment so far
- Previous episodes
- Other significant past history

EXAMINATION

- Appearance
- Pulse
- Capillary Refill Time
- Blood Pressure
- Heart Sounds
- Evidence of heart failure – JVP, oedema, creps
- Signs of chronic disease

PULSE

- Rate
- Rhythm
- Volume
- Location

CAPILLARY REFILL TIME

- Peripherally - Press down on nail bed for 5 seconds and then release.
  - Watch for return of normal colour
  - Normal is less than 2 seconds
  - Abnormal is 2 seconds or greater
- Centrally – same thing over the sternum
- What may potentially interfere with assessment of CRT?
  - (Cold ambient temperature → Peripheral Vasoconstriction to Conserve Heat)

BLOOD PRESSURE

- What is normal *for the patient*?
- What is low?
- What is raised?
- How and where to measure?

ASSESSMENT: MONITORING & INVESTIGATION:

- **MONITORING**
  - Direct CVS monitor
    - BP and Pules
    - ECG
  - Indirect CVS monitor
    - O2 sats
    - urine output (For kidney perfusion)
    - GCS (For brain perfusion)
- **INVESTIGATION**
  - ECG – (Detect Rhythm Abnormalities & Diagnose other Conditions)
  - Chest XR – (Detect CV Abnormalities and their effects such as Pulmonary Oedema)
  - Echocardiography
  - Pathology (Blood tests)

WHEN ASSESSMENT IS COMPLETE

- **SHOULD HAVE AN IDEA OF CAUSE**
  - Simple?: Shock, HT Crisis, Arrhythmia
  - Advanced?: Differential Diagnosis

- **SHOULD KNOW SEVERITY OF PROBLEM AND HENCE DEGREE OF URGENCY**

REMEMBER

- AS YOU IDENTIFY PROBLEMS IN THE PRIMARY SURVEY YOU NEED TO TREAT THEM

TREATMENT OF CVS EMERGENCIES:

- **SUPPORTIVE TREATMENT**
  - **AIM = To restore / maintain adequate tissue perfusion**
  - **Examples:**
    - Control of external bleeding
    - Correct circulating volume (Eg. IV fluids)
    - Help tissue oxygenation (Eg. With Supplemental O2)
    - Symptom relief (Eg. Nitroglycerin for Angina)
- **DEFINITIVE TREATMENT**
  - **AIM = Treating the underlying pathology**
    - (These are :. *diagnosis dependant*.)
  - **Examples**
    - Surgery to control bleeding in trauma (Eg. Ruptured Spleen)
    - Pacemaker for complete heart block (Pacing)
    - Restoration of coronary circulation in AMI (Eg. Thrombolytics/Angioplasty)
    - Long term Antihypertensives (Eg. Daily Antihypertensive Meds)

SUPPORTIVE TREATMENTS:

- **INTRAVENOUS CANNULATION**
  - Various sizes/sites/uses

- **CENTRAL VENOUS ACCESS**
  - Where do these go? (Subclavian, Juggular, Femoral)
  - What advantages or disadvantages do they have ?
    - + Big Vessels – irritating drugs can be administered
    - - Time/Expertise required
  - When would you use them?
    - Very ill patients in ICU

- **OTHER ACCESS SITES**
  - Intraosseus (With intraosseus needle – Can give fluid & drugs like normal IVs)
  - Venous Cutdowns (Cannula is inserted into surgically-exposed vein under direct vision)
  - Umbilical Veins

- **INTRAVENOUS FLUIDS**
  - Different types of fluids – what does this mean when choosing ?
    - Crystalloids (Eg. Electrolytes)
    - Colloids (Eg. Proteins)
    - Blood & Blood Products

- **MEDICATIONS**
  - Inotropic agents
  - Chronotropic agents
  - Rate control (eg. In Tachyarryhthmia/eg. Pacing in Heart Block)
  - Diuretics (Eg. If pulmonary oedema)
  - Oxygen

BASIC LIFE SUPPORT

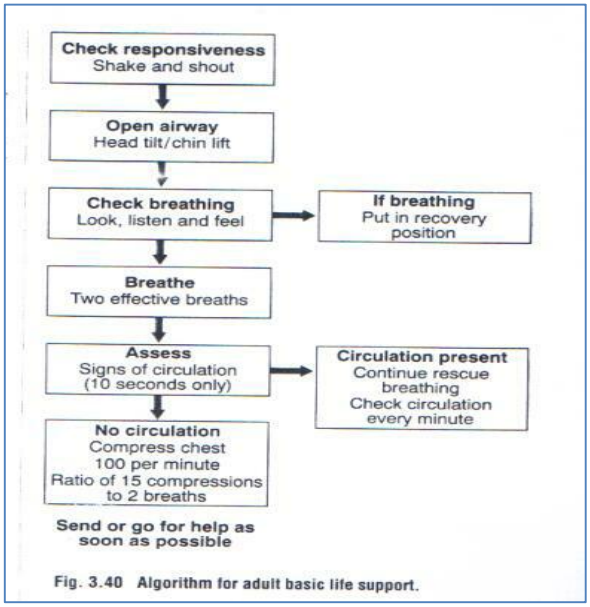


Fig. 3.40 Algorithm for adult basic life support.

ADVANCED LIFE SUPPORT

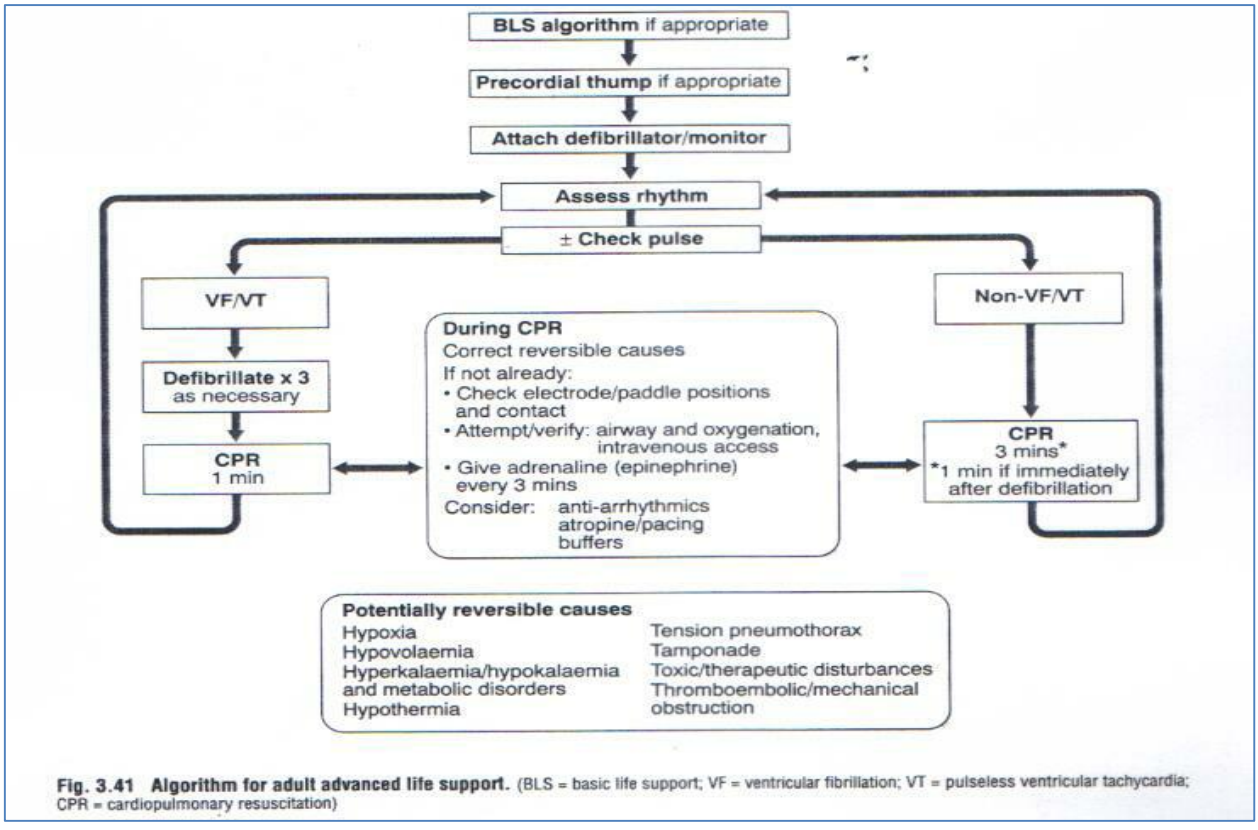


Fig. 3.41 Algorithm for adult advanced life support. (BLS = basic life support; VF = ventricular fibrillation; VT = pulseless ventricular tachycardia; CPR = cardiopulmonary resuscitation)

Emergency Medicine Notes
Loss of Consciousness/ALOC (Altered Level of Consciousness)

Causes of Loss of Consciousness:

- **Common Causes:**
  - Trauma
  - Drugs
  - Post-ictal
  - Hypoglycaemia
  - SAH (Sub-arachnoid Haemorrhage)
  - Systemic
- **Uncommon Causes:**
  - Infection
  - Endocrine
  - Metabolic
  - Tumour
  - Vascular
  - Paralysis
  - Hypothermia

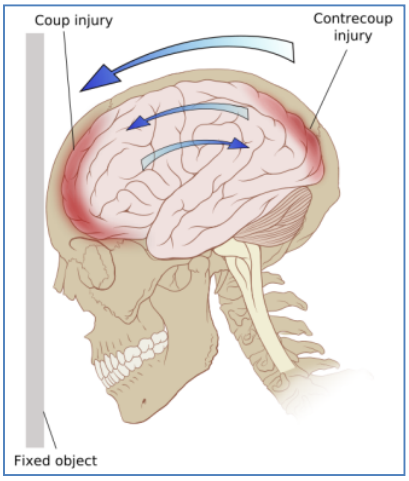
Head Trauma → Loss of Consciousness:

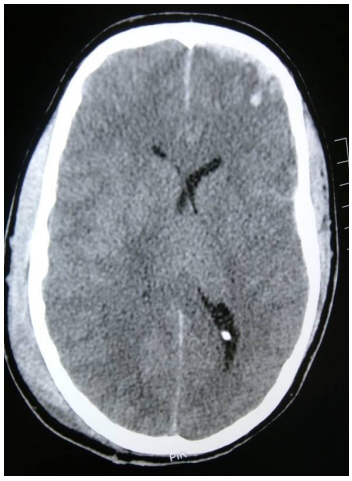
- **Primary Injury:**
  - Concussion
  - Contusion
  - Laceration
  - Diffuse Axonal Injury
  - **(NB: Primary injury determines the best possible outcome)**
- **Secondary Injury:**
  - Hypoxia
  - Hypoglycaemia
  - Decreased cerebral perfusion
    - Hypotension
    - Haemorrhage
    - Oedema
  - **(Secondary Injury determines the *Actual Outcome*)**
  - **(All treatments are focussed on preventing secondary injury)**

HEAD TRAUMA → ALOC:

- **Concussion:**
  - No structural damage
  - Usually only a brief Loss of Consciousness
  - Likely to fully recover (unless secondary injury)

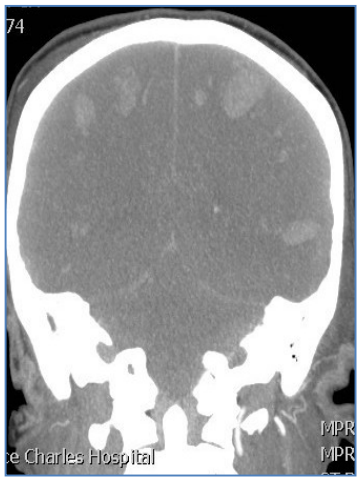
- **Contusion:**
  - Bruising, swelling, bleeding of the Brain Following Trauma.
  - **(Often a "Contre-Coup injury" =** Brain Injury on the Opposite Side of Impact – Due to Rebound of the Brain**)**
    - **(NB: "Coup Injuries" =** Brain Injury on the Side of Impact)
  - Some damage
  - Expect a reasonable recovery (but decreased memory, concentration; but still retain normal function)





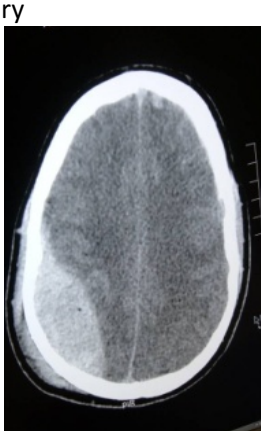
- **Laceration:**
  - An incised wound of brain tissue (Eg. Bullet/knife/etc)
  - Usually SEVERE damage
  - Typically a Poor Prognosis.

- **Diffuse Axonal Injury:**
  - High Energy Injury (Grey matter of whole areas of the brain have been sheared right off)
  - CT may appear normal initially (but by day 3, they have a non-functional brain)
  - GCS 3
  - → Brain Damage → Organ donor

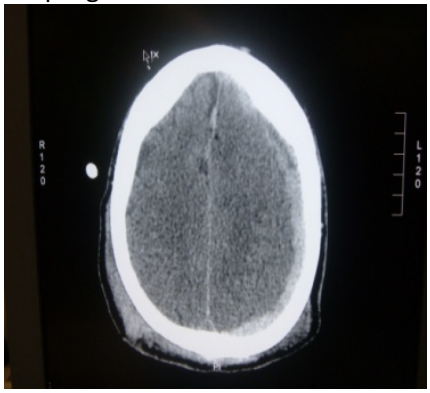


VASCULAR CAUSES OF ALOC:

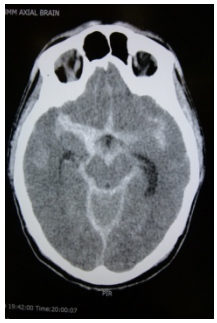
- **Ischaemic Stroke:**
  - Focal Ischaemia of the Brain caused by Emboli/Bleeding.
  - →Muscle weakness associated with severe headache
- **Or Haemorrhage:**
  - (Typically cause secondary injury → Increases ICP → Pressure on the brain → Hypoxia)
  - **Intracranial (Extradural) Haemorrhage:**
    - **Caused by:**
      - Typically a cranial fracture which cuts an **artery**
      - Or Blood vessel bursts spontaneously
    - **Leads to:**
      - → Dissects the dura from the skull → ↑Intracranial Pressure
      - →Sudden collapse with a severe headache, vomiting and altered consciousness.
      - (NB: Normal brain underneath)
    - **Imaging:**
      - Limited by attached dura
      - Clearly defined margin
    - **Rapid progression** (due to arterial source of blood)
    - **Good prognosis with surgery** (Drill a hole in the head to relieve the pressure)
      - Die without surgery



  - **Subdural Haematoma** (typically a venous bleed; happens slowly)
    - **Caused by:**
      - Mostly Old people with Low force trauma → Slow Venous Bleed
        - Because their brain is shrunken
        - Brain moves significantly under mild trauma
    - **Gradual onset** (:. Is often Chronic)
    - **Wide distribution** – ie. Over the entire hemisphere
    - **Presentation:**
      - Unsteady gait
      - Other abnormalities
    - **Abnormal Brain underneath** (Ie. Pt. Is often demented, or alcoholic, or have severe cerebral atrophy)
      - Therefore the prognosis is worse



- **Sub-Arachnoid Haemorrhage**
  - **Caused by:**
    - Often due to aneurysm of circle of willis
    - (Arterial Bleed)
  - **Presentation:**
    - Typically a fit, well person who has sudden onset, severe headache (or feel like they've been hit in the head with a baseball bat)
    - Loss of Consciousness.



OTHERS CAUSES OF ALOC:

Tumours:
- Rare
- Benign or Malignant
- May be metastasies

Systemic Causes of LOC:
- **Hypoxia**
- **Hypoventilation**
- **Hypotension**
- **Sepsis**

Endocrine Causes:
- **Diabetes**
  - **NB: In EVERY case of LOC, you MUST CHECK BLOOD SUGAR (despite obvious trauma/stroke/etc.)**
  - Hypoglycaemia
  - Hyperglycaemia/ketoacidosis
- **Hypothyroidism/Thyrotoxicosis**

Metabolic Causes:
- **Uraemia** (due to acute renal failure) → Requires renal transplant/dialysis.
- **Hepatic Failure** (eg. Alcoholic liver disease → Acute liver failure)

Infection:
- **Meningitis – Diagnosis is ESSENTIAL**
  - If pt. Has altered consciousness and fever, assume meningitis
  - → Give antibiotics unless proven otherwise
- **Encephalitis**
  - If pt. Has Altered consciousness and fever, can give antivirals
- **Tuberculosis**
  - Acute altered consciousness in immigrants.
- **AIDs**
  - Eg. Opportunistic infections
  - Or. Direct encephalitis by the HIV Virus
- **Malaria**
  - Mostly in immigrants
  - If been overseas, and come back with fever and altered consciousness → Suspect Malaria.

Drugs:

- **Prescription Medication:**
    - (Ie. Taken in accidental overdose – Esp. Old people/suicide)
    - (Or. Drug Interaction)
    - Analgesics
    - Sedatives
    - Antidepressants
- **Recreational Drugs:**
    - Alcohol
    - Opiates
    - Etc.
- **Treatment:**
    - Treat Specific OD
    - Assess Suicide Risk
    - Psych Review

Paralysis (Very rare):

- **Eg. Trauma/C-Spine Injury**
- **Eg. Snake Bite**
- →Pt is Unresponsive, may be Apnoeic.

ASSESSMENT OF LOC:

- **Glasgow Coma Scale (GCS) - KNOW THIS:**
  - **Eyes opening in response to:**
    - 4. Spontaneous
    - 3. Voice
    - 2. Pain
    - 1. Not
    - (Score out of 4)
  - **Speech:**
    - 5. Oriented
    - 4. Confused Speech
    - 3. Inappropriate Speech
    - 2. Incomprehensible/Incoherent
    - 1. None
    - (score out of 5)
  - **Motor**
    - Can either -Obeying commands/localising painful stimulus (directly respond to painful stimulus)/ withdraws from painful stimulus/flexing (Very bad)/Extension (very very Bad)/None
    - (Score out of 6)
  - **(Less than GCS10 = Deeply unconscious; Less than GCS8 = Coma)**
  - **(Max Score = 15 = Awake, Alert, Responsive = Conscious)**
  - **(Min Score = 3 = Dead)**

GLASCOW COMA SCALE		
EYE OPENING	Spontaneous	4
	To Voice	3
	To Pain	2
	None	1
VERBAL RESPONSE	Oriented	5
	Confused	4
	Inappropriate Words	3
	Incomprehensive Words	2
	None	1
MOTOR RESPONSE	Obeys Commands	6
	Localizes Pain	5
	Withdraws (Pain)	4
	Flexion (Pain)	3
	Extension (Pain)	2
	None	1

- **AVPU**
  - **Conscious**
    - **A**wake
    - *responding to* **V**oice
  - **Unconscious**
    - Only Responsive to **P**ain
    - **U**nresponsive

Table II: Example of AVPU Assessment Tool

A=Alert	Patient is alert and con scious
V=Verbal	Patient responds to verbal stimulus
P=Pain	Patient responds to painful stimulus
U=Unresponsive	Patient is unresponsive to any form of stimulus

PRIMARY SURVEY (ABSOLUTELY CRITICAL IN CASES OF LOC):

- **ABCDEFG:**
  - **Airway:**
    - **Commonest cause of Unconsciousness is Airway Obstruction**
    - **Clear The Airway:**
      - Left Lateral Position/Suction/Guedell Airway/Jaw Thrust
    - **(Generally GCS<8 require definitive airway management – Endotracheal tube/Intubation →Ventilation)**
  - **Breathing:**
    - Essential to ensure adequate ventilation
    - (without adequate ventilation, pt will suffer a secondary hypoxic brain injury)
    - **Give Oxygen**
    - **Maintain Sats & Normocarbia**
      - If hypoventilation → Hypercarbia → Cerebral Vasodilation → Increases Intracranial Pressure → Decreases Cerebral Perfusion.
  - **Circulation:**
    - Must ensure adequate circulation (IV fluids)
    - Cerebral perfusion pressure = Blood Pressure – Intracranial Pressure
      - As ICP goes up, must ensure at least a normal BP in order to maintain cerebral circulation
    - In head trauma, 5 minutes of Hypotension → 25% increase in mortality.
  - **Disability (Ie. Level of Consciousness):**
    - Assess level of consciousness & Document it.
    - Monitor for changes
    - Assess Pupillary Size/Reactivity
  - **Exposure:**
    - Examine the Whole Patient (Including the Back)
  - **DON"T EVER FORGET GLUCOSE**

History:

- Past history
- Medication, drug use
- Collateral History
- Trauma?
- Overseas Travel?

Examination:

- Primary Survey
- Look for Trauma
- Look for needle marks (Drug use)
- Signs of Infection (Fever, neck stiffness, focal neurology?)
- Respiratory Effort

Investigation:

- Check Glucose
- Urine & Elecrolytes
- FBC
- Head CT

Disposition:

- **2 groups of unconscious patients:**
  - **Active management (ICU):**
    - Vast majority of patients
    - Aggressive treatments
  - **Palliative Care:**
    - → ward or back to nursing home.

Specific Treatment:

- **Depends entirely on cause**
- **All receive Supportive Care:**
    - O2
    - Iv fluids
    - Glucose
    - Antibiotics
    - (All of these apply *even* if you don't know what's wrong)
- **Rarely surgery**
- **Rarely Drugs-Antidotes:**
    - Eg. Naxolone/Narcan (Opioid Antagonist – Reverses Respiratory Depression)

KEYPAD SESSION:

LOSS OF CONSCIOUSNESS QUESTIONS

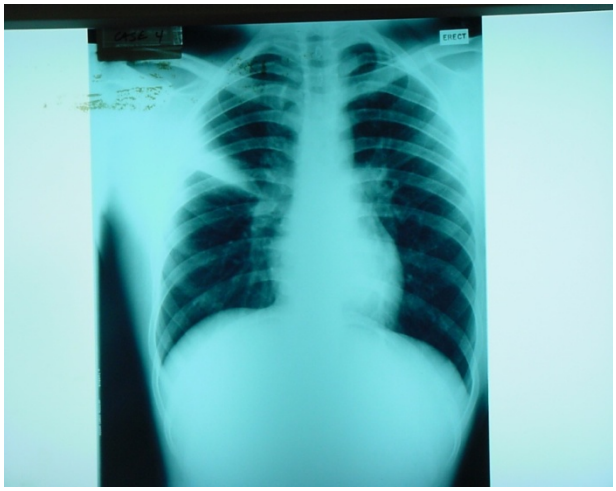
1. Adult female, overdose, eyes ope ot voice, withdraws from pain & swears at you. GCS?
    a. 10
2. Which is an uncommon cause of altered consciousness:
    a. Hypoglycaemia
    b. Trauma
    c. Drugs
    d. Sepsis
    e. Meningitis – (Usually only cases headache, nausea, vomiting, neck stiffness; but not altered consciousness)
3. Which is a cause of primary brain injury (Cf Secondary Brain Injury):
    a. Hypovolaemia
    b. Hypoglycaemia
    c. Seizures
    d. Diffuse Axonal Injury – (Caused when the brain hits the skull)
4. A Contre-coup injury is related to which of the following?
    a. Concussion
    b. Contusion (Contre-Coup = A contusion opposite to the site of injury)
    c. Laceration
    d. Diffuse Axonal Injury
5. Subdural Haematoma – Which is true?
    a. Associated with good outcome following surgery
    b. Associated with arterial bleeding
    c. Is often chronic
    d. Mostly affects young people with severe trauma.
6. Extradural Haematoma – Which is True?
    a. Associated with arterial bleeding
    b. Is usually slow in onset
    c. Has a poor prognosis after surger
    d. Is associated with alcoholism
7. Which is true for Diffuse Axonal Injury?
    a. Has a good prognosis
    b. Is associated with high energy injury
    c. Is usually obvious on initial CT scan
    d. Is caused by shearing between the brain stem and spinal cord.
8. Which is a *systemic* cause of altered consciousness?
    a. Septic Shock
    b. Encephalitis (Local)
    c. Brain Metastases (Local)
    d. Hypertensive Crisis
9. Which of the following drugs typically cause respiratory depression in overdose?
    a. Opiates
    b. Amphetamines
    c. Paracetamol
    d. Beta Blockers
10. Which antidote is commonly used for unconscious patiens?
    a. NAC (N Acetyl Cysteine – for Paracetammol Overdose)
    b. Flumazenil (Benzo Antidote)
    c. Pralidoxime (For Overdose of Organophosphate poisoning)
    d. Naloxone (An opioid Antagonist)

11. Which is true for an unconscious patient presenting to ED:
    a. The airway is usually patent on arrival
    b. Cervical spine precautions are unnecessary unless here is a clear history of trauma
    c. The glucose should always be measured immediately
    d. Paralysis is never a cause of unconsciousness
12. Which of the following is true for eth Glasgow Coma Scalew:
    a. The lowest score possible is zero (Lowest is 3)
    b. A GCS of less than 8 roughly equates with coma
    c. A GCS of more than 13 is consistent with a moderate head injury (*Less than 13 is moderate head injury*)
    d. Response to pain is usually tested peripherally
13. What GCS score is incoherent speech?
    a. 0 (No such thing)
    b. 1 (No speech)
    c. 2 (Incoherent)
    d. 3 (Inappropriate)
    e. 4 (Confused)
    f. 5 (Normal)
14. What score does a motor response of abnormal extension get?
    a. 0 (No such thing)
    b. 1
    c. 2
    d. 3
    e. 4
15. What score does a motor response "Obeying commands" get?
    a. 2
    b. 3
    c. 4
    d. 5
    e. 6
16. On the AVPU scale:
    a. A corresponds to Agitated
    b. V Equates to Veralising
    c. P Equates roughly to unconscious
    d. U Means Unresponsive to Voice
17. Which best applies to airway management in an unconscious patient?
    a. The LMA (Laryngeal Mask) is a suitable definitive airway (A "Definitive Airway" = an inflated tube *Within* the airway)
    b. Head tilt, chin lift is the most effective way to clear the airway (Not as effective as a jaw thrust)
    c. A Guedel Airway reliable ensures a clear airway
    d. If possible, Endotracheal Intubation is the best way of ensuring a clear airway.
18. Which applies to Hypercarbia?
    a. Associated with cerebral vasodilation & increased intracranial pressure
    b. Associated with hyperventilation
    c. Causes peripheral cvasoconstriction
    d. Associated with decreased cardiac output.
19. Which is true for ventilation of an unconscious patient:
    a. All unconscious patients require assisted ventilation
    b. Ventilation is perforemed in order to produce hypercarbia
    c. Deliberate slight hyperventilation is possibly beneficial – (Useful in short-term, not long term → ↓CO2)
    d. Ventilation using an LMA is usually inadequate

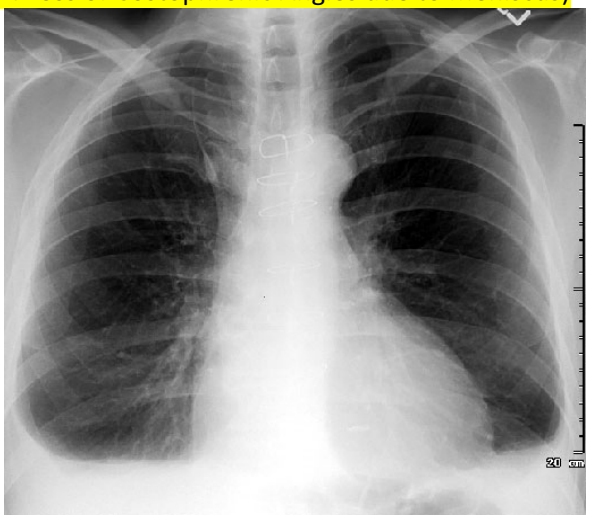
20. Which is true for IV therapy in an unconscious patient?
    a. Aggressive IV resuscitation should be avoided as it causes cerebral oedema
    b. Even brief hypotension is associated with an increase in mortality in head trauma
    c. IV glucose should always be given early (Only given if sugar is low)
    d. Permissive hypotension may be beneficial in trauma

CHEST XRAY QUESTIONS:

21. Which structure contributes to the Right heart border on a CXR?
    a. Right ventricle
    b. Aortic knuckle
    c. IVC
    d. Diaphragm
22. What's the best way to differentiate between AP and PA?
    a. Look for position of the scapulae (If retracted, it's PA)
    b. Look for widening of teh mediastinum
    c. Look for Kerley B Lines
    d. Look for ECG leads
23. What abnormality is present on this CXR?
    a. Left upper lobe pneumonia
    b. Cardiomegaly
    c. Right upper lobe pneumonia
    d. Pulmonary embolus



24. What abnormality does this CXR show?
    a. Widening of the mediastinum
    b. Pneumothorax
    c. Pneumoniwa
    d. Pleural effusions (NB: Loss of Costophrenic Angles due to Meniscus)



25. Which suggests a technically adequate film?
    a. Vertebral discs clearly through the mediastinum
    b. Clearly visible costo-phrenic angles
    c. Widening of the mediastinum
    d. Cervical spines well centred between the medial ends o the clavicles (Ensures no rotation)

Emergency Medicine Notes
Envenomation

Objectives

- **Understanding of basic terminology**
  - Poison = Ingested Toxins (Typically Chemicals)
  - Venom = Injected Peptides/Proteins by an Animal from a Gland where it's kept.
  - Toxin = Either Poisons or Venoms.
  - Envenomation = Being injected with a venom
  - Toxinology = study of envenomation
  - Toxicology = study of clinical effects of drugs (man made)
- **Envenomation by Critter**
  - Toxin
  - Signs, symptoms
  - Investigation
  - Management

Recognition & Assessment of Envenomation:

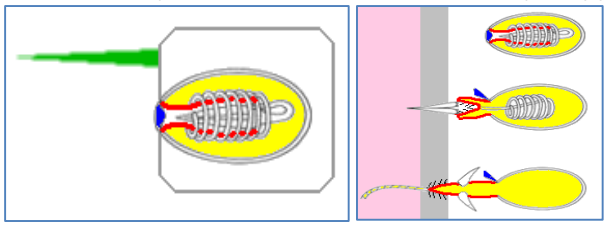
- **Clinical Assessment**
  - History (when? Where? How? What? Symptoms?)
  - Increased vigilence in children & with ALOC
  - Examination (signs)
  - Investigation - blood, urine, VDK, nematocysts
- **Syndromic approach to diagnosis**
- **Identification of animal**
  - description, morphology, scale counts
  - characteristics of sting site, nematocysts

Approach to the Envenomated Patient:

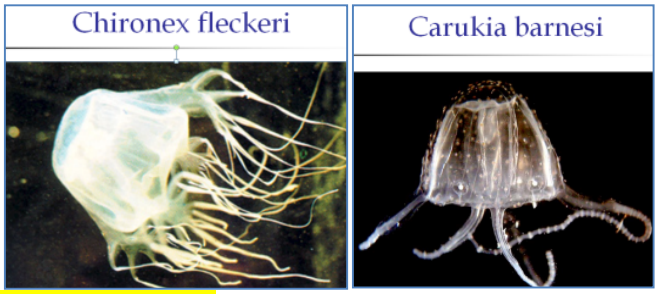
- First aid
- ABCDE (primary survey)
- Secondary survey
- Supportive care - analgesia, respiratory, cardiovascular, renal, metabolic.
- Definitive care – antivenoms, disposition

Marine Envenomation:

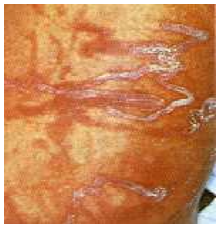
- **Cnidarians**
  - **Contain Stinging Organelles Called 'Cnidomes' or 'Nematocysts':**
    - Millions of Nematocysts line the surface of the body & appendages of cnidarians



  - **Notables:**



    - **Cubozoa - Box Jellyfish**
      - **Chironex Fleckeri – Multitentacled**
        - 2-30cm Diameter
        - Shallow Water
        - Rudimentary Senses (Vision, Balance, Vibration)
        - **Venom – Clinical Actions (Dose Dependent – body surface area):**
          - Severe Local Pain
          - Dermatonecrotic component - scarring
          - Haemotoxin - not significant in human stings
          - Lethal factor(s) - act by increasing ionic (Na+ & Ca++) conduction across nerve & muscle membranes- rapidly cardiotoxic
        - **Visual Appearance of Sting:**
          - Whip Wheals
          - Frosty Appearance
          - Ladder Pattern



        - **Management:**
          - Pre-hospital
            - Vinegar – inactivates the nematocysts that haven fired.
            - Anti-venom
          - Emergency
            - Supportive – (Analgesia/Oxygen/Fluids/Monitoring)
            - Anti-venom- (Only with evidence of envenomation – eg. Arrhythmias/10% of Body SA)
            - Analgaesia/tetanus

- **Carukia Barnesi (Irukandji) – 4 Tentacles:**
    - Small cube shaped 1.2 - 2.5cm diameter
    - 4 tentacles - 1 in each corner - retractile from 60 - 8cm, 1mm diam
    - Nematocysts on both bell & tentacles
    - Virtually transparent & rarely seen in the water (They may not have seen it)
    - **Venom – Clinical Actions:**
        - Delay to systemic Symptoms
        - Irukandji Syndrome
            - Severe *Generalised* Pain. Comes in Waves
            - Autonomic Dysfunction
                - Sweating
                - Hypertensive
                - Tachycardic
            - Cardiopulmonary decompensation
                - Bite → Massive Catecholamine Release → Cardiomyopathy → Heart Failure → Pulmonary Oedema.
    - **Management:**
        - Ice + vinegar
        - Supportive
        - Monitor – vitals and cardiac markers (Troponins)
        - Opiates
        - Nitrates
        - MgSO4 (magnesium)
        - (midazolam/promethazine/chlorpromazine/phentolamine)
- hydroids (bluebottles, fireweeds)
- hard & soft corals, anemones
- medusoid or blubber jellyfish

- **Molluscs**
    - poisonous shell fish
    - venomous cone shells

- **Venomous Fish Spines**
    - **Stonefish**
        - 13 dorsal fin spines
        - Each paired venom glands
        - Spines erect when fish trodden on and venom into wound
        - Neurotoxic, myotoxic, vascular and myocardial effects
        - Excruciating pain
        - **Treatment:**
            - Immerse in hot water (As hot as bearable – Max 43^{O}C)
            - Opioid analgesia – ?effective
            - Regional anaesthesia (Ankle Block)
            - Antivenom – if evidence of envenomation.
                - How much? 1 Vial/2-3 spines.
            - Debride the wound – (X-Ray to see remaining spines)
            - ADT
    - Stingrays
- **Venomous spiny starfish**
- **poisonous fish** - tetrodotoxin
- **Blue ring octopus** - tetrodotoxin
- **Sea snakes**

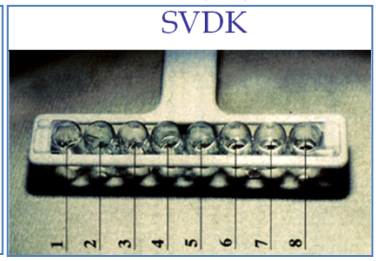
Terrestrial Envenomation

- **Terrestrial venomous animals:**
  - **Hymenoptera:**
    - Wasps
    - Bees
    - Ants
  - **Arachnida**
    - **Spiders:**
      - **Redbacks (latrodectus)** - single greatest cause of envenomation in Australia:
        - Found throughout Australia
        - Most bites in urban area's
        - **Venom (latrotoxin)** →Uncontrolled Release of NTs *ACh*, *NE*, and *GABA*
        - Many bites will not result in envenoming or symptoms
        - 20% of bite victims require treatment
        - **Symptoms**
          - extreme pain - locally
          - local sweating and sometimes piloerection
          - Swollen or tender regional lymph nodes
          - Malaise, nausea, vomiting
          - Chest or abdo pain
          - Headache, fever, hypertension
          - Rare – seizures, acute pul. Oedema, coma, death
        - **Treatment**
          - **Antivenom** – Generalised Symptoms i.e. headache, vomiting etc
          - **Analgesia and ice -** Local Symptoms
          - Usually 6 hours observation
          - Give antivenom IM not IV
          - Have adrenaline ready – anaphylaxis
          - Usually need 1 vial – occasionally 2

      - **Funnel-webs:** all have very toxic venom
      - **Hadronyche** - widespread coastal Qld
      - **Mouse spiders (stocky trapdoors)** - can cause significant systemic illness especially in children
      - **Bird eating spiders (theraphosidae)** - can cause systemic symptoms & illness
      - **Fiddle-back (loxosceles)** - can cause systemic illness & probably the major cause of necrotic aracnidism. Numerous spiders including huntsman, wolf, white-tailed, black house (window), brown house (cupboard) & 'large sac' species can cause painful bites &/or systemic illness &/or necrotic ulcers.
    - Scorpians
    - Ticks

- **Snakes:**
  - Australia has most venomous snakes in the world
  - Fatalities uncommon (1-2/year)
  - Venom injected form venom glands – modified parotid glands
  - **Signs of Envenomation**
    - **General** – headache, N & V, Abdo pain
    - **Neurotoxins** – ptosis, weakness, blurred vision, cardio – resp failure, dysarthria
    - **Myotoxins** – Rhabdomyolysis, myoglobulinuria
    - **Procoagulants** – Afibrinogenaemia, bleeding diathesis
    - **Local cytotoxins** – tissue damage
  - **Venom – Mechs of Action:**
    - **Potent prothrombin activators**
      - →Consumptive coagulopathy
    - **Tiger and taipan powerful pre-synaptic neurotoxins – block NT release**
      - →Descending paralysis – difficult to reverse
    - **Death adder post synaptic neurotoxins** – reversible. Block Ach receptors.
    - **Rhabdomyolysis** – renal failure
  - **5 Main Venom Groups:**
    - **Taipan**
      - Rate of envenomation - >80 %
      - **Venom contains:**
        - myotoxins
      - **Clinical effects:**
        - haemotoxins →Consumptive coagulopathy in mins
        - Myotoxins + Pre/post Synaptic Neurotoxins →
          - Flaccid paralysis in hours
          - Autonomic Dysregulation
        - Renal failure late – sec to coagulopathy
      - **Treatment:**
        - Monovalent antivenom
    - **Brown:**
      - Envenomation rate – 20 -40 %
      - **Clinical effects**
        - Coagulopathy common
        - Renal damage – sec to coagulopathy
        - Paralysis unusual
      - **Treatment:**
        - Monovalent brown antivenom
    - **Death adder:**
      - Envenomation 40 – 60 %
      - **Clinical effects:**
        - Paralysis
        - Nil coagulation effects
        - Post synaptic neurotoxins
      - **Treatment:**
        - Reversible with antivenom
    - **Black:**
      - Envenomation 40 – 60 %
      - **Clinical effects:**
        - Local effects at site
        - Lethality rate - < 1%
        - Minor coagulopathy only
        - Anticoagulant effect- no consumption of clotting factors

- **Aims of Treatment**
  - Reduce risk systemic absorption
  - Eliminate clinical effects of venom
  - Reduce risk death
  - SPECIFIC – antivenoms
  - **GENERAL – 1st aid, wound care, prevent reactions to antivenom**
    - Pressure immobilisation bandage + Splinting the leg
    - Go over site and then proximally
    - Complete immobilisation
    - **Don't wash site – You'll need to swab it.**
    - Leave bandage in situ until antivenom or normal bloods and no clinical signs
- **Identifying the Culprit:**
  - Never rely on "experts" or colour
  - SVDK – venom group not species
  - SVDK – cut hole in bandage
  - Urine VDK – no bite site seen
    - SVDK neg but clinically enven
    - discard first urine
  - Monovalent if VDK positive
  - No blood VDK
  - Specific antivenoms less hazardous than polyvalent



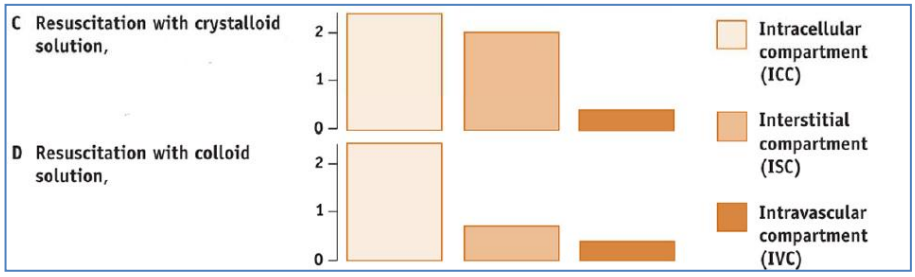
- **ANTIVENOMS:**
  - Horse serum
  - Risk anaphylaxis < 1%
  - 4% assoc with minor reactions
  - Delayed serum sickness
  - Tasmania – tiger
  - Victoria – tiger and brown
  - Elsewhere – everything!!
  - **HOW MUCH?**
    - Iniatially 1 amp polyvalent if clinically envenomated – await VDK
    - May need several doses
    - Titrate to clinical effect
    - Brown snake – 4 amps
    - Dilute 1:10 with crystalloid over 30 mins
    - Pre medication - controversial
    - Have adrenaline infusion ready

FLUID RESUSCITATION

FLUID REPLACEMENT THERAPY:

Crystalloid Vs. Colloid Solution:

- **Crystalloids:**
  - = Aqueous Solutions of Mineral Salts or other water soluble molecules.
  - Crystalloids have a *Low* Osmotic-Pressure in Blood due to Haemodilution.
- **Colloids:**
  - = Mixtures of Larger Insoluble Molecules. (NB: Blood *itself* is a colloid)
  - Colloids Preserve a *High* Colloid-Osmotic Pressure in the Blood.



Crystalloid Solutions:

- ***Saline:**
  - The Most Commonly used Crystalloid.
  - **Advantage –** Is *Isotonic* → Does not cause dangerous *fluid shifts*.
  - **Disadvantage –** If you only replace fluid, O_2 Carrying Capacity goes down (Dilution Anaemia)
    - Also, since it raises Extracellular Fluid, it's not suitable for Pts. with Heart Failure/Oedema.
  - Used for General Extracellular Fluid Replacement
- **Dextrose:**
  - Saline with 5% Dextrose – Used if Pt is at risk of Hypoglycaemia; or Hypernatraemia.
  - NB: Becomes Hypotonic when Glucose is Metabolised → Can cause fluid overload.
- **Lactated Ringer's/Hartmann's Solution:**
  - A Solution of Multiple Electrolytes:
    - Sodium
    - Chloride
    - Lactate
    - Potassium
    - Calcium
  - Used in Pts with Haemorrhage, Trauma, Surgery or Burns.
  - Also used to Buffer Acidosis

Colloid Solutions:

- **Albumin:**
  - Albumin 40g/100ml - Used in Liver Disease, Severe Sepsis, or Extensive Surgery.
  - Albumin 200g/100ml – Used in Haemorrhage/Plasma loss due to Burns/Crush Injury/Peritonitis/ /Pancreatitis; or Hypoproteinaemia; or Haemodialysis
- **Polygeline (Haemaccel):**
  - = Gelatin Cross-linked with urea.
  - Used in Dehydration due to GI Upsets (Vom/Diarrhoea)

Blood Products:

- **Whole Blood:**
  - RBCs, WBCs, Plasma, Platelets, Clotting Factors, Electrolytes (Na/K/Ca/Cl).
  - Used to Replace Blood Volume & Maintain Haemoglobin Level → ↑O_2-Carrying Capacity
- **RBCs:**
  - Used to Increase Haematocrit (proportion of RBCs) → ↑O_2-Carrying Capacity

- **Plasma:**
    - Plasma (With Plasma Proteins), Clotting Factors, Fibrinogen, Electrolytes (Na/K/Ca/Cl).
    - Used to restore Plasma Volume in Hypovolaemic Shock & Restore Clotting Factors.

Fluid Resuscitation Principles

- **Good to know where the fluids that you give actually go:**
    - **Eg. Average 70kg man**:
        - 60% water :. Total body water (42L)
        - 2/3 Intracellular Fluid (28L)
        - 1/3 Extracellular Fluid (14L)
        - 5L blood [1/3 RBC (1.5L); 2/3 Plasma (3.5L)]
- **So What happens to the Different IV Fluids?:**
    - **Glucose is actively taken into cells**
        - (volume of distribution is large, as none effectively remains in blood)
        - Therefore Glucose IS NOT suitable for pressure fluid resuscitation.
    - **Crystalloids (ie. Saline) – is kept in ECF by Na/K-ATPase**
        - :. Volume of distribution = 14, and of that, 25% remains in circulation (3.5L)
    - **Colloid, kept in blood by capillary membrane (Albumin, Gelatine, etc)**
        - Volume of Distribution is 3.5L, and ALL of that remains in Circulation.
    - (500mL of Colloid = 2L of Crystalloid) – (NB: So long as you give the right amounts,
- **Blood:**
    - **The best fluid to replace blood loss is Blood**
    - But, either saline/hartmanns or colloid are still ok.
    - NB: Blood has risks (immunogenic/infections/etc)
    - NOT Glucose

Fluid Management (Surgical Context)

NBs:

- Water moves with solute (Esp. Salt & Sugar)
- Propofol → ↓PVR, Preload & Contractility → ↓BP
- Neuromuscular Blockers → Venous Pooling → ↓BP
- NB: *Anuria* = Obstruction....NOT Hypovolaemia

Indications:

- **Dehydration**
- **Fluid Loss**
- **Blood Loss**
- **3rd Spacing**

Fluid Replacement Goals:

- Replace Pre-Existing Deficits
- Replace Ongoing Normal Losses (Obligatory Losses)
- Replace 3rd Space Losses
- Replace Bleeding

2 Types of Fluids:

- **Crystalloids (Salts/Sugars/Lactate):**
  - Normal Saline (0.9% NaCl)
  - Dextrose (4% Dextrose + 0.5% NaCl)
  - Hartmann's (Lactate)
  - (Cheap, Effective, Safe)
- **Colloids (4% Albumin):**
  - ONLY for Surgical / Renal Patients
  - NOT for Resuscitation
  - (Expensive, but Pulls fluid into IV Space)
- **(Blood Products)**
  - Whole Blood
  - Packed RBCs
  - FFP

3 Compartments:

- **1. Intravascular** (5L)
- **2. Interstitial** (10L)
- **3. Intracellular** (30L)

"1/3 Rule":

- For Every 1L of Crystalloid Fluid, only 300mL ends up IV.
  - ∴ 1L Blood Loss requires 3L of fluids.

"35-45 Rule":

- **Normal Ranges:**
  - **Na** 135-145
  - **K** 1.35-1.45
  - **pH** 7.35-7.45

"4-2-1 Rule of Fluid Replacement":

- **Bolus (Vol. Of Estimated Acute Losses)**
- 4mL/kg/hr for the 1st 10kg
- 2mL/kg/hr for the next 10kg
- 1mL/kg/hr for remaining kgs.
- **(Ie. 60mL/hr for 1st 20kg, + 1mL/kg/hr for remaining kgs)**
- **(Ie. 100ml/hr for 1st 60kg, + 1ml/kg/hr for remaining kgs)**

OSCE Station: "Assess Fluid Balance":

- **1. ABCDE –** (Airway, Breathing, Circulation, Disability, Expose)
- **2. Specific Signs of Fluid Balance:**
  - **Fluid Overload:**
    - Pulmonary Oedema (Basal Crepitations/Cough/Dyspnoea)
      - **Rx: Call for help + LMNOP:**
        - **L**asix (Frusemide → ↓Fluid Volume)
        - **M**orphine (Vasodilate)
        - **N**itrates (Vasodilate)
        - **O**xygen (Maximise Sats)
        - **P**ositioning (Sit up 90^{o})
    - Hypertension
    - ↑JVP
    - Pleural Effusion
    - Ascites
    - Peripheral Oedema
    - Polyuria (Normal = >>500mL/day)
  - **Fluid Depletion:**
    - Dry Mucous Membranes
    - Postural Hypotension
    - Tachycardia
    - Supine Hypotension
    - Loss of Skin Turgor
    - Lethargy/Confusion
    - Oliguria (Normal = >>500mL/day)
- **3. Cannulate**
  - Just stick the needle to the arm with tape to simulate cannulation.
- **4. Write up fluids in chart!!!**
- **5. Hook up bag:**
  - Aseptic technique
  - Prime line
  - Purge line of bubbles
  - Hook up to cannula

Emergency Medicine Notes
GI Emergencies

Principle Topics This Week:

- Difference between different types of Abdominal Pain
- GI Tract Bleeding
- Infection/Inflammation
- Hepatic Dysfunction
- Pancreatic Dysfunction

Gastrointestinal Emergencies:

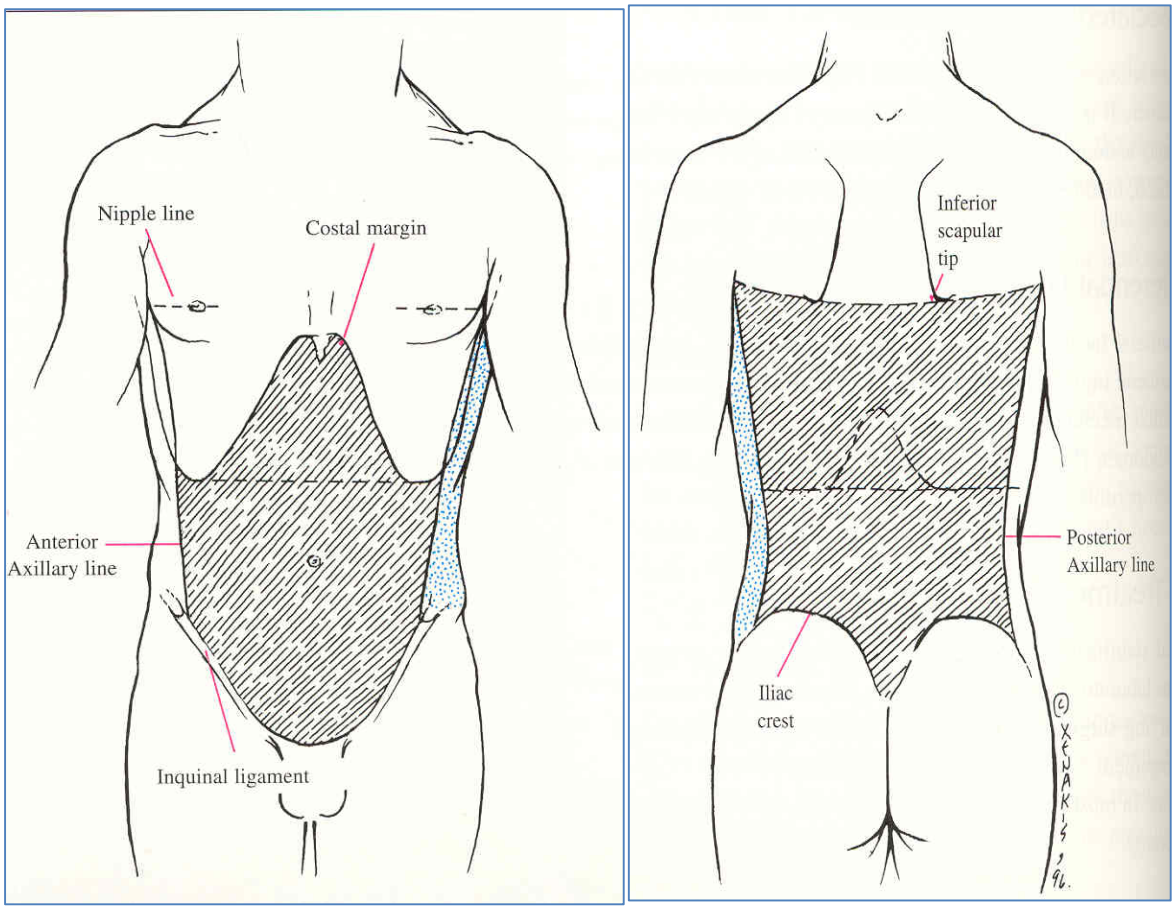
- Is ***not*** (adult) gastroenterology
- Covers all ages and many disciplines

An Important distinction:

- Gastrointestinal emergency - Vs - Intra-abdominal emergency (Not the same thing)

Anatomy:

- **The Abdomen**

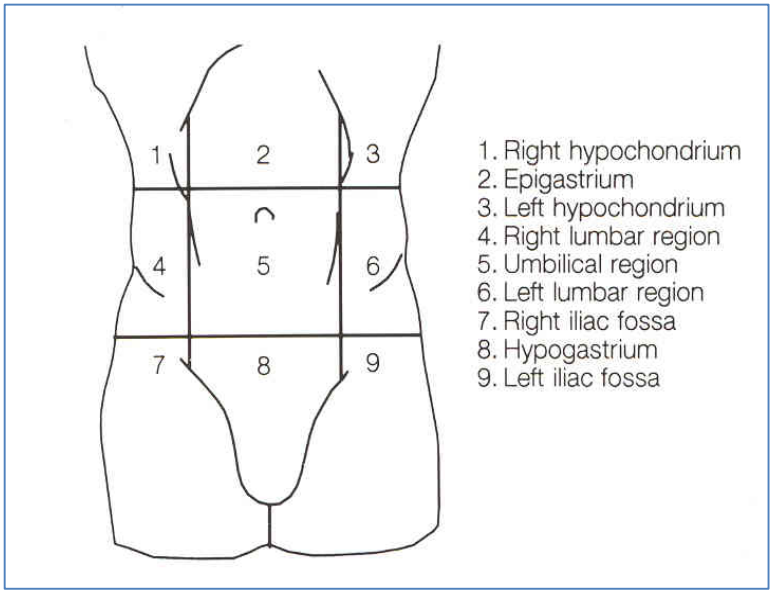


Note that the chest and abdominal boundaries *aren't* mutually exclusive

Abdominal injuries can involve the chest

Chest injuries can involve the abdomen

- **Abdominal Quadrants**



Note that the chest and abdominal boundaries *aren't* mutually exclusive

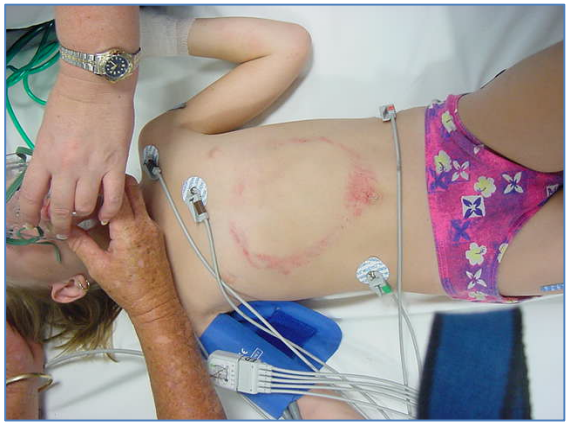
Abdominal injuries can involve the chest

Chest injuries can involve the abdomen

- **Abdominal Contents – Not just the GI Tract!**
  - **Gastrointestinal Tract:**
    - Oesophagus
    - Stomach
    - Duodenum
    - Small Bowel (jejunum and ileum)
    - Appendix
    - Large Bowel/Rectum/Anus
  - **Other 'Intra-Abdominal' Contents:**
    - Liver and Pancreas
    - Gallbladder
    - Spleen
    - Kidneys
    - Ureters
    - Bladder
    - Uterus and ovaries
    - Aorta and Vena Cava
    - Lymphatics, Nerves, Muscles

- **Retroperitoneal Organs:**
  - **Urinary System:**
    - Adrenal Glands
    - Kidneys
    - Ureters
    - Bladder
  - **Cardiovascular System:**
    - Aorta
    - Inferior Vena Cava
  - **GI Tract:**
    - Oesophagus
    - Head & Neck of Pancreas
    - Rectum

Gastrointestinal Emergencies:

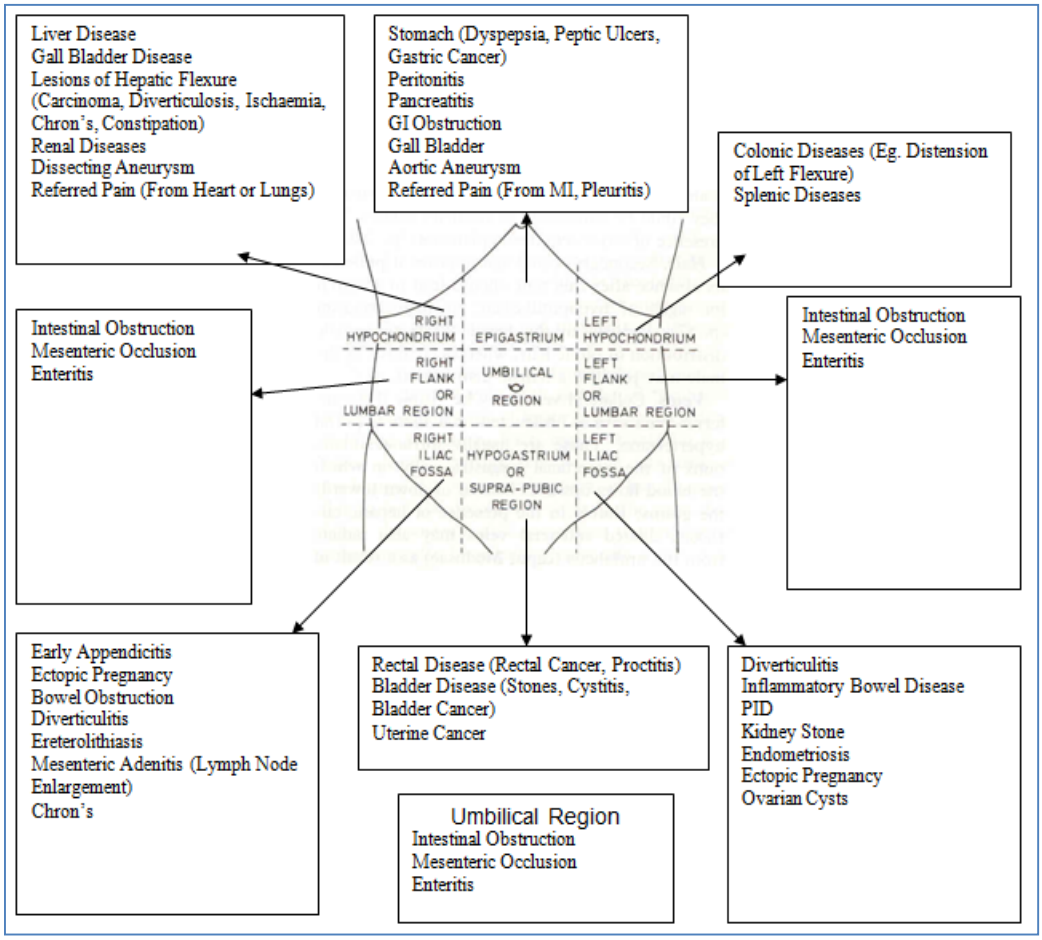


(Pressure mark on abdomen – suggest possible traumatic injury)

- **Abdominal Pain:**
  - A very common reason for patients to come to the Emergency Department
  - **We will focus on the *GIT causes of Abdominal Pain*:**
    - Almost endless!
    - Think of an organ then apply the 'surgical sieve'
      - Infection
      - Trauma
      - Neoplasia
      - Haemorrhage
      - Toxins/Drugs
  - **3 Types of Abdominal Pain:**
    - **1. Visceral ('Colicky') Pain:**
      - Pain Arising from abdominal *viscera*
        - *Typically due to Pressure*
        - *Typically Diffuse Pain* (Poorly localised – often, but not always, felt in periumbilical region)
        - Fluctuating in Intensity (Comes & Goes)
      - Transmitted by autonomic nerve fibres
      - Often associated with nausea and autonomic symptoms (e.g. sweating)
      - Pts tend to move around a lot (Can't get comfortable)
    - **2. Somatic/Parietal Pain:**
      - Pain due to Inflammation/Irritation of the Parietal ***Peritoneum***
        - *Typically Very Localised*
        - *Sharp Pain (Hurts to Move, Cough, Breathe*)
        - *Irritated by Movement*
      - Usually implies involvement of the (parietal) peritoneum
      - Transmitted via somatic nerves
      - ***Well localised***
    - **3. Referred Pain:**
      - Pain referred to one location from pathology in a different location
      - Usually associated with Embryonic Dermatome Origins of the Affected Structures
      - Eg. Diaphragmatic Pain felt at the Shoulder Tip
      - **Beware of extra-abdominal pain referred *to the abdomen***
        - ***Eg. Myocardial ischaemia*/Infarction:**
          - Inferior Infarcts often → Epigastric pain
        - ***Eg. Testicular pathology***
          - Eg. Testicular Torsion → hypogastric pain

- **Assessing Pain:**
  - **Pain Qualities:**
    - **Poorly defined or well localised**
    - **What does the pain make you do?**
      - **If Move around (can't get comfortable) – Somatic**
      - **If Can't move – Visceral**
    - **Waxing and waning (typical of colicky pain)**
      - Pain comes & goes
      - Usually an obstruction of something
    - **Constant – sharp or dull**
      - **Sharp implies peritoneal pain**
      - **Dull implies Visceral Pain**
    - **Exacerbating or mitigating factors (e.g. movement)**
      - Better when you eat?
      - Worse when you eat? (Eg. Gastric/duodenal ulcer)
      - Worse when you move?
    - **Progression from visceral to parietal as pathology progresses (e.g. appendicitis:**
      - Begins as Visceral
      - → Irritates Peritoneum → Somatic/Parietal Pain
  - **What does the patient do?**
    - **Visceral Pain - Colic- can't get comfortable, moves around.**
      - Biliary colic
      - Renal colic
    - **Parietal Pain - worse with movement, tend to keep still**
      - Bleeding or infection
  - **Radiation?**
    - Where does the pain go?

Potential Sources of Pain in Each Abdominal Quadrant:



- **GI Bleeding:**
  - A common problem – most people will have some form of GI bleeding during their life time!
  - Can be Trivial/life-threatening
  - May be painful or painless
  - Underlying pathology may or may not be serious
  - **Terms:**
    - **Haematemesis**
      - Vomiting blood – may be fresh or denatured (dark)
      - Can be VERY SERIOUS
      - Usually Implies bleeding from the ***Stomach***
      - **Priority** = Get Large-Bore IV Access & Cross-Match Blood type (Because people can bleed out very quickly from the stomach)
    - **Haematochezia**
      - Rectal Bleeding
      - The passage of bloody stools
      - Usually implies bleeding from ***lower GIT***
    - **Melaena**
      - black tar-like stool (usually from upper GI bleeding)
      - Usually implies bleeding from ***upper GIT***
  - **Origins:**
    - Can originate from *anywhere* in the GI tract from oropharynx to anal margin
    - Broad range of pathology e.gs:
      - Inflammation/Ulceration
      - Infection
      - Neoplasia
      - Trauma
  - **Occult (Hidden) GI Bleeding:**
    - GI tract bleeding may result in *occult blood loss* (occult = hidden)
    - An important cause of Chronic Anaemia
    - Detected by testing stool for 'faecal occult blood'
  - **NB: Acute GI tract bleeding may initially be *concealed.***
    - **:. Consider GI Bleeding in any patient with signs of hypovolaemic shock**

- **Infection/Inflammation:**
  - Inflammation *may or may not* be caused by infection
  - **Think of organ then add 'itis' to end:**
    - Gastritis
    - Hepatitis
    - Colitis
  - **Causes of inflammation include:**
    - Infection
    - Immune response (Incl. Autoimmune)
    - Drugs/Toxins
    - Trauma
  - **May have local and/or systemic manifestations**
    - **Local:**
      - Pain
      - Swelling
      - Organ dysfunction
    - **Systemic:**
      - feeling unwell
      - fever
      - sweating
      - circulatory changes or compromise

- **Dysfunction:**
  - Results from **too little** or **too much function**
  - Think of the underlying organ and its function then apply the 'too little' or 'too much' concept
    - Just applied physiology!
    - Think through the 'surgical sieve'
      - Infection
      - Trauma
      - Neoplasia
      - Haemorrhage
      - Toxins/Drugs
  - **Liver:**
    - **Functions:**
      - Production of Coagulation Factors
      - Production of Albumin
      - Produces Angiotensinogen
      - Production of Bile
      - Detoxification of Blood/Enzymatic Conversion of Drugs
      - Metabolism of Bilirubin & Biliverdin
      - Carbohydrate Metabolism
        - Gluconeogenesis
        - Glycogenolysis
        - Glycogenesis
      - Cholesterol & Fat Packaging
      - Amino Acid Metabolism & Synthesis
      - Converts Ammonia to Urea
    - **Dysfunction →:**
      - Easy Bruising (due to low Coagulation Factors)
      - Oedema due to Low Albumin
      - Unexplained Hypoglycaemia
      - Jaundice (Hyperbilirubinaemia →Bilirubin deposits in skin → Intense Itch)
      - Pale Stools (If bilirubin/biliverdin/stercobilin isn't excreted in bile)
      - Dark Urine (if bilirubin mixes with urine)
      - Abnormal Metabolism of fats
      - Excessive Fatigue
    - **Markers for Evaluating Liver Pathology/Dysfunction:**
      - Liver Function Tests
        - ALT
        - GGT
      - INR (Coagulation Test)
      - Abnormal Serum Glucose
      - Albumin Levels
      - **NB:** Inflammation → Release of AST & ALT (liver cell damage) – But doesn't imply dysfunction.

- **Pancreas:**
  - **Functions:**
    - Endocrine:
      - (Beta Islet Cells) → Insulin Secretion
      - Glucagon
      - Somatostatin
    - Exocrine:
      - (Acinus)
      - Digestive Enzyme Production
        - Amylase
        - Pancreatic Lipase
        - Trypsinogen
        - Chymotrypsinogen
        - Elastase
        - Carboxypeptidase
  - **Dysfunction →:**
    - Dysregulated Glucose Metabolism (Diabetes)
    - ↓Pancreatic Lipase → ↓Fat Absorption → Fatty Stools
    - ↓Other Pancreatic Enzymes → ↓Digestion/Absorption of other nutrients (eg. poor Protein Digestion)
    - → Malabsorption & Malnutrition
  - **Markers for Evaluating Pancreas Pathology/Dysfunction:**
    - Elevated Serum Amylase
    - Elevated Serum Lipase
  - **Location of Pain:**
    - Severe Epigastric pain radiating to the back

ASSESSMENT/MANAGEMENT

- **DON'T FORGET TO MAKE THE MAIN THING THE MAIN THING!**
- **ABC Approach:**
  - (Stabilise the unstable)
  - Airway- OK
  - Breathing- OK
  - Circulation - IV access, IV analgesia, titrated opiates, IV antiemetics, IV fluids
- **Analgesia**
- **Start correcting obvious fluid and electrolyte disturbances**
  - Especially in Vomiting (Hypokalaemia, etc)
- **History/Examination/Investigation to define pathology**
  - **Careful examination- Other causes? ESP AAA**
  - **Investigations**
    - Immediate- Urine FWT- Blood?
    - FAST scan- free fluid, AAA?
    - U&E's, FBC, etc
  - **Imaging:**
    - CT to image stone, hydronephrosis
    - X-Ray (Now obsolete to the CT for Abdominal Imaging)
      - Mainly used for diagnosing Bowel Obstructions – (Can see multiple fluid levels)
- **Specific Treatments if required:**
  - **Eg. Nasogastric Tube:**
    - **Indications:**
      - Parenteral Feeding
      - Parenteral Drug Administration
      - Administering activated charcoal
      - Treatment of Anorexia Nervosa
      - Aspiration of stomachs contents
    - **Procedure for Insertion:**
      1) measure with the tube from the tip of the patient's nose to their ear and down to the xyphoid process
      2) The tube is marked at this level to ensure that the tube has been inserted far enough into the patient's stomach.
      3) The end of a plastic tube is lubricated (local anaesthetic, such as 2% xylocaine gel, may be used
      4) The tube should be directed aiming down and back as it is moved through the nasal cavity and down into the throat.
      5) Patient may gag → Asked to swallow
      6) Once the tube enters the oesophagus, it is easily inserted into the stomach
      7) Ensure the tube hasn't passed through the larynx into the trachea.
      8) Inject air into the tube & auscultate for bubbles in stomach.
      9) X-ray to ensure correct position
    - **NB: Careful not to pass it through the larynx. – Confirmed by:**
      - Inject air into the tube & auscultate for bubbles in stomach.
      - X-ray to ensure correct position
      - Laryngoscope

- **Case: Renal colic?**
  - **60 year old man**
  - **Symptoms:**
    - severe left loin pain,
      - Diffuse
      - Radiates to Groin
    - sweating
    - vomiting
    - distressed
  - **Vitals:**
    - P 110 (Bit Tachy)
    - BP 120/90 (Normotensive)
  - **Assessment?**
    - A- OK
    - B- OK
    - C?- Volume (Is he hypovolaemic)
      - Feel periphery (if cold, probably hypovolaemic)
  - **Priorities:**
    - IV access, IV analgesia, titrated opiates, IV antiemetics, IV fluids
  - **Suspicions:**
    - Renal Colic
    - Abdominal Aortic Aneurysm (AAA)
  - **Careful Examination:**
    - Most Likely Renal Colic
    - Other Causes?
    - Look at the patient (What are they doing?)
      - Are they moving around/Can't get comfortable (Typical renal colic)
  - **Investigations:**
    - Immediate:
      - Urine (is there Blood) – If so, suggests renal colic.
      - Ultrasound – Check for Aneurysm
    - Quick:
      - FBC
      - CT

SS Questions:

Which is Retroperitoneal?

- Stomach
- Duodenum
- Transverse Colon
- Sigmoid Colon

Which is Intraperitoneal?:

- Pancreas
- Kidneys
- Adrenals
- Liver

Which typically produces pain referred to the right iliac fossa?

- Appendicitis
- MI
- Ruptured Ectopic Pregnancy
- Testicular Torsion

Which is non-abdominal cause of abdo pain?

- Appendicitis
- Ectopic Pregnancy
- Diverticular Disease
- Pneumonia

Which causes parietal pain?

- Biliary Colic
- Renal Colic
- Ruptured Ectopic Pregnancy
- Bowel Obstruction

Gas under diaphragm caused by:

- Bowel obstruction
- Ruptured ectopic pregnancy
- Abdominal viscus perforation
- Bowel obstruction

Which suggest liver failure?

- Raised urea
- Raised glucose
- Raised INR (↓production of coagulation factors by the liver)
- Raised AST

Which liver function test measures synthetic function:

- Bilirubin
- Albumin
- ALP
- Gamma Glutyl-Transferase

Which suggests liver inflammation?

- Raised AST
- Raised Gamma GT
- Raised INR
- Raised Immunoglobulin

Which is raised in pancreatitis?

- INR
- APPT
- Lipase
- Glucose

50yrs male presents with severe central abdo pain radiating through to the back. The most likely organ is involved is?

- Stomach
- Liver
- Gall Bladder
- Pancreas

20yrs female presents with severe shoulder tip pain with tachycardia and hypotension. What is most likely?

- Ruptured Ectopic Pregnancy
- Ruptured Aortic Aneurysm
- Dissection of Thoracic Aorta
- Bleeding Gastric Ulcer

35yrs presents with left loin pain radiating to his groin. Diagnosis?

- Ruptured AAA
- Left Renal Colic
- Splenic Infarct
- Pancreatitis

70yrs presents with collapse and left sided loin pain radiating to his groin. Which is most likely?

- Ruptured AAA
- Ruptured Gastric Ulcer
- Myocardial Infarction
- Urinary Sepsis

Alcoholic presents with bright haematemesis, haemodynamically stable. What is the priority?

- 22G IV, blood tests
- Large Bore IV + Cross match
- Immediate surgery
- Immediate gastroscopy

14yr boy presents with URTI symptoms and diffuse pain in his right iliac fossae. What is the diagnosis?

- Pancreatitis
- Mesenteric Adenitis (Relates to the URTI Symptoms)
- Appendicitis
- Cholecystitis

Emergency Medicine Notes
Haematological & Renal Emergencies

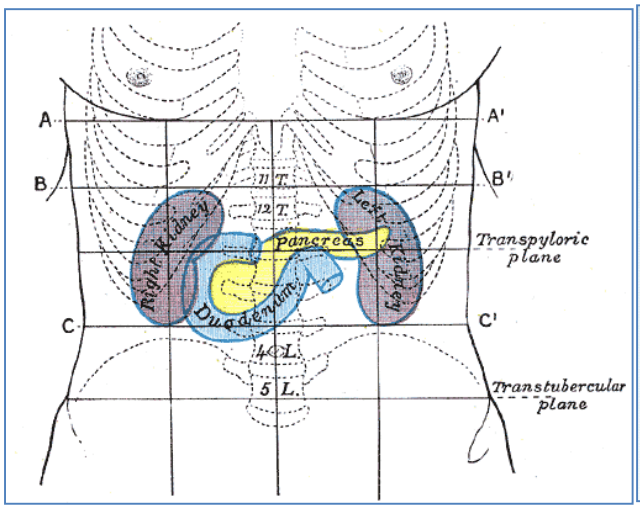
RENAL STUFF

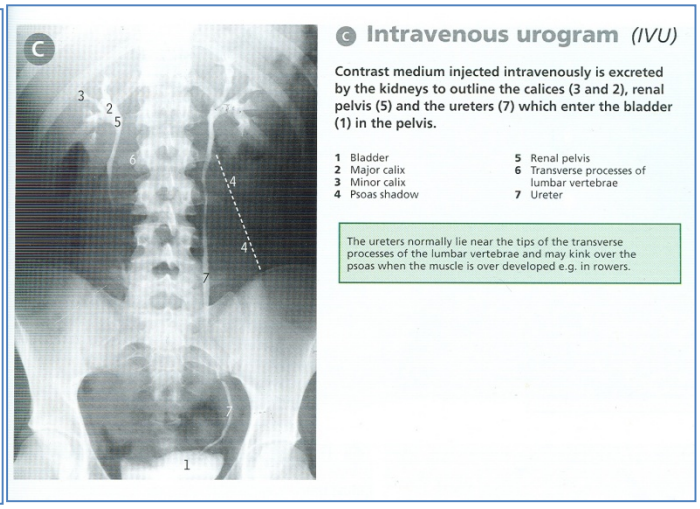
Overview:

- **Renal:**
  - Functions of the kidney
  - Renal failure – acute (and chronic)
  - Urogenic Pain
  - Catheterization of females and males
- **Haematology:**
  - Coagulation
  - Coagulopathies
  - Blood products and transfusions
  - Won't deal with: anaemia, neutropenia, etc or leukaemic emergencies per se

Basic Anatomy:

- **Kidneys:**
  - Approximate Size ≈ 10cm x 5cm x 3cm
  - Location = The hilar of each kidney is near the transpyloric plane (T12-L1)
  - Retroperitoneal. :. Rupture → Retroperitoneal Bleeding
- **Ureters:**
  - Travel from the Kidney → Bladder
  - Close association with Psoas Major Muscle
  - Length ≈ 20-30cm
  - Narrowest Parts (Ie. Where Renal Calculi are most likely to get Stuck):
    - Renal Pelvis
    - Where the Ureters cross the Pelvic Rim
    - Entrance to Bladder
- **Urethra:**
  - **Male – Longer (8 inches)**
    - Prostatic Urethra, Membranous Urethra, Spongy Urethra
    - Kinks at the prostate
    - Exits at the end of the penis
  - **Female – Shorter (1.5-2 inches)**
    - Obliquely downward and forward
    - Exits between the clitoris & the vagina



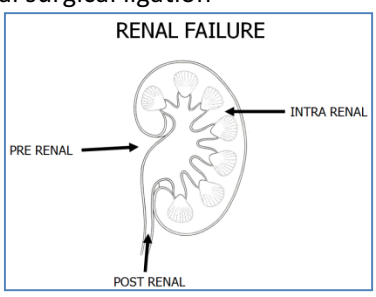


The Kidney

- **Functions of the kidney**
  - Erythropoiesis
  - Urine Production
  - Calcium Metabolism
  - Fluid/Electrolyte Balance (Na/K)
  - Renin Angiotensin System (BP Regulation)
  - Filters out toxins
  - Urea Excretion
  - Acid/Base Balance

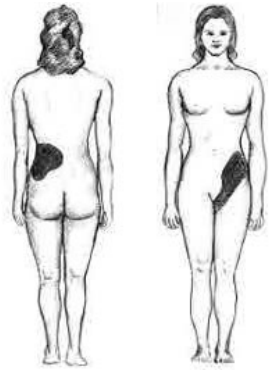
Renal Failure:

- **General Effects/Problems Encountered in Renal Failure:**
  - (Recall the functions of the kidney and then infer what happens when they are eliminated!)
    - Acid Base Balance (Renal Failure → Met. Acidosis)
    - Electrolyte Balance (Renal Failure → Na^+ & K^+ Retention)
    - Fluid Balance (Renal Failure → Fluid Overload & Hypertension)
    - ↓Erythropoiesis (Renal Failure → Anaemia)
    - Renin Angiotensin System
    - Calcium Metabolism (Renal Failure → Osteoporosis)
    - Uraemia
    - ↓Urine Output
    - ↓Toxin Excretion (Renal Failure → Accumulation of Urea & Creatinine)
- **Classification Of Renal Failure:**
  - **1. *Pre-Renal* Renal Failure:**
    - *Before the Blood Reaches the Kidney* (Ie. Impaired Glomerular Perfusion)
    - Structurally intact kidney fails because of ***impaired glomerular perfusion***
    - **Causes:**
      - Hypovolaemia (Eg. Blood Loss)
      - Decreased cardiac output (Eg. Heart Failure)
      - Renal artery obstruction (Eg. Embolism)
  - **2. *Intra-Renal* Renal Failure**
    - The ***kidney itself is damaged*:**
      - Problems can occur at any location along the structures.
    - **Causes**
      - *Acute glomerular nephritis*
      - *Tubular diseases* e.g. acute tubular necrosis
      - *Interstitial diseases* e.g. auto immune disorders such as SLE
      - *Vascular diseases* e.g. polyarteritis nodosa
  - **3. *Post-Renal* Renal Failure**
    - Due to ***outflow obstruction*** from the kidneys
    - HAS TO BE ***BILATERAL*** - unless the patient has only one kidney(!)
    - **Post-renal examples**
      - Cancer – esp. cervix or prostate
      - Blood clot
      - Calculi (Kidney stones – Bilateral)
      - Accidental surgical ligation

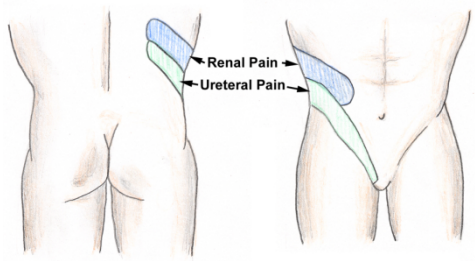


Urogenic Pain:

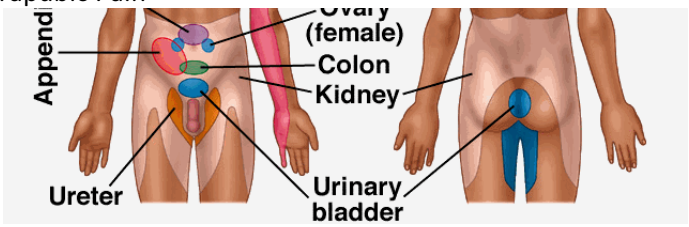
- **Nature of Pain may Vary:**
  - **Colicky Pain (Comes & Goes):**
    - Commonly caused by kidney stones
    - Pain comes in Waves due to Ureteric Peristalsis
  - **Constant Pain:**
    - Caused by a constant pathological process (Eg. Pyelonephritis, Ascending UTI, etc)
- **Location of Pain Varies Depending on Organ Affected:**
  - **Kidney Pain:**
    - Unilateral Flank/Back pain Radiating to Groin.



  - **Ureteral Pain:**
    - Flank-Groin Colicky-Type (Comes & Goes) Pain



  - **Bladder Pain:**
    - Suprapubic Pain

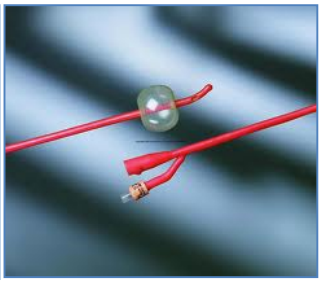


  - **Urethra Pain:**
    - Localised to the Urethra.

Catheterization (Females and Males):

- **Indications:**
  - Urinary retention
  - Urine Sample
  - Post-operative to assess urinary output, perfusion.
  - Prostatic obstruction:
    - BPH [most likely].
    - CA of prostate.
  - Other obstructions:
    - Clots.
    - Stones.
    - Bladder CA.
  - Trauma.
  - Paralysis.
- **Peri-Urethral Structures that might Interfere with Catheterisation:**
  - Labia
  - Foreskin
  - Prostate
  - Urethral Sphincters
- **Different Types of Catheters:**
  - **Foley (Brown Latex):** Cheapest, Commonest
  - **Silastic (Clear Silicone):** can leave in longer than Foley with less chance of complications
  - **Robinson's:** Has no balloon, is used for Short term drainage
  - **Coude:** Angled for easier insertion around prostate

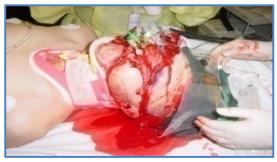


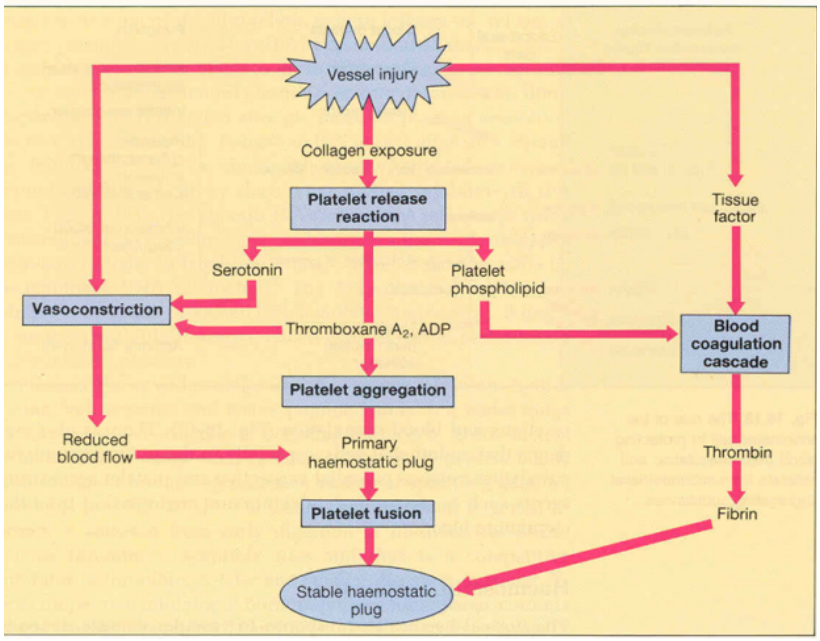
- **Basic Process of Catheterisation:**
  - Initial Steps:
    - Gather Equipment
    - Explain Procedure and get Consent
    - Lay pt into supine position + Spread Legs
    - Prepare Sterile Field + Apply Gloves
    - Cleanse Periurethral Mucosa with Cleansing Solution
  - Check Balloon for Patency
  - Coat the distal 2-5cm with Lubricant
  - Gently Insert Catheter into Urethra until 1-2inches *beyond* the point of Urine Flow.
  - Inflate Balloon with 10cc of Sterile Liquid.
  - Gently Pull Catheter back until Balloon is snug against bladder neck.
  - Connect To Drainage System + Make sure bag is below the level of the bladder.
- **Complications:**
  - Tissue Trauma
  - Infection
  - Bacteruria
  - Renal Inflammation
  - Pyelonephritis
- **Suprapubic Catheters:**
  - If trans-urethral catheterization isn't possible.
  - Involves piercing the bladder (via the peritoneal cavity) with a syringe.

HAEMATOLOGICAL STUFF

Haemorrhage Control:

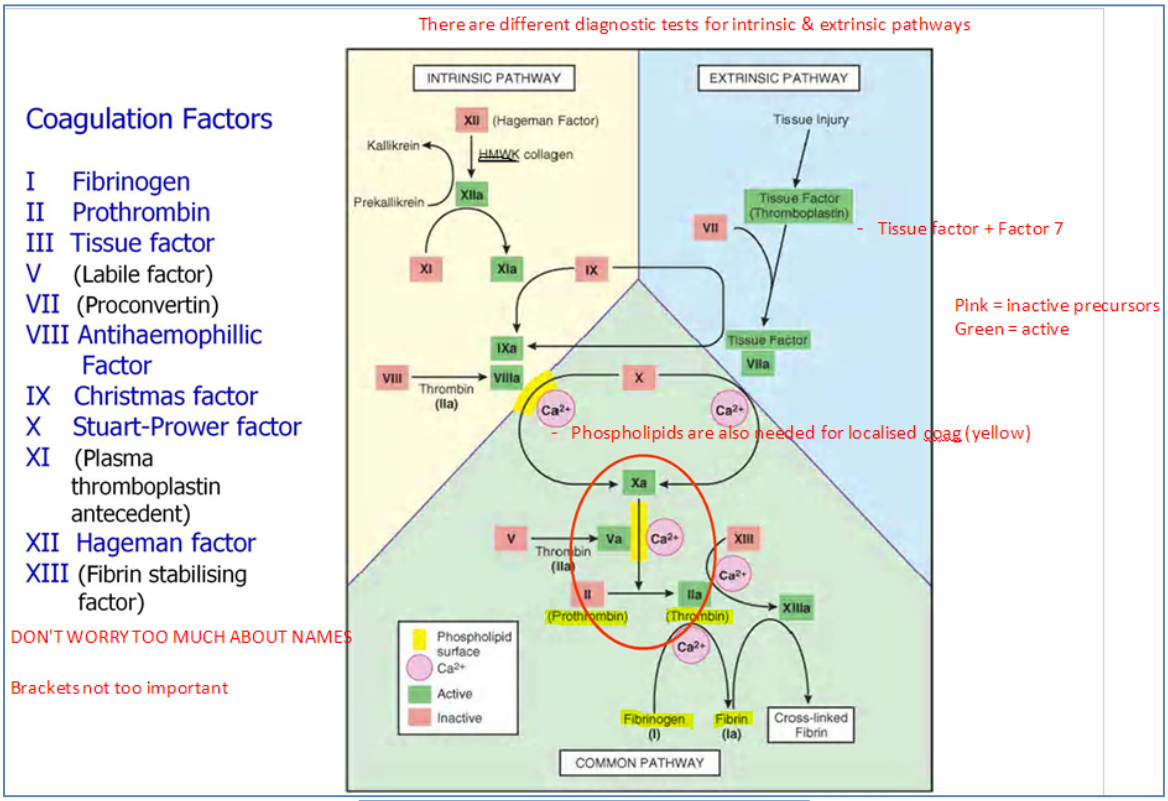


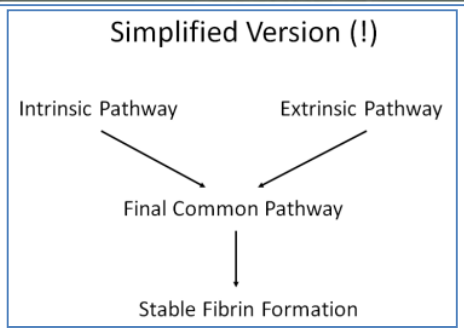
- **Coagulation:**
  - **We live on a knife-edge between *bleeding to death* and *clotting to death!***
    - A **constant** process of *clot formation* and *clot lysis.*
    - A battle between *pro-coagulants* and *anti-coagulants*
- **Terms:**
  - ***Anti-coagulation*** is inhibition of clot formation
  - ***Fibrinolysis*** is dissolution of a clot by 'dissolving' fibrin strands
- **Haemostasis – the big picture:**
  - Vessel Injury →
    - Vasoconstriction
    - Platelet Activation, Aggregation
    - Blood Coagulation Cascade



- **Key points:**
  - **Vasoconstriction:**
    - Important vascular reflex
    - Neural + local myogenic spasm + local humoral factors
    - More pronounced with vessel crush injury (vs. cutting)
  - **Platelet activation:**
    - Very Central Role - The underrated hero of coagulation
    - Platelets are 2 - 4µm in diameter
    - Contain Chemical Mediators →Vasoconstrict & initiate Coagulation Cascade + Repair.
      - →Form *Platelet Plugs*
    - Platelet Deficiencies = Either ↓*number* and/or ↓*function*
  - **Fibrin formation:**
    - Via the Coagulation Cascade

- **<u>Coagulation Cascade – *THE FOCUS OF THIS WEEK*:</u>**
  - **Intrinsic Pathway:**
    - Begins in the blood itself i.e. *intrinsic* to the blood
    - Initiated by activation of *Hageman Factor (Factor XII)*
  - **Extrinsic Pathway:**
    - Activated by damage to a vessel wall i.e. *extrinsic* to the blood
    - Initiated by *Tissue Factor (Factor III)*
  - **Common Pathway:**
    - Both Pathways → Formation of a: ***'Prothrombin Activator Complex' (PAC)***
    - PAC then activates final common pathway leading to formation of fibrin
  - **Formation and stabilization of Fibrin:**
    - Prothrombin → Thrombin
      - Fibrinogen → Fibrin
        - → Fibrin Deposition
- **NB: Calcium Ions:**
  - Essential for the coagulation cascade to function
  - Promote ***all*** reactions *except* the first two of the *intrinsic* pathway





The coagulation system is '***irreducibly complex***'

What does that mean? – Requires everything there for it to function. (An all or nothing process)

Abnormal Bleeding:

- **May or may not involve dysfunction of coagulation system:**
  - Coagulation cascade dysfunction (Coagulopathy)
  - Thromboycytopenia or platelet dysfunction
  - Loss of vessel integrity (especially the microvasculature e.g arteritis)
- **Coagulopathy - *Inborn* or *Acquired*:**
  - **Inborn Coagulopathy:**
    - Abnormal or deficient clotting factors
    - Genetic – usually hereditary
    - Eg. Haemophilia
  - **Aquired coagulopathy:**
    - **Problem with Factor Synthesis:**
      - Liver Disease
      - Vit.K Deficiency
    - **Inhibition of Coagulation:**
      - Coagulation Factor Antibodies
      - Drug Therapy (Eg. Heparin/Warfarin)
    - **Excess Consumption of Clotting Factors:**
      - Eg. Disseminated Intravascular Coagulation (DIC)
- **Bleeding Management Principles:**
  - ABC
  - If Obvious Exsanguinating Haemorrhage → control with Direct Pressure or Tourniquet.
  - If controllable → restore BP to normal
  - If not controllable → keep SBP ≤ 90mmHg until surgical control of bleeding achieved
  - Correct any underlying coagulopathy

Blood Transfusion:

- **Common Blood Groups & Characteristics:**

Phenotype	Genotype	RBC Antigens	Naturally occurring antibodies	Frequency
O	OO	None	AB	40%
A	AA, AO	A	B	30%
B	BB, BO	B	A	25%
AB	AB	A & B	None	5%
(Rh-D Positive)		D	No anti-D	
(Rh-D Negative)		-	Anti-D	

- **Universal Donor:**
  - O-Negative
    - No A or B Antigens
    - No Rh-D Antigens

- **Universal Recipient:**
  - AB-Positive
    - No anti-A or anti-B Antibodies
    - No anti-Rh-D Antibodies

- **Group Specific Blood Vs. Cross Matched Blood:**
  - **Group Specific =** Blood of any 'Type' (ABO,D) that's compatible with the Recipient. (20mins)
  - **Cross Matched =** Complex Pre-Transfusion Testing for Compatibility across *all* Blood Types. (1hr)

- **In Emergency Situations:**
  - Sometimes there isn't time to obtain a blood group or do a full cross match, so it is best to give O-Neg in an emergency situation.

- **What infections are screened for in donated blood and how long does the screening process take?**
  - HIV
  - Hep B/C (Not Hep A)
  - $HTLV_{1/2}$
  - Syphilis

- **What is the difference between *whole blood* and *packed red cells*?**
  - Whole blood = All blood components
  - Packed Red Cells = RBCs only

- **What is added to blood when it's donated? Why?**
  - Anticoagulants
  - Ca-Citrate
  - Glucose

- **How long can blood be stored for?**
  - 5-6 weeks

Cases:

- **1.** Driver of Semi—trailer, lost control crossing a bridge, crashed into the creek.
    - **Initial Assessment:**
        - Entrapped.
        - GCS 14
        - HR 150
        - BP 95/40
        - RR 40
    - **Problems:**
        - Trapped
        - Compound # right femur – Bleeding
        - Facial Lacerations
    - **Potential Issues:**
        - Crush Legs
        - ? Neck Injury
        - ?Chest – Heart/Lungs/Mediastinum
        - ?Liver/Spleen/Pancreas
        - ?Spine Injury
    - **What happened:**
        - Pre-hospital blood transfusion started
        - Wife arrived at the scene.
        - Fluid Replacement
            - 10L of Crystalloid
            - 14 units of blood
            - Fresh frozen plasma
        - Difficult extrication (trapped for 3hrs)
        - Intubated and MAST/PASG suit (An Anti-Shock “G-Suit” Trowsers) applied immediately after extrication.
    - **On Arrival:**
        - HR 135
        - BP Low
        - Hypothermic
        - Profuse blood loss
    - **Outcome:**
        - Died of persistent blood loss the next day.

Week 11 Renal and Haematological Emergencies MCQ's

1. All of the following are recognised functions of the kidney **except**:
    a. Acid-base homeostasis
    b. Fluid balance
    c. Blood pressure regulation
    d. Glucose homeostasis
    e. Regulation of erythropoiesis

Answer: D

2. All of the following are causes of intra-renal renal failure **except**:
    a. Acute tubular necrosis
    b. Cardiogenic shock
    c. Glomerular nephritis
    d. Interstitial nephritis
    e. Arteritis involving the vasa recta

Answer: B

3. The transpyloric plane is situated:
    a. At the level of the iliac crests
    b. At the level of the umbilicus
    c. At a point midway between the sternal notch and the symphysis pubis
    d. At the level of the xiphoid process
    e. None of the above

Answer: C

4. Urogenic pain radiating from the flank to groin **most** likely arises from the:
    a. Kidney
    b. Ureter
    c. Vesicoureteric junction
    d. Bladder
    e. Urethra

Answer: B

5. Which of the following clotting factors is **not** part of the intrinsic pathway?
    a. XI
    b. IX
    c. VII
    d. III
    e. XII

Answer: D

6. Blood group 'B' is **best** described as:
    a. Having 'B' RBC antigens and 'anti-A' antibodies
    b. Having 'B' RBC antigens and 'anti-B' antibodies
    c. Having 'A' RBC antigens and 'anti-B' antibodies
    d. Having 'A' RBC antigens and 'anti-A' antibodies
    e. Having 'AB' RBC antigens and 'anti-A' antibodies

Answer: A

7. All of the following may result in coagulopathy **except**:
    a. Factor VIII deficiency
    b. Liver disease
    c. Vitamin K deficiency
    d. Snake evenomation
    e. Thrombocythemia

Answer: E

8. Which blood group is regarded as the 'universal donor'?
    a. O –ve
    b. O +ve
    c. AB –ve
    d. AB +ve
    e. None of the above

Answer: A

9. Which blood group is regarded as the 'universal recipient'?
    a. O –ve
    b. O +ve
    c. AB –ve
    d. AB +ve
    e. None of the above

Answer: D

10. Approximately how long does it take to fully cross match blood?
    a. 10 minutes
    b. 20 minutes
    c. 30 minutes
    d. 40 minutes
    e. 50 minutes

Answer: E

11. Donated blood is screened for all of the following **except**:
    a. HIV
    b. Hepatitis B
    c. Hepatitis C
    d. Syphylis
    e. CMV

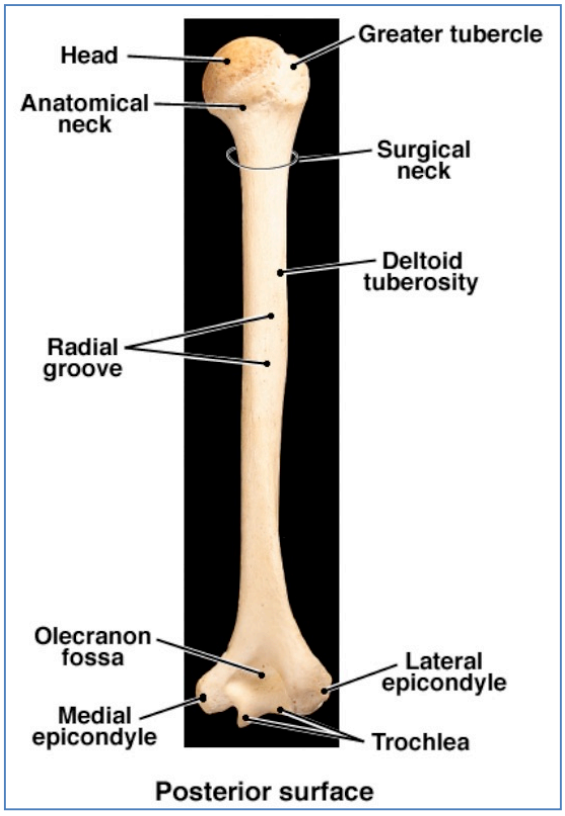
Answer: E

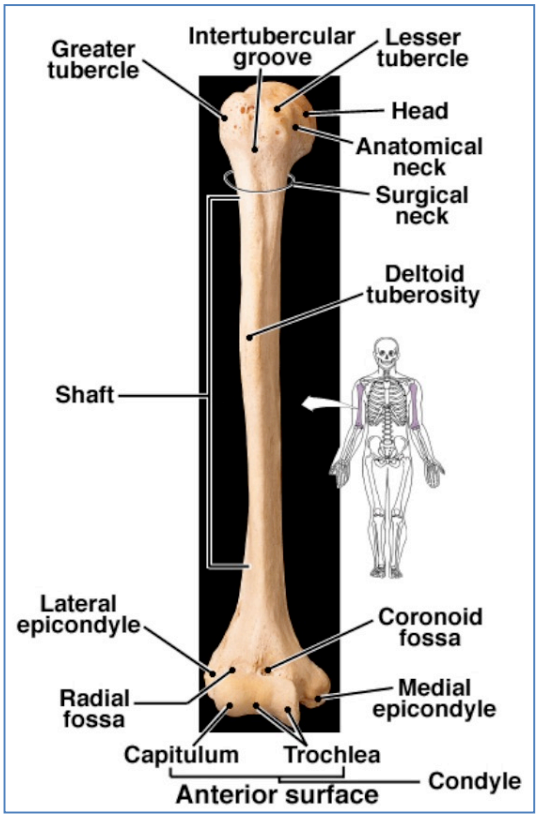
Emergency Medicine Notes
Revision of MSK Anatomy: Relevant to Common MSK Injuries

Shoulder Region, Arm & Hand: Bones, Muscles, Nerves, Veins & Arteries.

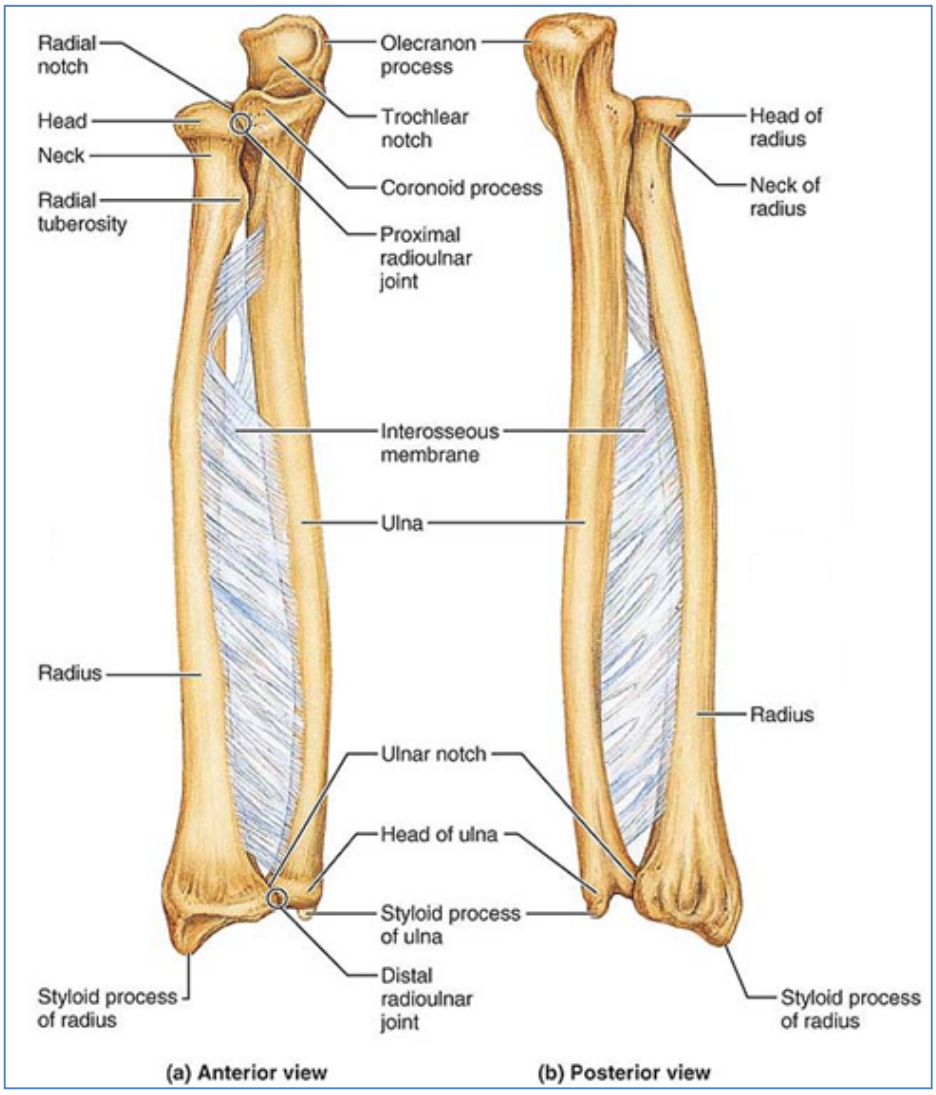
Bones & Landmarks:

- **Manubrium of Sternum (breastplate)**
  - Flat bone
- **Clavicle**
  - Long bone
  - **Articulations:**
    - Manubrium of Sternum
    - Acromion of Scapula
- **Scapula**
  - Irregular bone
  - Connects Humerus → Clavicle
  - **Articulations:**
    - Lateral ends of Clavicle
    - Head of Humerus
- **Humerus**
  - Long Bone
  - **Landmarks:**
    - Head
    - Neck
    - Medial Epicondyle
    - Lateral Epicondyle
  - **Articulations:**
    - Glenoid Process of Scapula
    - Radius
    - Ulnar

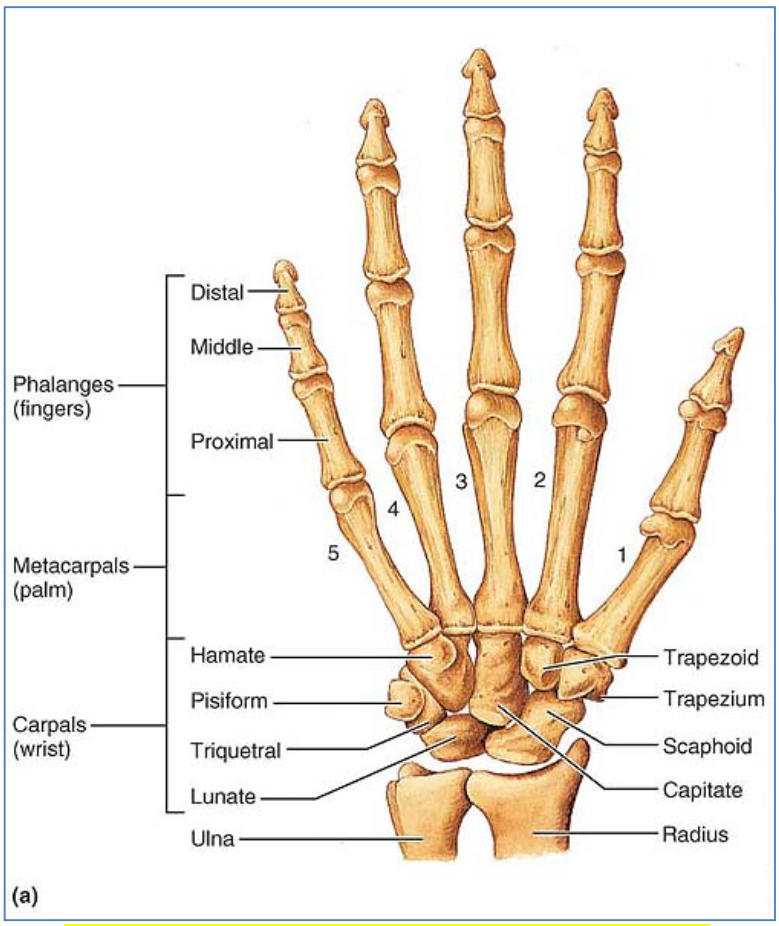




- **Ulna: "Elbow"**
  - Major forearm bone contributing to Elbow Joint
  - **Landmarks:**
    - Olecranon Process (Posterior Proximal)
    - Styloid Process of Ulna
    - Head of Ulna (Articulates with Wrist via Disc of Fibrocartilage)
  - **Articulations:**
    - Trochlea of Humerus
    - Bones of Wrist
    - Radius – via Interosseous Membrane (flat, flexible ligament spanning entire length)
- **Radius: "Rod"**
  - Major forearm bone contributing to Wrist Joint
  - **Landmarks:**
    - Head – concave (Articulates with Capitulum of Humerus)
    - Neck
    - Styloid Process Of Radius
  - **Articulations:**
    - Humerus
    - Bones of Wrist
    - Ulna – via Interosseous Membrane (flat, flexible ligament spanning entire length)



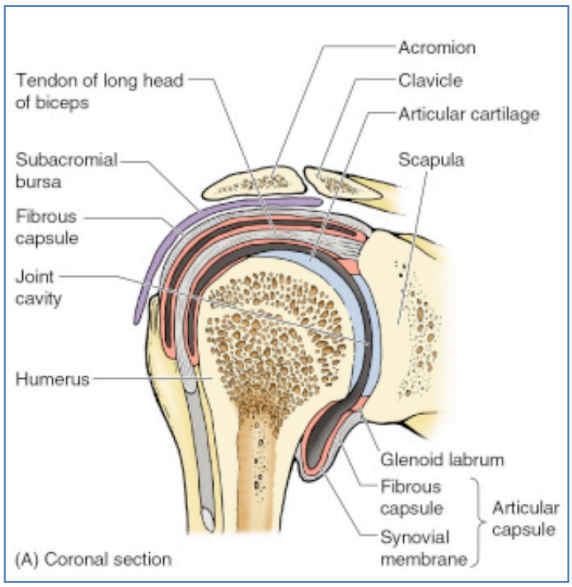
- **‘Hand’:**
  - **Carpals (“Wrist”):**
    - Scaphoid
    - Lunate
    - Triquetral
    - Pisiform
    - Trapezium
    - Trapezoid
    - Capitate
    - Hamate
  - **Metacarpals (“Palm”):**
    - Metacarpals # 1-5
  - **Phalanges (“Fingers”):**
    - Distal # 1-5
    - Middle # 1-5
    - Proximal # 1-5

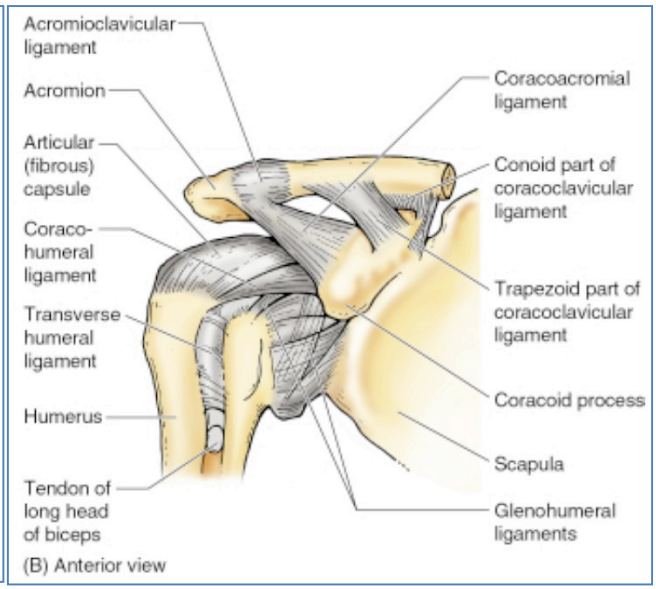


NB: Some **L**overs **T**ry **P**ositions **T**hat **T**hey **C**an’t **H**andle
Scaphoid, Lunate, Triquetrium, Pisiform, Trapezium, Trapezoid, Capitate, Hamate.

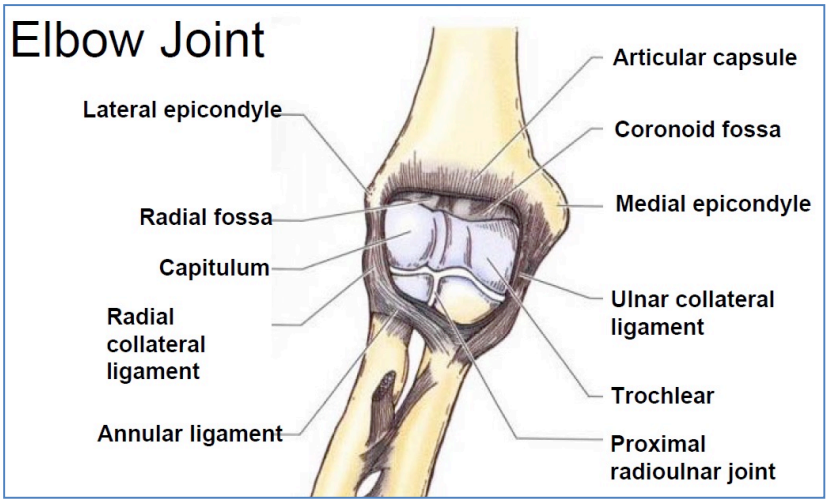
Joints & Ligaments:

- **GlenoHumeral Joint:**
  - **Features:**
    - Joins Humerous & Glenoid Fossa (cavity) of Scapula
    - Synovial ball & socket
    - Glenoid Fossa = Shallow → allows huge angle of movement.
      - High Mobility
      - Low Stability
  - **Bones:**
    - Head of Humerus
    - Glenoid Fossa of Scapula

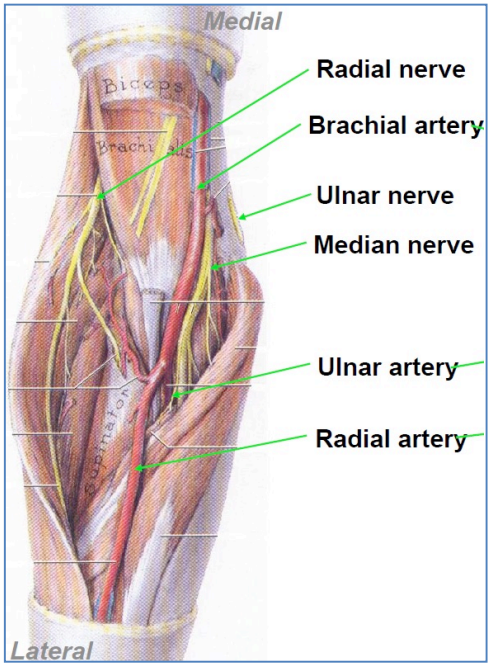


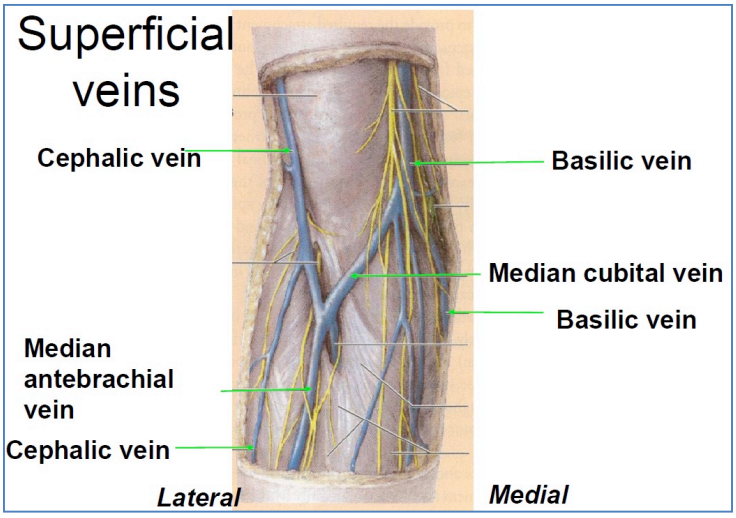


- **Elbow Joint**
  - **HumeroUlnar Joint:**
    - **Features:**
      - Joins Distal Humerus to Proximal Ulna
      - Synovial Hinge Joint
      - Very Stable – Due to Bony Congruency
    - **Bones:**
      - Humerus
        - Medial Epicondyle
        - Lateral Epicondyle
      - Ulna

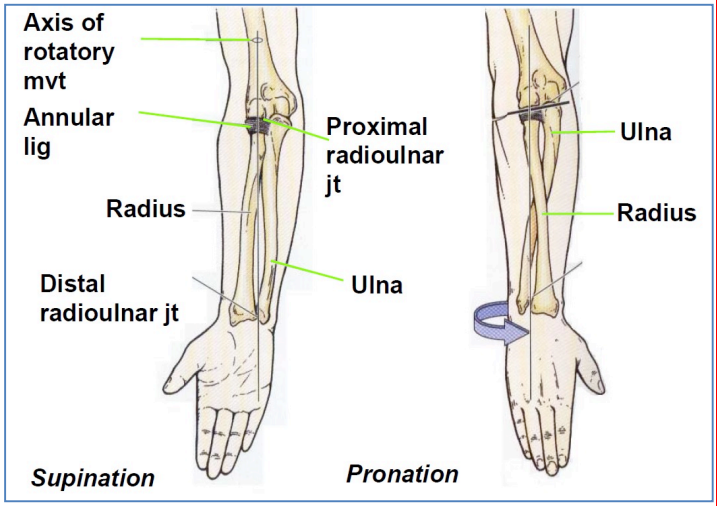


- **Cubital Fossa**

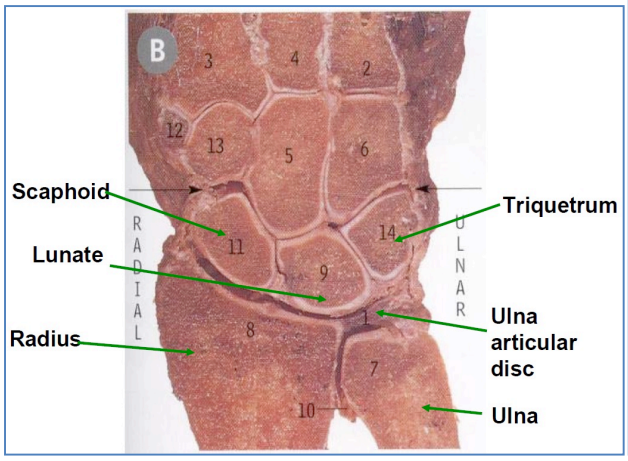


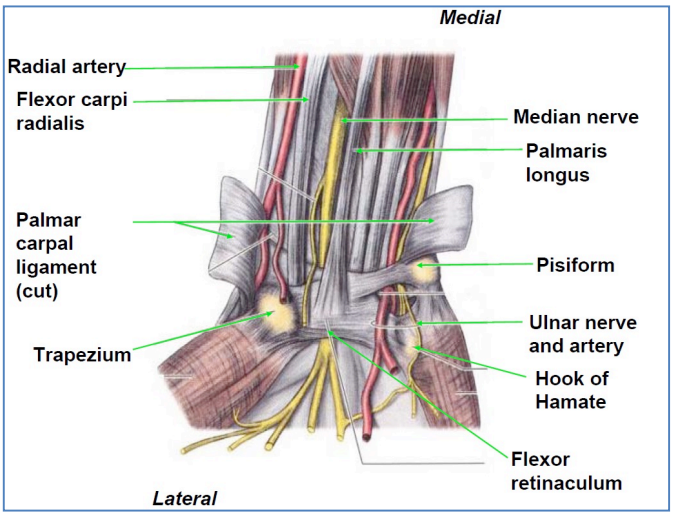


- **RadioUlnar Joint:**
  - **Proximal:**
    - **Features:**
      - Joins Radius & Ulna
      - Synovial Pivot Joint
  - **Distal:**
    - **Features:**
      - Joins Radius & Ulna
      - Synovial Pivot + Articular Disc

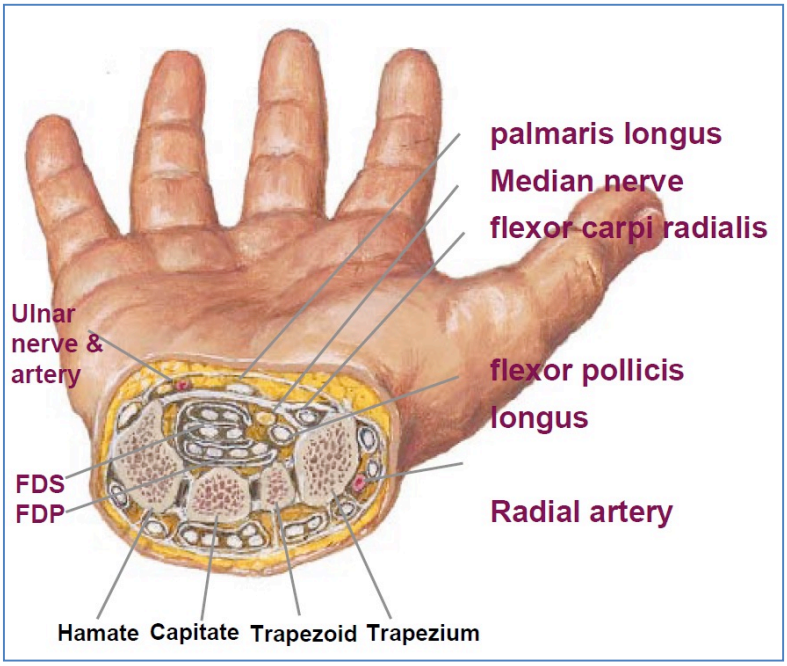


- **Wrist Joint:**
  - **CarpiRadialis Joint:**
    - **Features:**
      - Joins Radius & Proximal Carpals
    - **Bones:**
      - Radius
      - Proximal Carpals

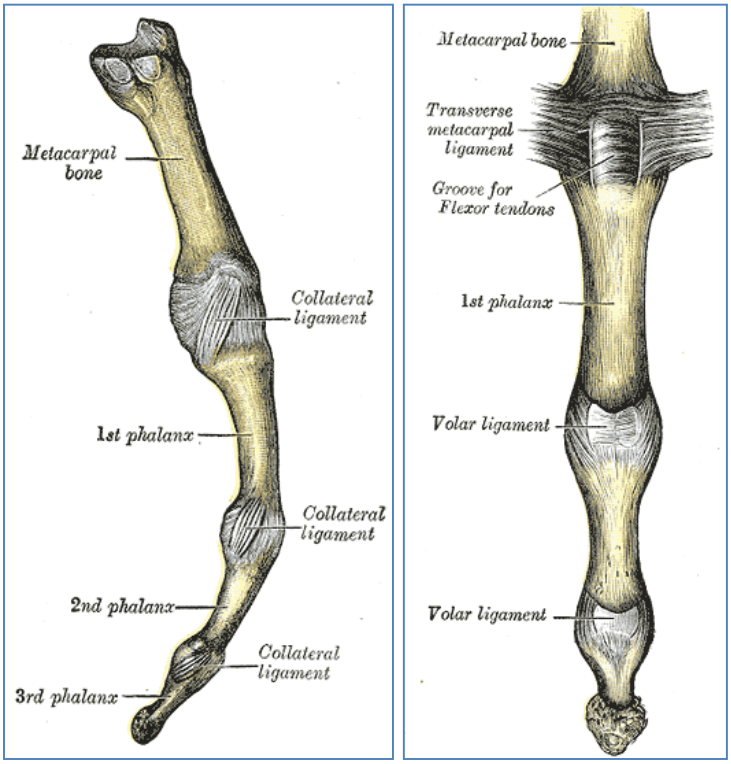




- **Carpal Tunnel (Anterior Aspect):**
  - Median Nerve
  - Wrist Flexor Tendons
  - Ulnar Artery
  - Ulnar Nerve

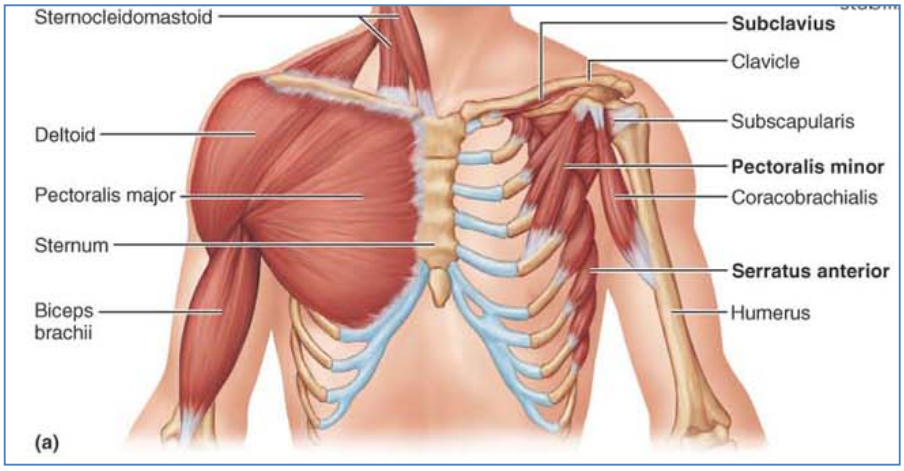


- **CarpoMetacarpal Joints:**
  - **Features:**
    - Joins Carpals & Metacarpals
- **Metacarpo-Phalangeal Joints (Knuckes):**
  - **Features:**
    - Joins Metacarpals & Phalanges
- **InterPhalangeal Joints (Fingers):**
  - **Features:**
    - Joins adjacent Phalanges

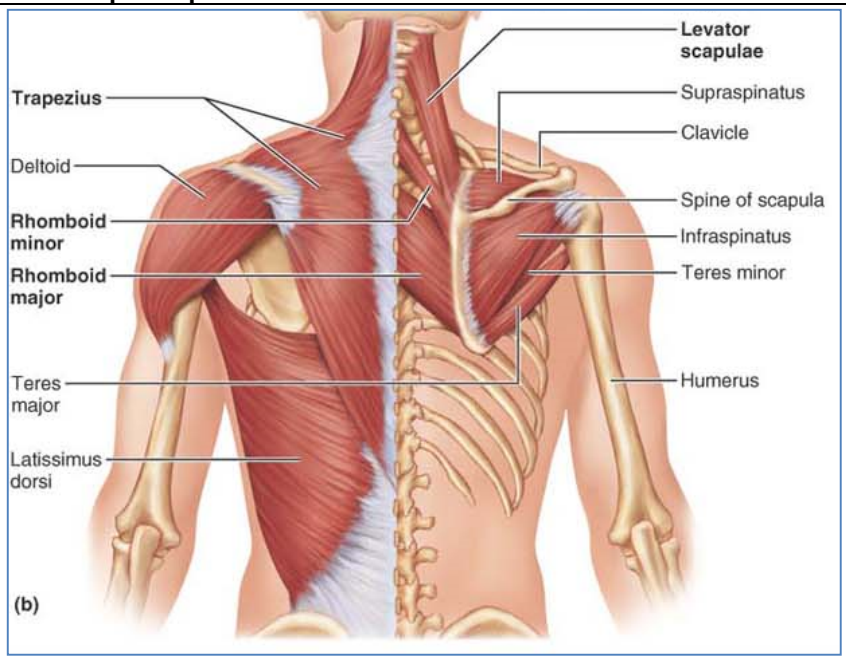


Muscles:

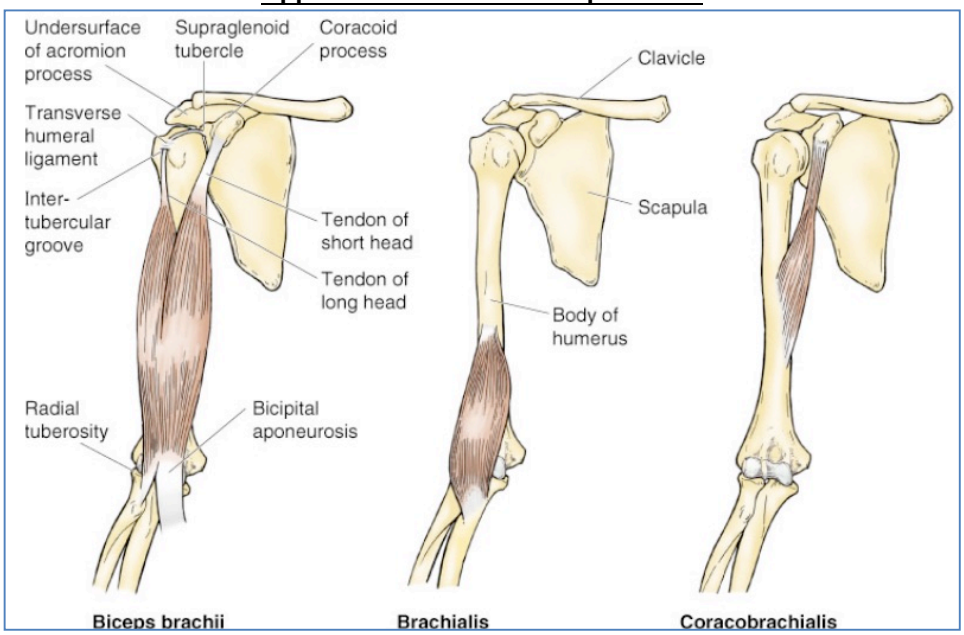
Anterior View: Deep & Superficial Muscles of The Pectoral Girdle & The Glenohumeral Joint



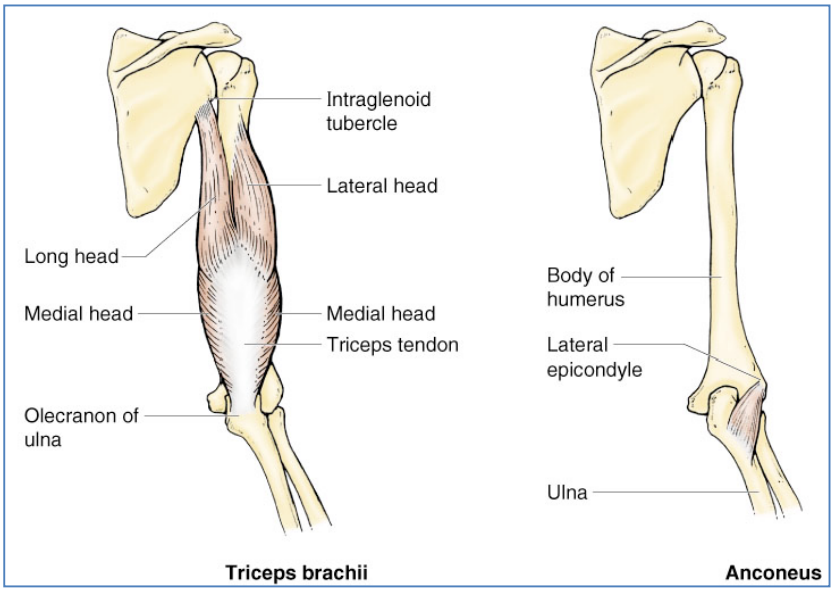
Posterior View: Deep & Superficial Movers of The Pectoral Girdle & The Glenohumeral Joint



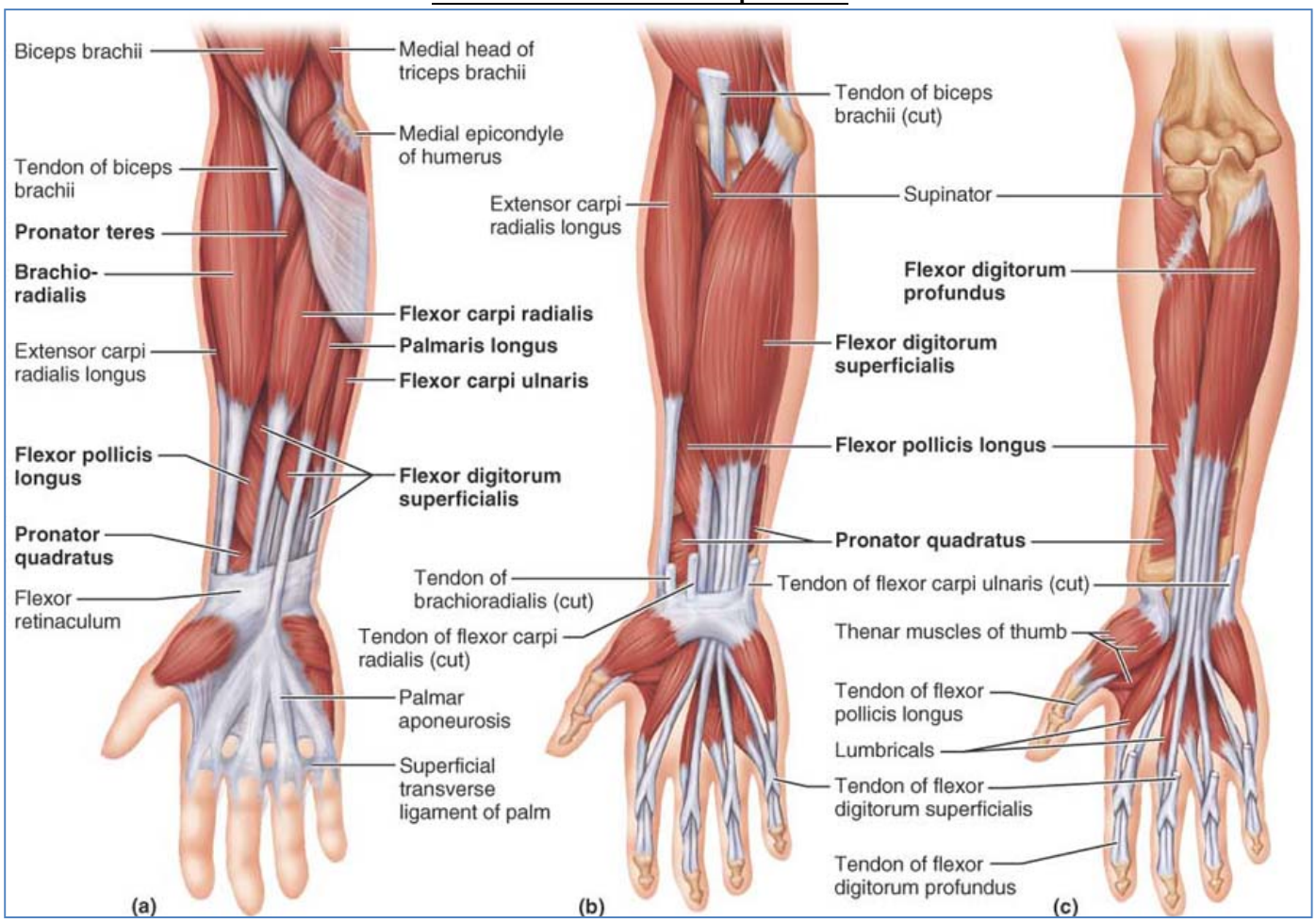
Upper Arm – Anterior Compartment



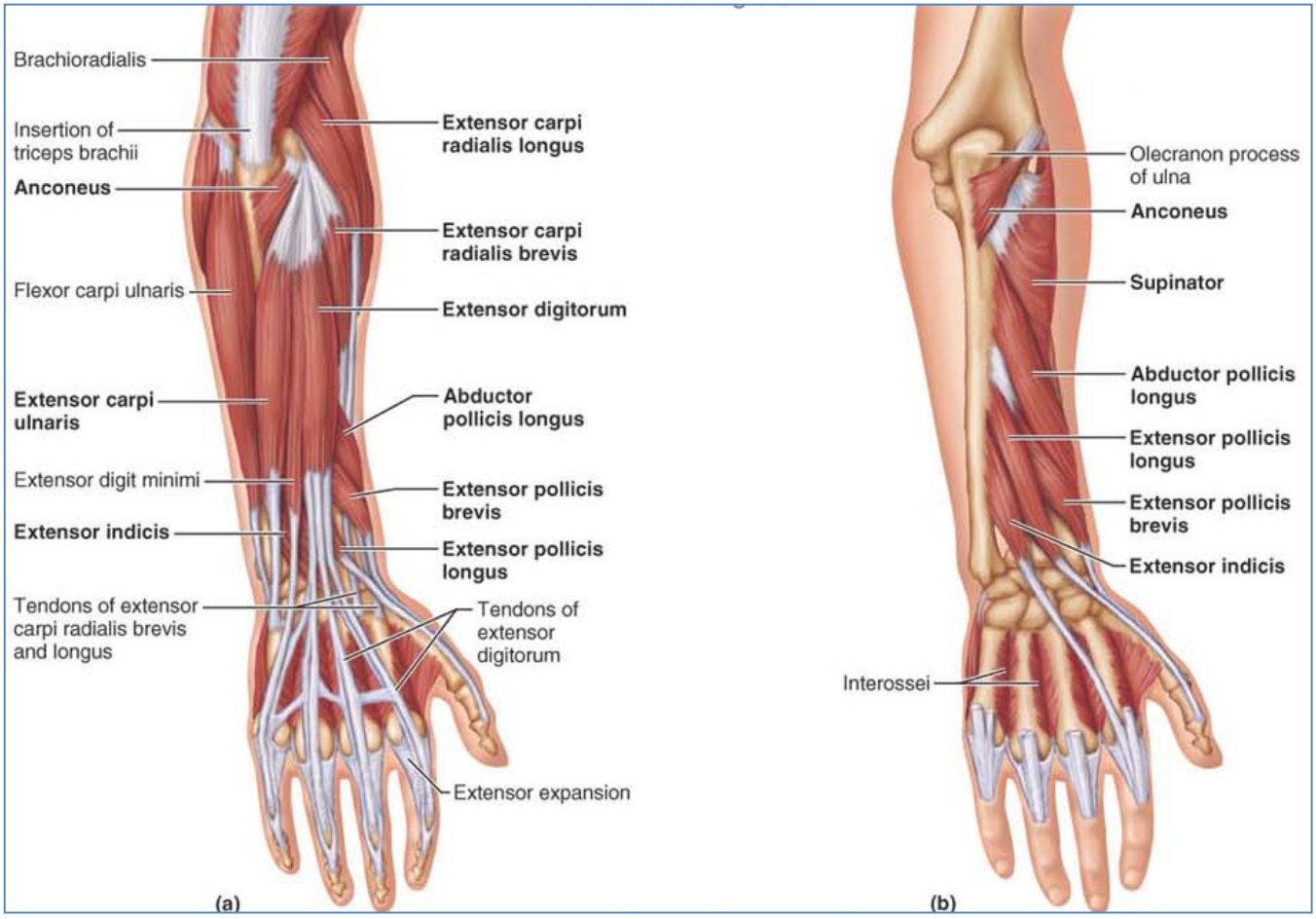
Upper Arm – Posterior Compartment



Lower Arm – Anterior Compartment

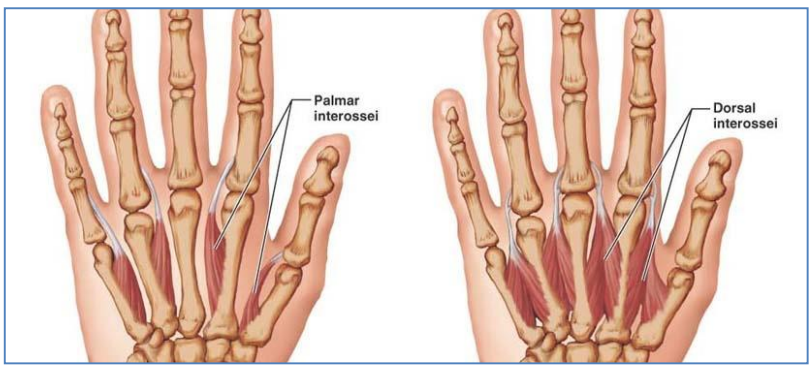


Lower Arm – Posterior Compartment

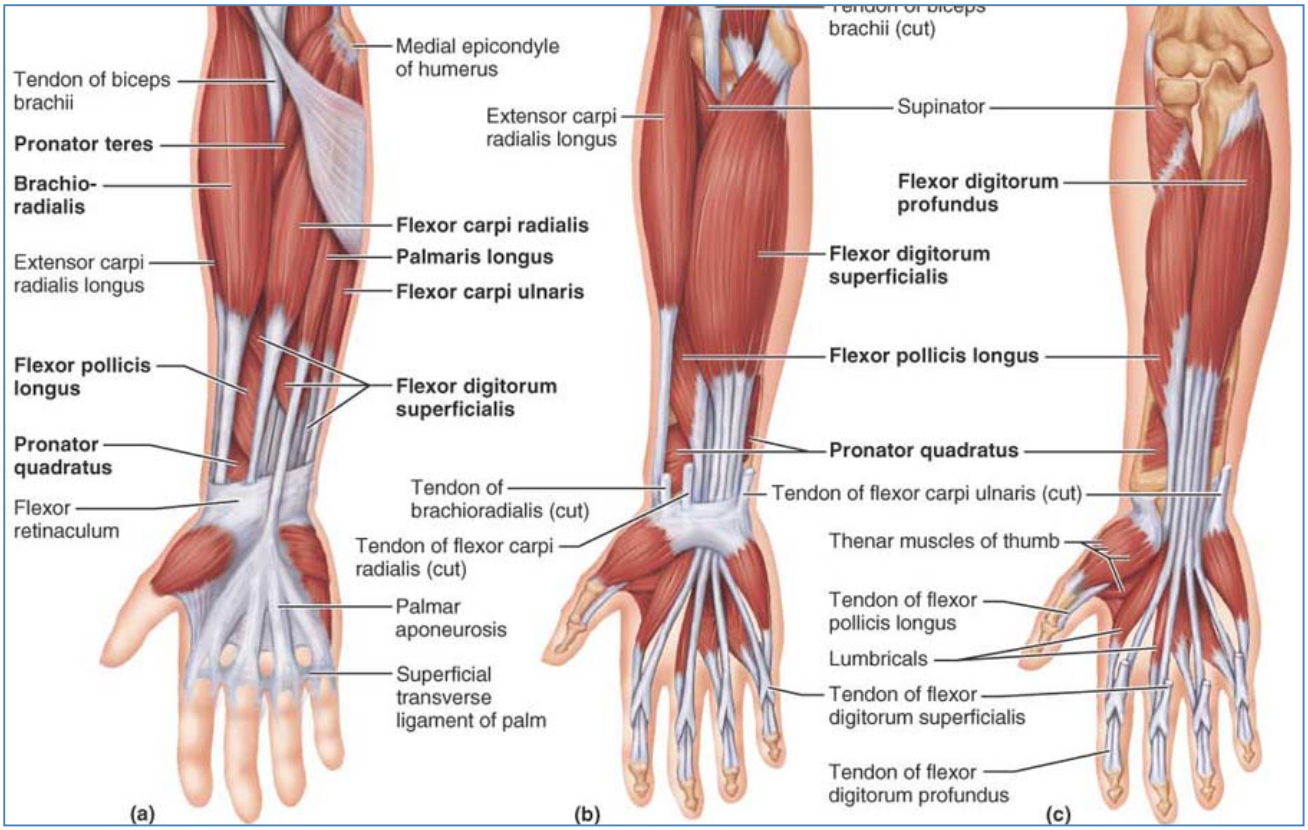


The Hand:

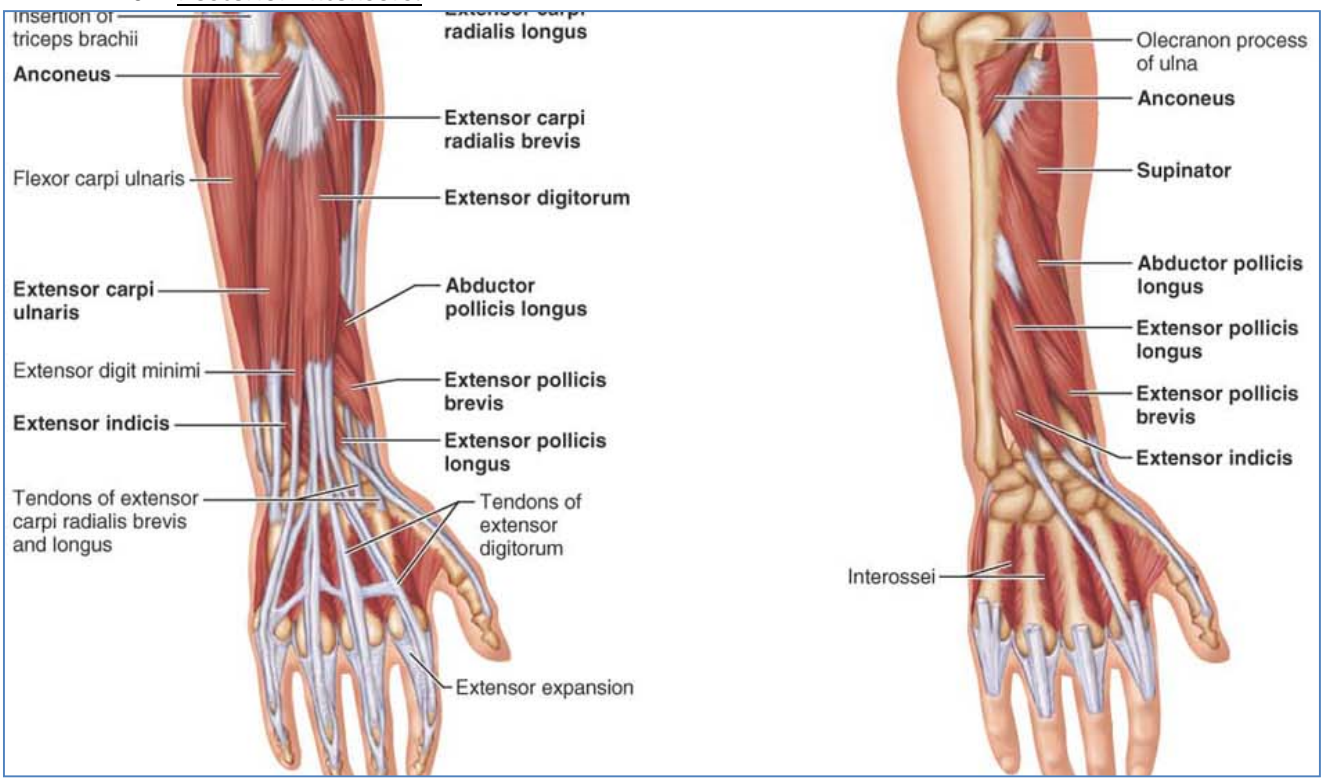
- **Intrinsic Muscles of the Hand:**
  - **Thenar Muscles:**
    - "Ball" of the Thumb.
    - **Median Nerve** (Except *Adductor Pollicis = Ulnar Nerve)
  - **Hypothenar Muscles:**
    - "Ball" of the Little Finger
    - **Ulnar Nerve**
  - **Lumbricals:**
    - 4 worm-shaped muscles in the Palm
    - One to each finger (except thumb)
    - **Median Nerve** (Lateral 2) & **Ulnar Nerve** (Medial 2)
  - **Interossei:**
    - **Palmar Interossei: (PAD – Palmar Adduct Fingers)**
      - **Ulnar Nerve**
    - **Dorsal Interossei: (DAB – Dorsal Abduct Fingers)**
      - **Ulnar Nerve**



- **Extrinsic Muscles of the Hand:**
  - Insert via Long Tendons from the Forearm
  - **Anterior Flexors:**

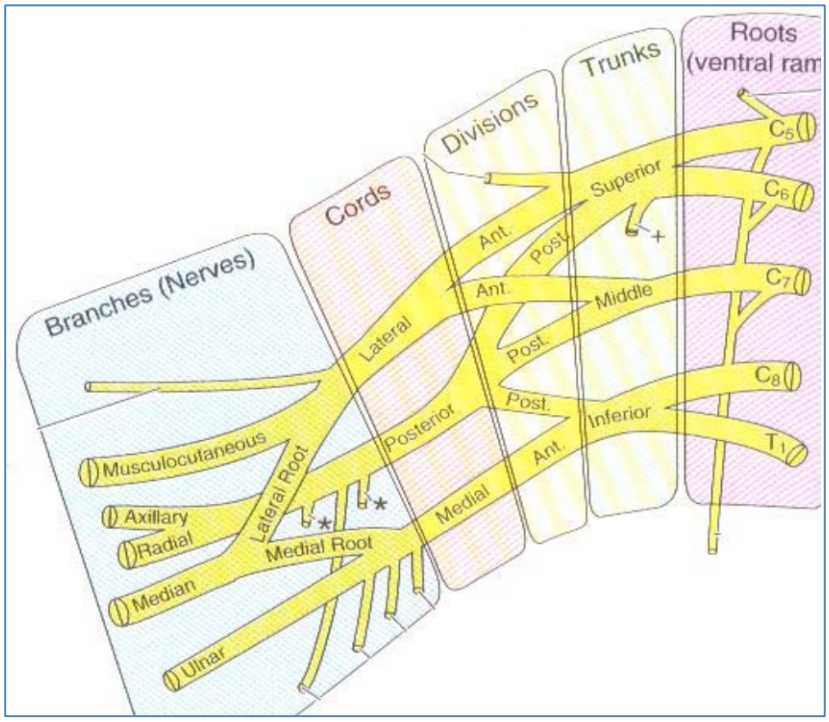


  - **Posterior Extensors:**

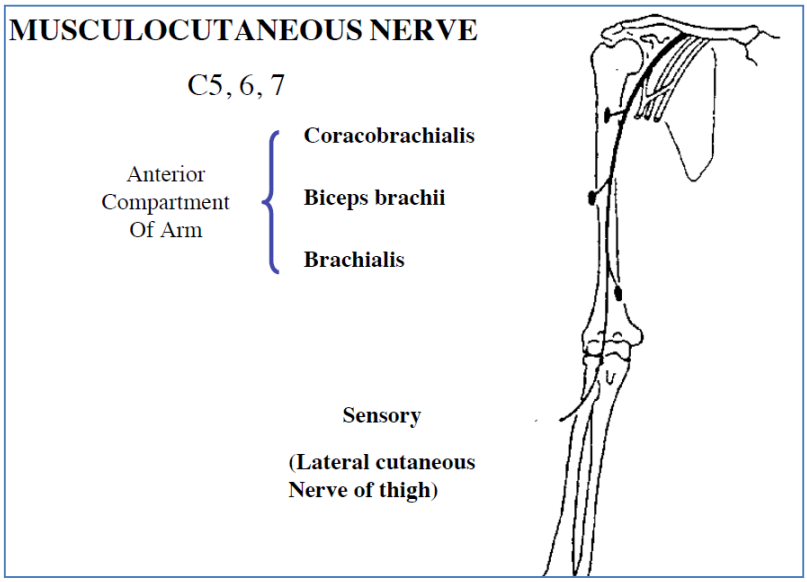


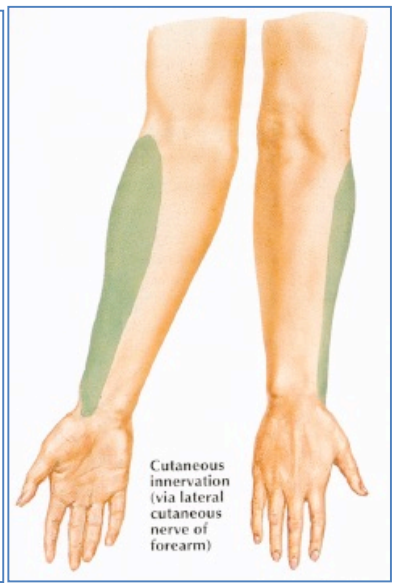
Innervation (Peripheral)**:**

- **Axilla (Brachial Plexus)**
  - **Roots (Ventral Rami)**
    - C5, C6, C7, C8, T1
  - **Terminal Branches (Nerves)**
    - Musculocutaneous
    - Median
    - Ulnar
    - Radial
    - Axillary

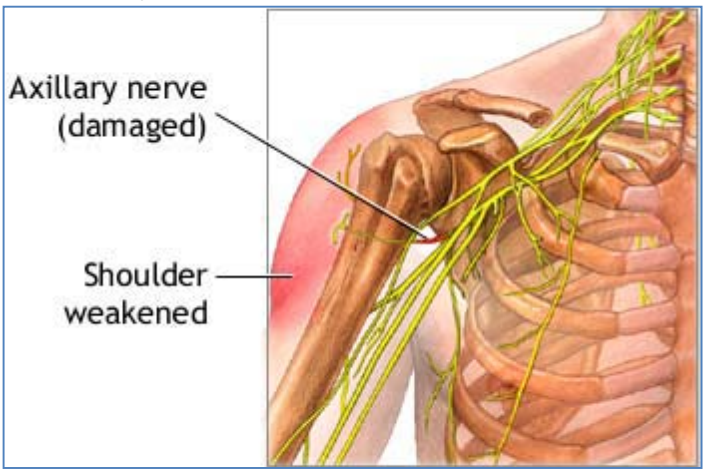


- **Musculocutaneous Nerve:**
  - Runs down Anterior Arm
  - Innnervates:
    - Flexors of Arm:
      - Biceps Brachii
      - Brachialis
      - Coracobrachialis
    - Skin of Anterio-Lateral Forearm

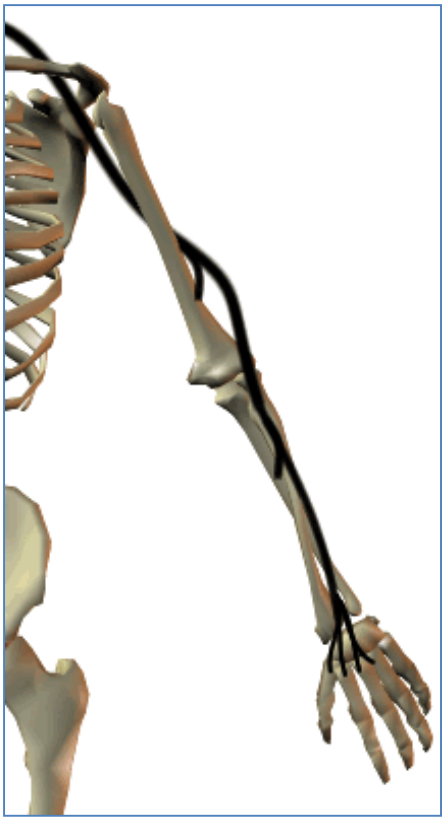


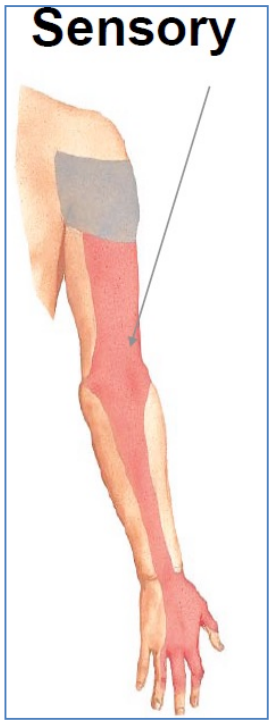


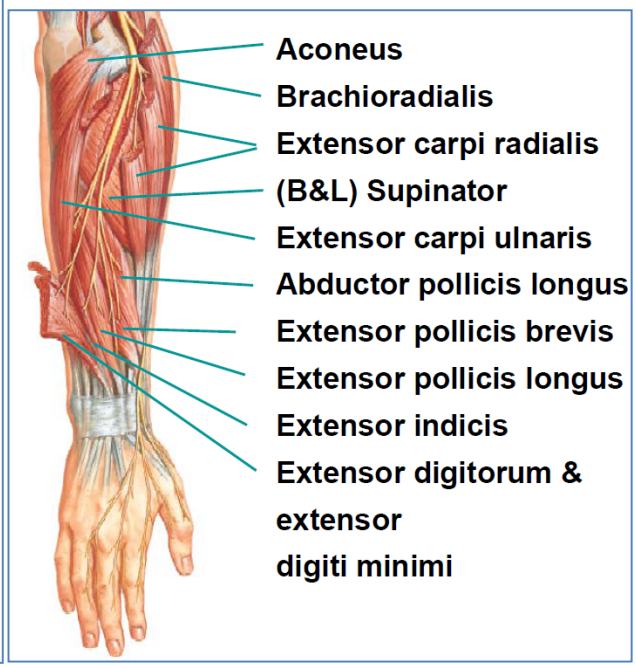
- **Axillary Nerve:**
  - Runs Posterior to Neck of Humerus
  - Innervates:
    - Deltoid
    - Teres Minor
    - Skin & Joint Capsule of Shoulder



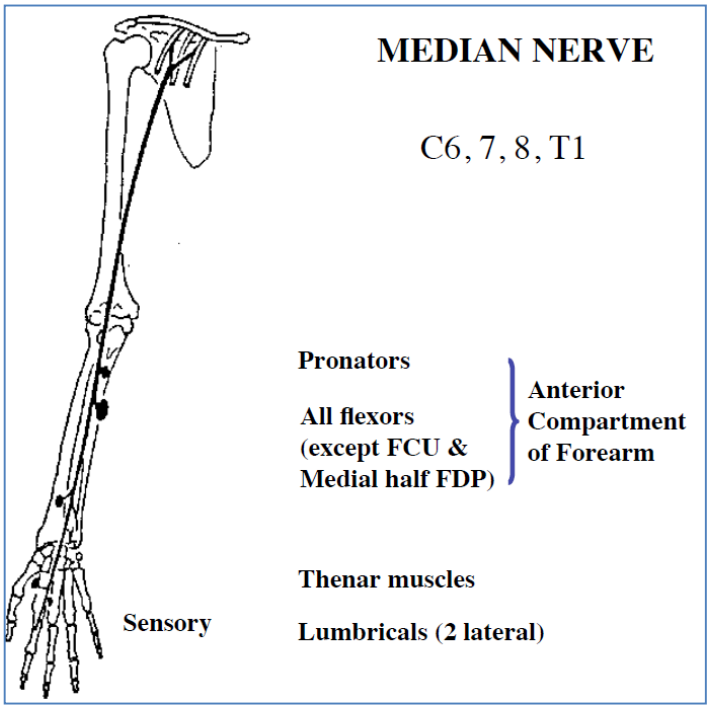
- **Radial Nerve:**
  - Runs around Posterior Humerus (Radial-groove)→Anteriorly around Lateral Epicondyle→2 Branches:
    - Superficial: Follows Lateral edge of Radius → Hand
    - Deep: Runs Posteriorly to Radius
  - Innervates:
    - ALL Posterior Extensor Muscles of Arm, Forearm & Hand
    - Skin of Entire Latero-Posterior Arm & Forearm & Hand (except dorsum of fingers 2 & 3)

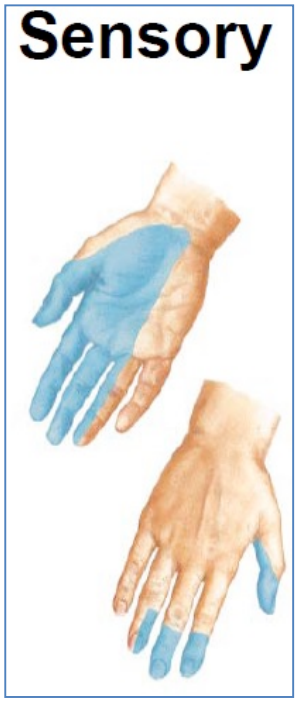




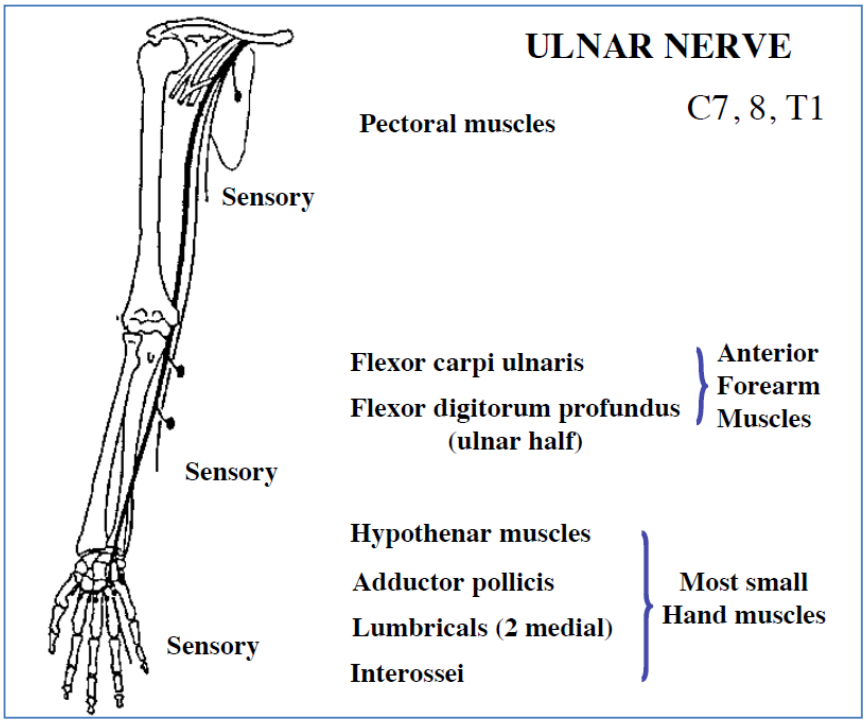


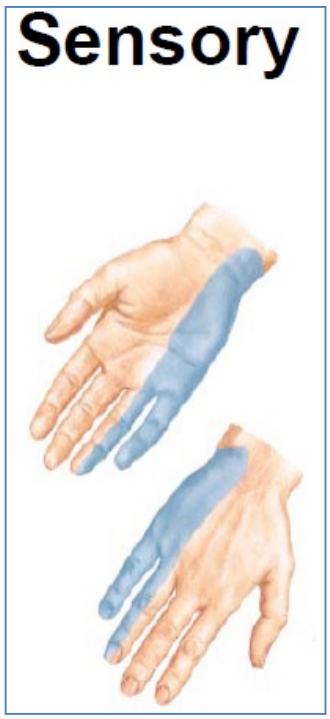
- **Median Nerve:**
  - Runs down Anterior Arm & Forearm
  - Innervates:
    - Flexors of Anterior Forearm
    - Thenar Muscles (Intrinsic muscles of Lateral Palm)
    - Lumbricals #1 & #2
    - Skin of Lateral 2/3 of Hand, Palm Side & Dorsum of Fingers 2 & 3





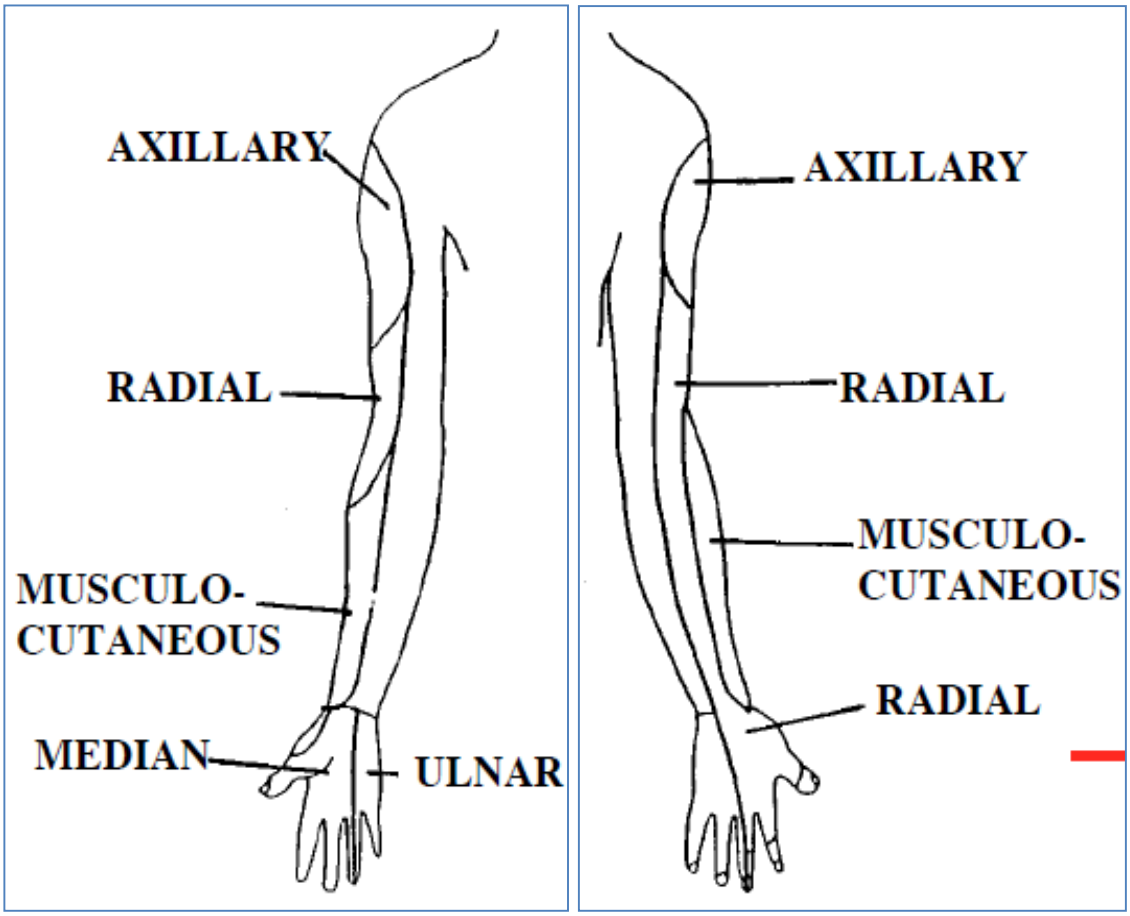
- **Ulnar Nerve:**
  - Runs down Medial Arm→Behind Medial Epicondyle→Follows Ulna down Medial Forearm→Hand
  - Innervates:
    - Flexors of Anterior Forearm
    - Majority of Intrinsic Muscles of Hand
      - Incl. Lumricals #3 & #4
      - Interossei
    - Skin of Medial 1/3 of Hand (Ant & Post).



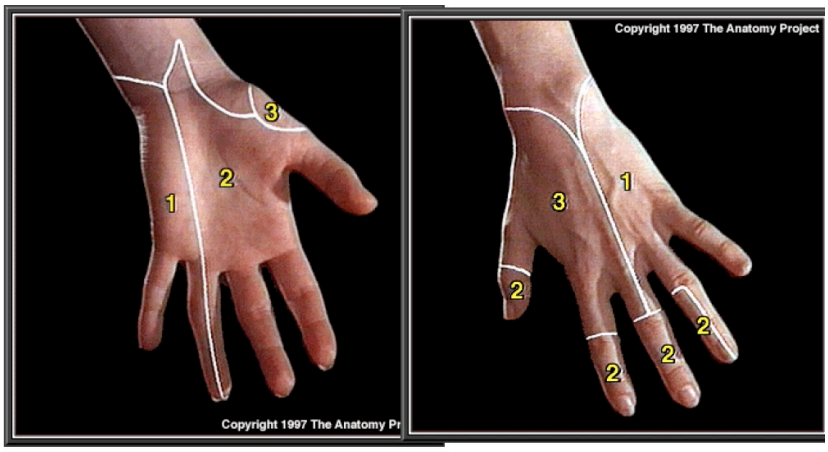


Dermatomes of Upper Limb: (CUTANEOUS INNERVATION – NOT MYOTOMES!!)

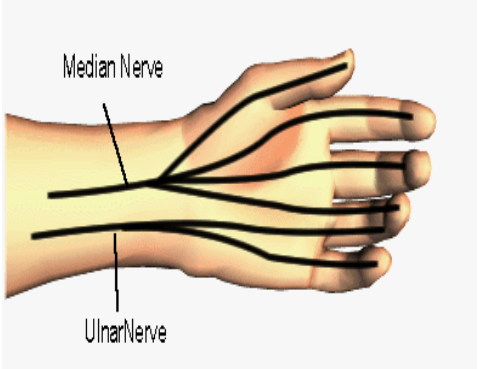
Anterior **Posterior**



- **Dermatomes of the Hand:**



1. Ulnar; 2. Median; 3. Radial



Nerve Lesions:

- **Afferent: Sensory Loss**
  - Sensory impulses don't reach spinal cord
  - Loss of cutaneous sensation
  - Loss of spinal reflexes
  - Weird sensations – tingling, itching, pain, etc.
- **Efferent: Paralysis**
  - If impulse can't reach muscle.
  - Muscle becomes flaccid – no tone.
  - Complete/Incomplete
  - Loss of spinal reflexes

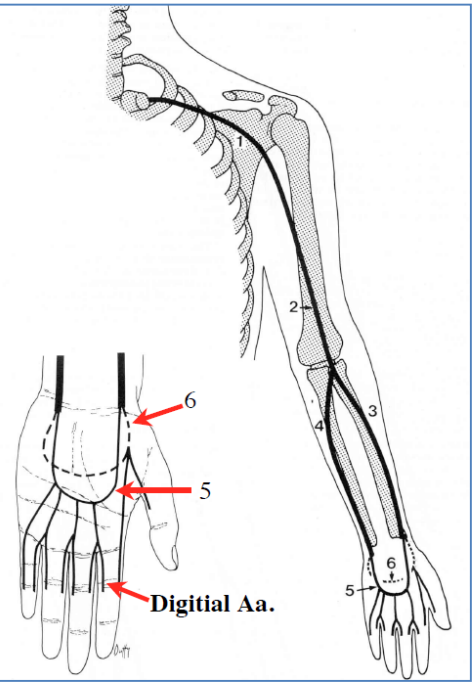
Nerve	Site of Injury	Paralysis	Motor Loss	Sensory Loss
Axillary	Axilla	Deltoid	Shoulder Abduction	Deltoid Region
Musculocutaneous	Axilla	Arm Flexors	Forearm Flexion	Lateral Forearm
Radial	Axilla	Arm Extensors + Supinator	Elbow Extension, Supination, Wrist Extension	Lateral Dorsum of Hand
	Cubital Fossa	Arm Extensors, *Except* Triceps	Supination, Wrist Extension	
Median	Elbow	Wrist Flexors, Thenar Muscles, Lateral 2x Lumbricals	Weak Wrist Flexion, Thumb Opposition, Lateral 2x Finger-Flexion	Lateral 3.5 Fingers
	Wrist	Thenar Muscles, Lateral 2x Lumbricals	Thumb Opposition, Lateral 2x Finger-Flexion	
Ulnar	Above Elbow	Wrist Flexors, Hypothenar Muscles, Medial 2x Lumbricals	Weak Wrist Flexion, Medial 2x Finter-Flexion	Palm, Medial 1.5 Fingers
	At Wrist	Hypothenar Muscles, Medial 2x Lumbricals	Medial 2x Finter-Flexion	

Arterial Blood Supply:

ARTERIES OF UPPER LIMB

- Summary

1. Axillary
2. Brachial
3. Radial
4. Ulnar
5. Superficial palmar arch
6. Deep palmar arch

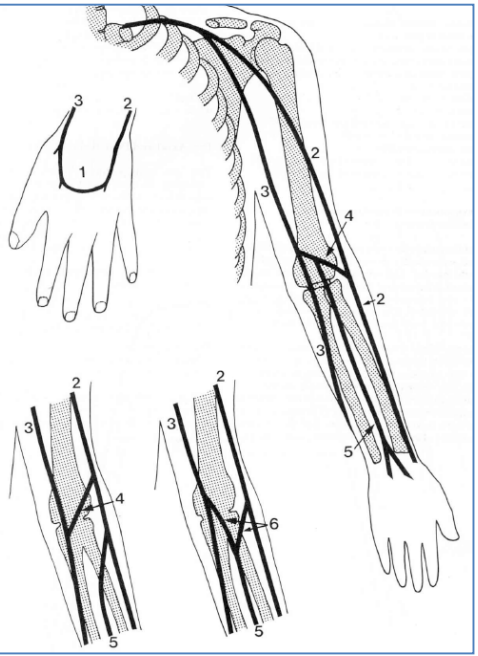


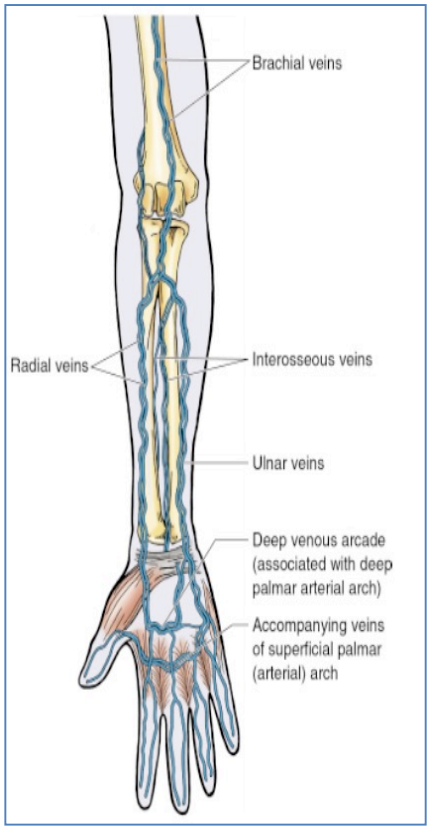
Venous Blood Drainage:

- Deep & Superficial
- Deep Veins run in pairs clinging to Arteries.
- **Hand, Forearm & Upper Arm**

- Summary

1. Dorsal venous arch
2. Cephalic vein
3. Basilic vein
4. Median cubital
5. Median antebrachial
6. Median cephalic & median basilic



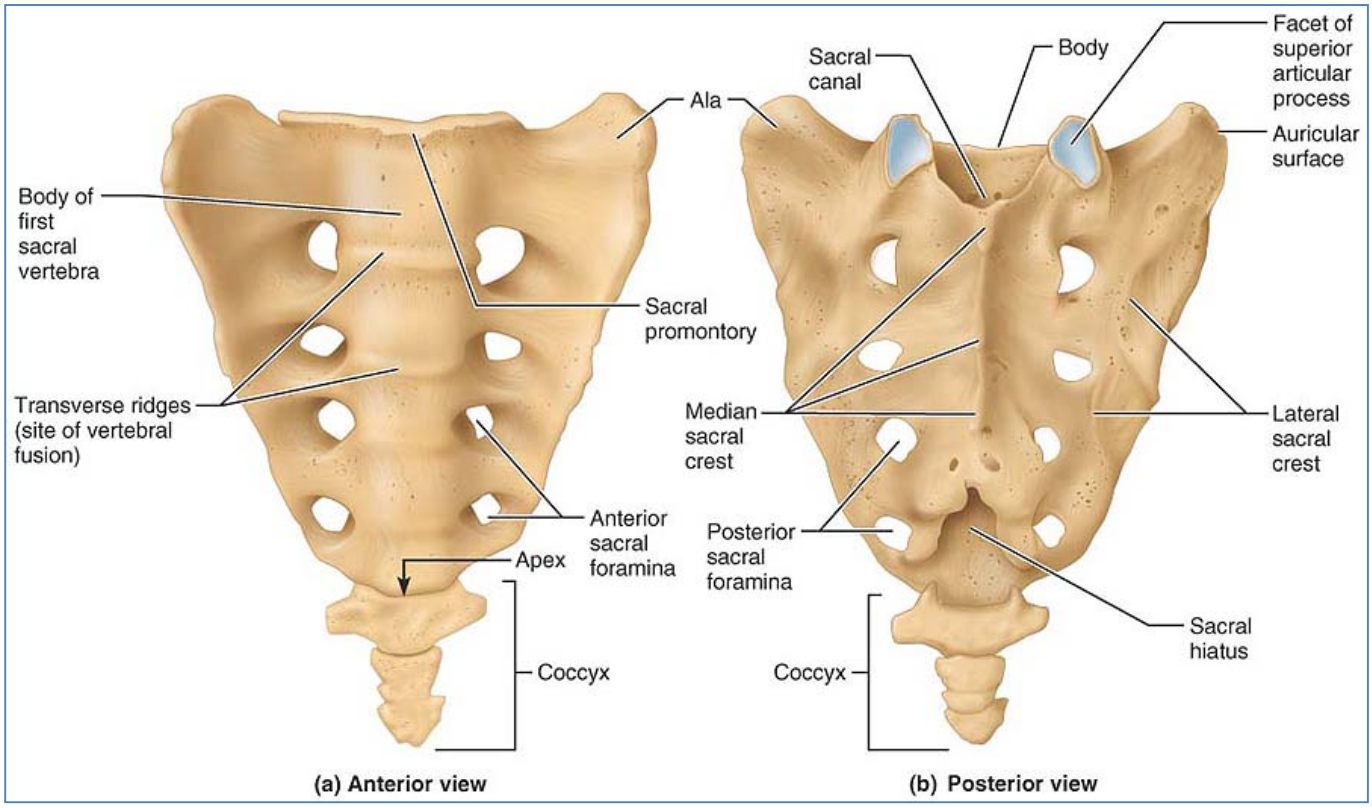


- **Axilla:**
  - Axillary Vein →
  - Subclavian Vein
- **Thoracic:**
  - Subclavian Vein→
  - BrachioCephalic →
  - Superior Vena Cava →Heart

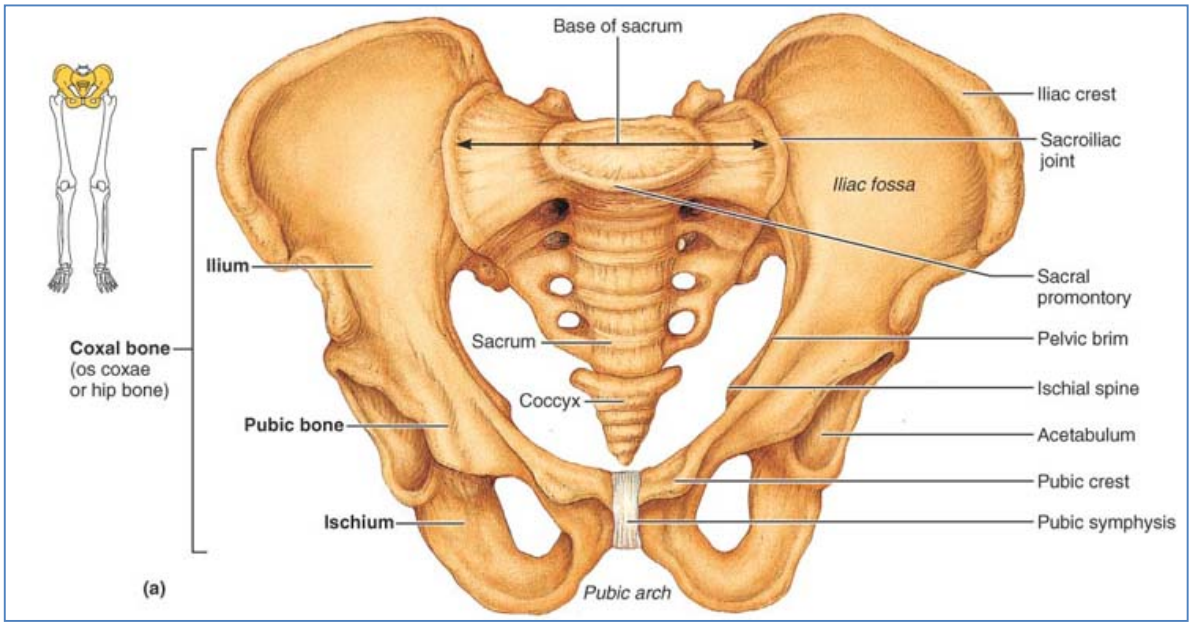
The Pelvic Girdle & The Lower Limb: Bones, Joints, Muscles, Nerves & Vasculation

Bones:

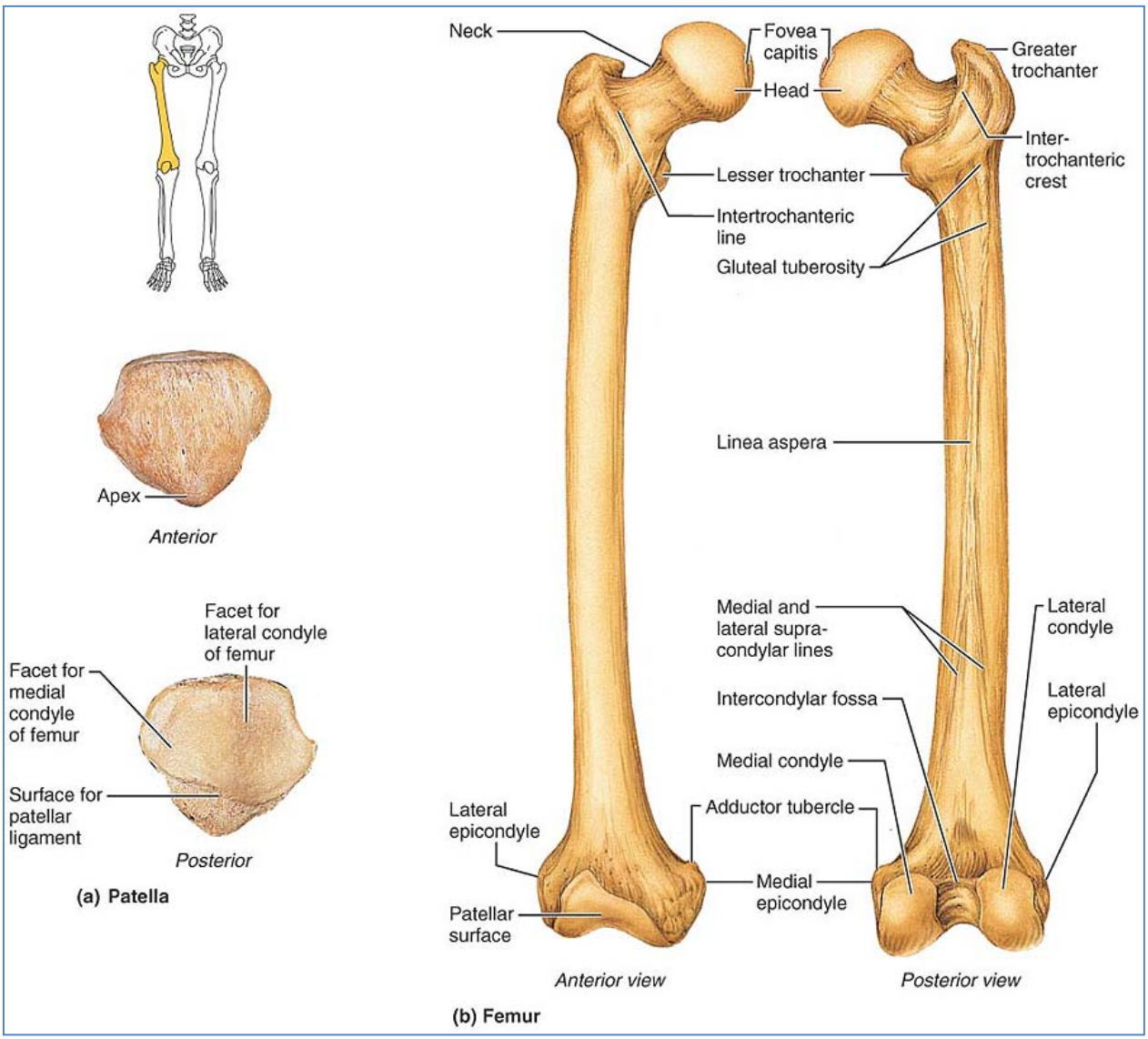
- **The "Bony Pelvis":**
  - **Sacrum:**



  - **Coxal Bones (hip bones):**
    - **Landmarks:**
      - Acetabulum ("Wine Cup") – Hemispherical Socket
      - Pelvic Brim
      - **Ilium**
        - Iliac Crest
      - **Ischium**
      - **Pubis**
        - Pubic symphysis
        - Pubic Arch/Subpubic Angle (Wide in females)

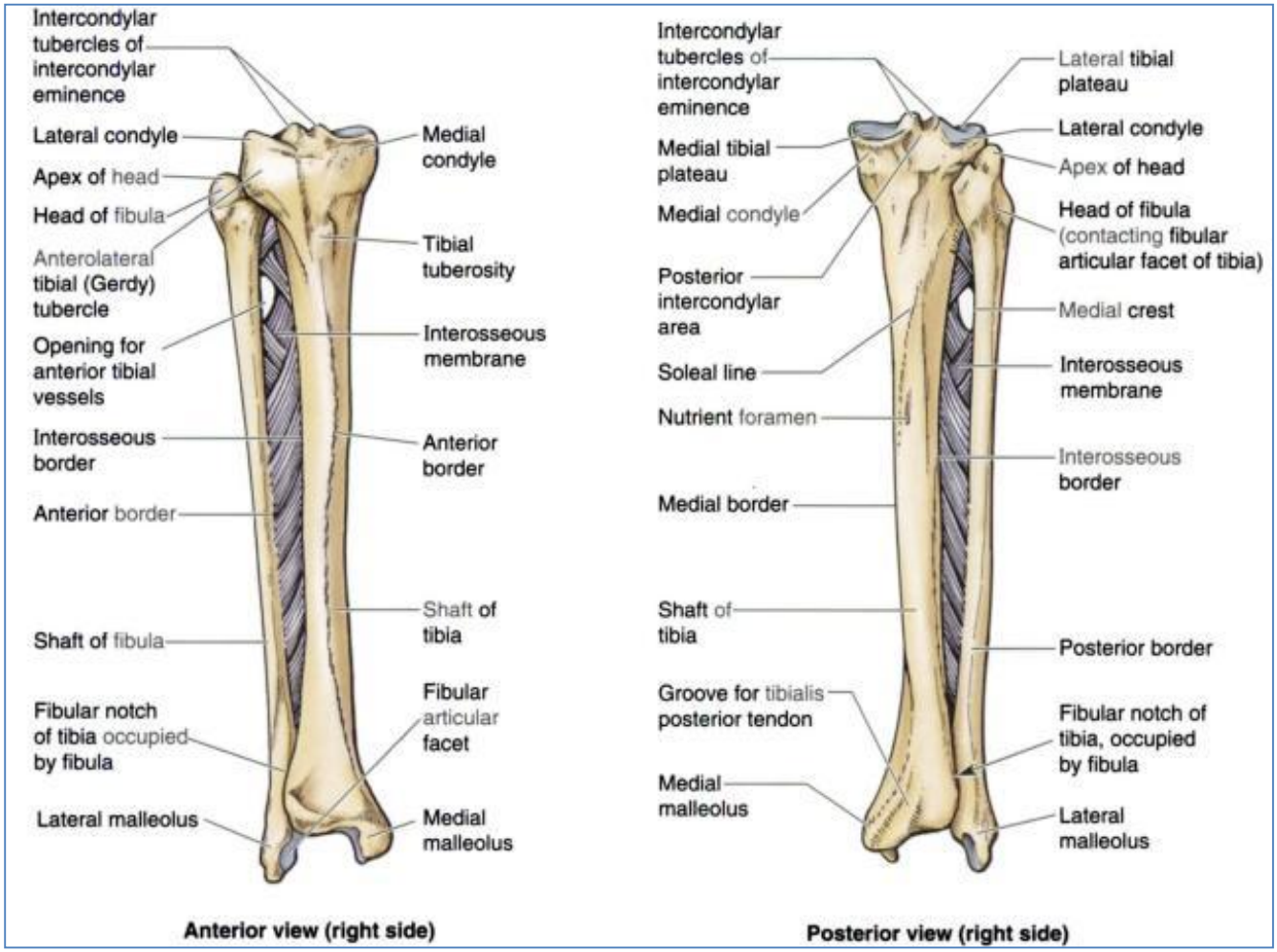


- **Femur:**
  - **Type/Features:**
    - Long bone
    - Neck of femur – prone to fracture due to lack of trabeculae.
  - **Landmarks:**
    - Head
    - Neck
    - Greater Trochanter
    - Lesser Trochanter
    - Lateral Epicondyle
    - Medial Epicondyle
    - Lateral Condyle
    - Medial Condyle
    - Patellar Surface
  - **Articulations:**
    - Acetabulum of the Coxal Bones of the Hip
    - Patella
    - Tibia

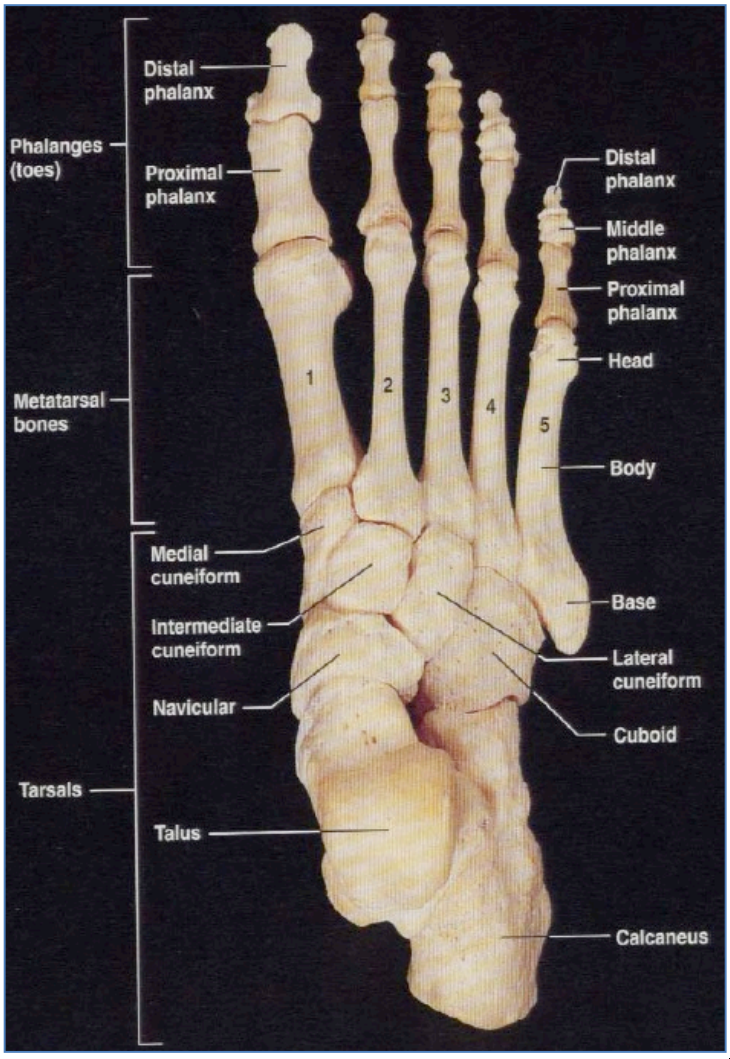


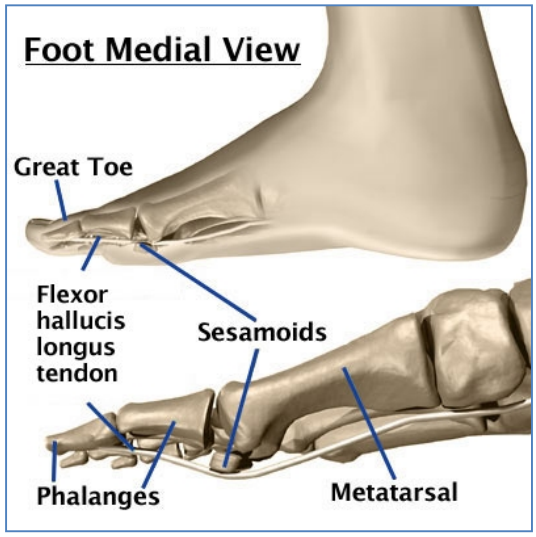
- **Patella**
  - **Type/Features:**
    - Triangular Sesamoid Bone
    - Enclosed in Quadriceps Tendon

- **Tibia**
  - **Type/Features:**
    - Long Bone
    - Weight Bearing (not fibula)
  - **Landmarks:**
    - Condyles – Medial & Lateral
    - Tibial Plateau – (superior articular surface)
    - Tibial Tuberosity
    - Medial Malleolus
  - **Articulations:**
    - Condyles of Femur
    - Fibula – Fibular Facet (proximally)
    - Talus Bone of the Foot.
    - Fibula – Fibular Notch (distally)
- **Fibula**
  - **Type/Features:**
    - Slender Long Bone
    - No function in weight-bearing – mainly for muscle attachment
  - **Landmarks:**
    - Head
    - Neck
    - Shaft
    - Lateral Malleolus
  - **Articulations:**
    - Tibia Proximally & Distally
    - Trochlea of Talus Bone of Tarsals of the Foot.



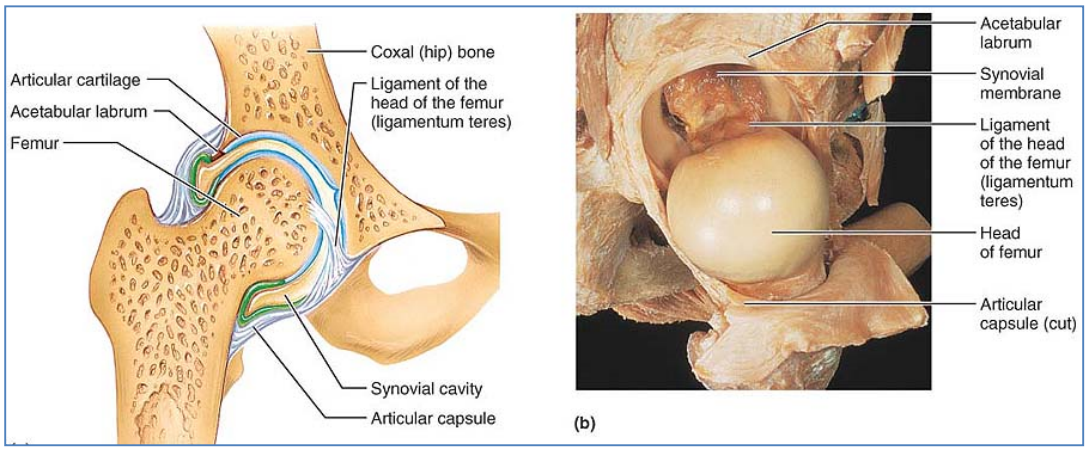
- **Foot:**
  - **7x Tarsals:**
    - **Talus**
    - **Calcaneus**
    - **Navicular**
    - **Cuboid**
    - **Lateral Cuneiform**
    - **Intermediate Cuneiform**
    - **Medial Cuneiform**
  - **5x MetaTarsals:**
    - **1 → 5**
  - **Phalanges:**
    - **1: Proximal & Distal**
    - **2→5: Proximal, Middle & Distal**
  - **2x Sesamoids:**
    - **"Ball" of the foot.**





Joints:

- **Hip Joints:**
  - **Features:**
    - Synovial, MultiAxial Ball & Socket
    - Acetabular Labrum (lip) of fibrocartilage – deepens socket – High Bony Congruency
  - **Bones:**
    - Rounded head of Femur
    - Acetabulum of Innominate Bones.

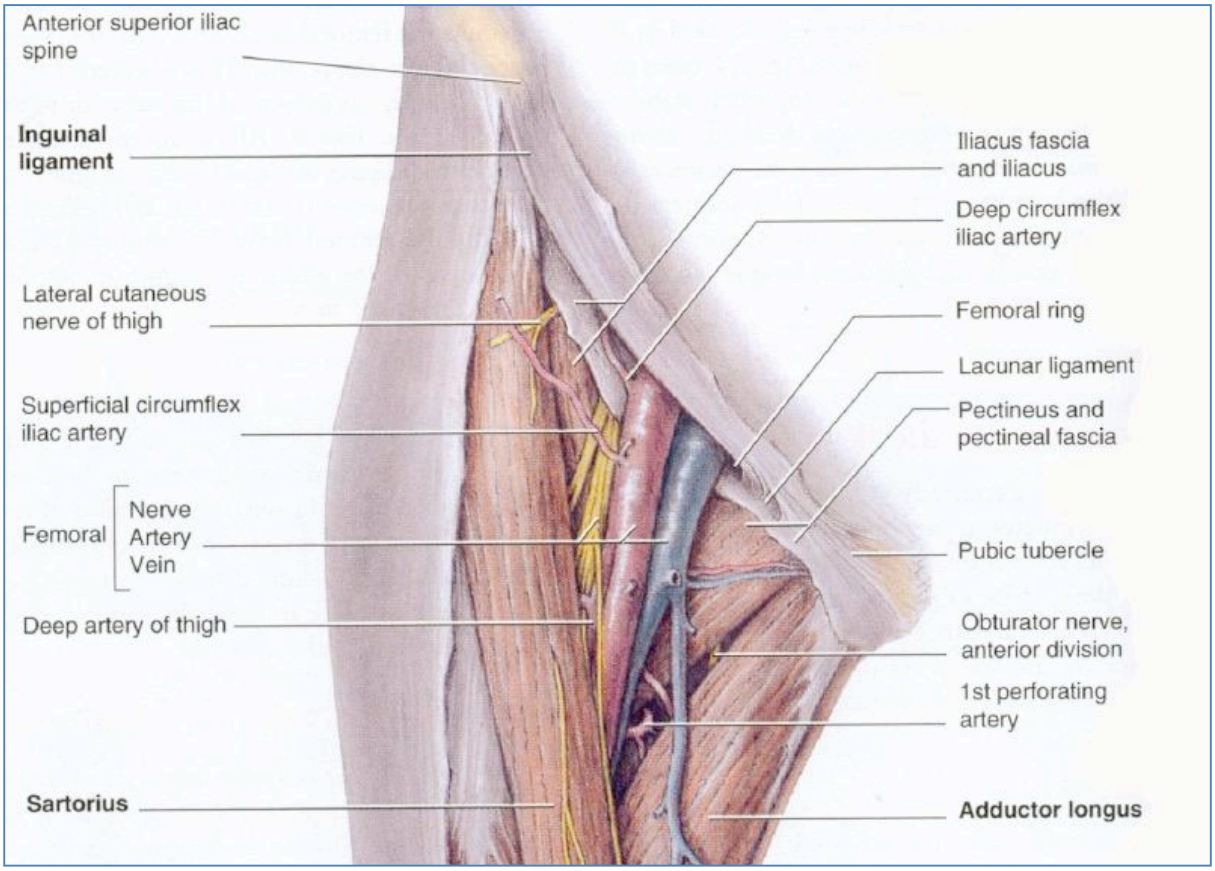


The "Femoral Triangle"

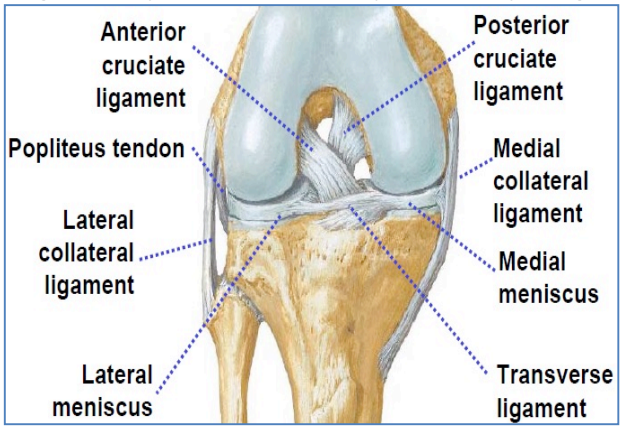
Contents:

- Femoral **N**erve
- Femoral **A**rtery
- Femoral **V**ein
- **E**mpty Space
- Inguinal **L**ymphatics

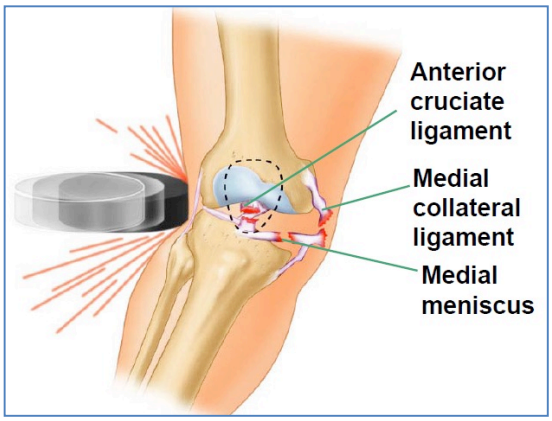
"NAVEL"



- **Knee Joint:**
  - **Features of The Knee Joint:**
    - Synovial Modified Bicondylar **Hinge** Joint
    - **Relatively Unstable:**
      - Ligaments provide the stability – not Bony Congruity.

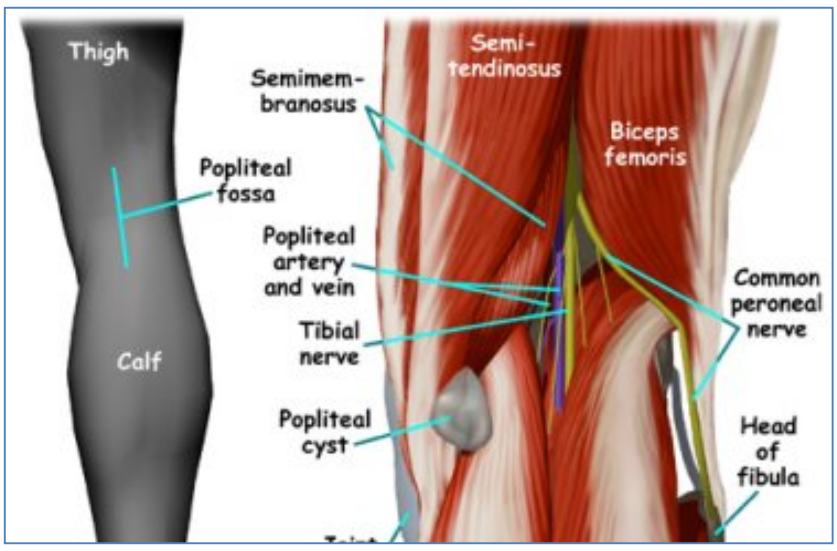


  - **Bones:**
    - Femur
    - Patella
    - Tibia
  - **Injury: The Unhappy Triad:**
    - Anterior Cruciate Ligament
    - Medial Collateral Ligament
    - Medial Meniscus

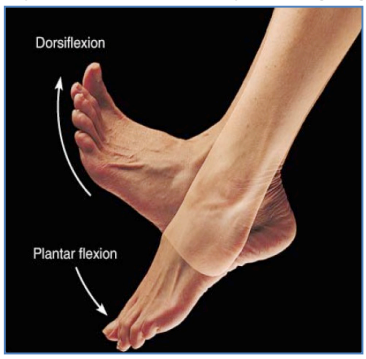


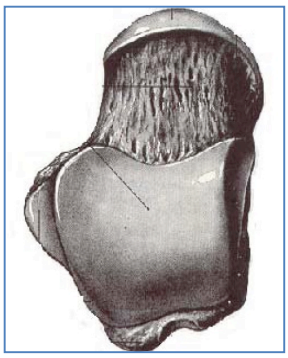
The Popliteal Fossa:

- **Contents:**
  - Popliteal Artery
  - Popliteal Vein
  - Tibial Nerve
  - Common Fibular Nerve



- **<u>Ankle Joint (Talo-Crural):</u>**
  - Ie. “Talus-Leg”
  - **Features:**
    - Synovial Hinge Joint
    - Good bony congruity
    - Stability comes from Very Strong Ligaments

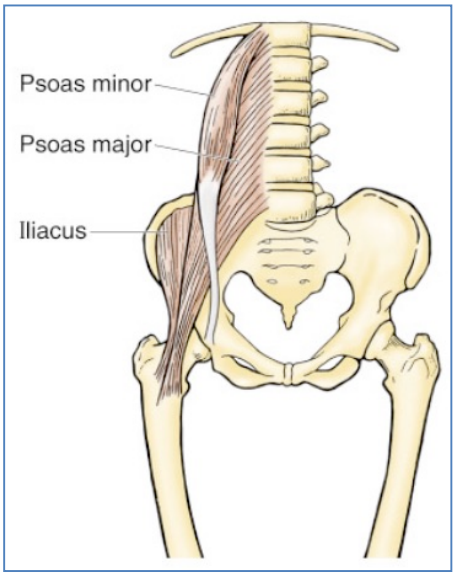




  - **Bones:**
    - Trochlea of Talus
    - Distal end of Tibia
    - Distal end of Fibula

Muscles:

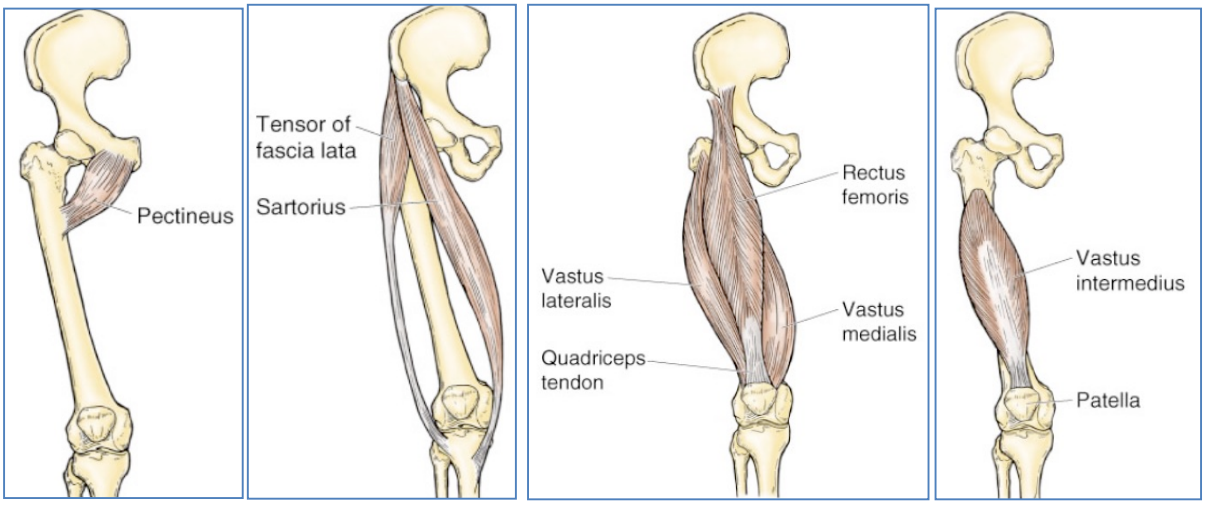
Iliac Region



Iliopsoas: Iliacus, Psoas Major, Psoas Minor

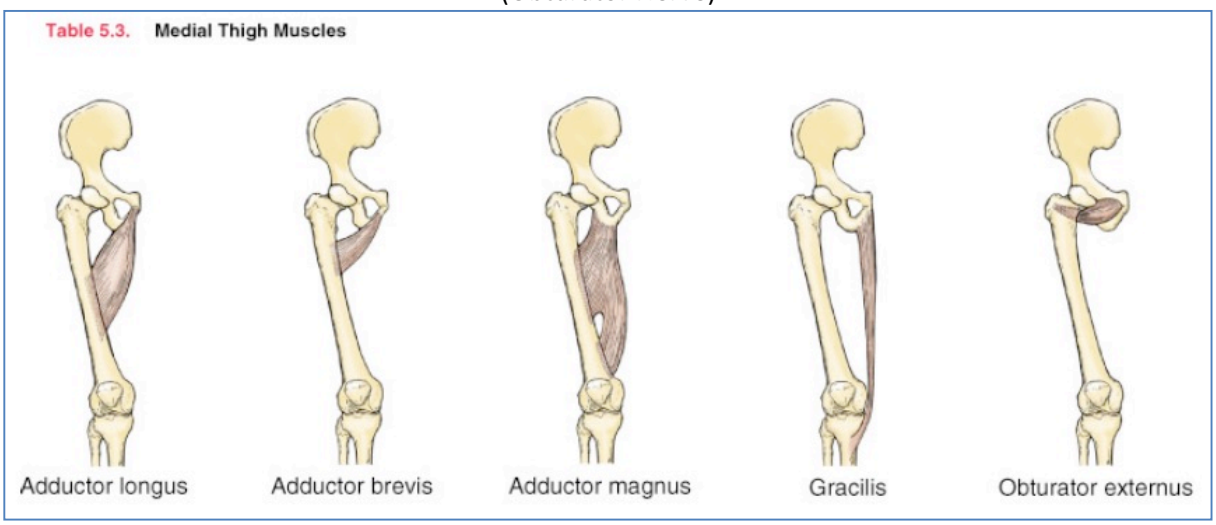
Thigh: Anterior:

(Femoral Nerve)



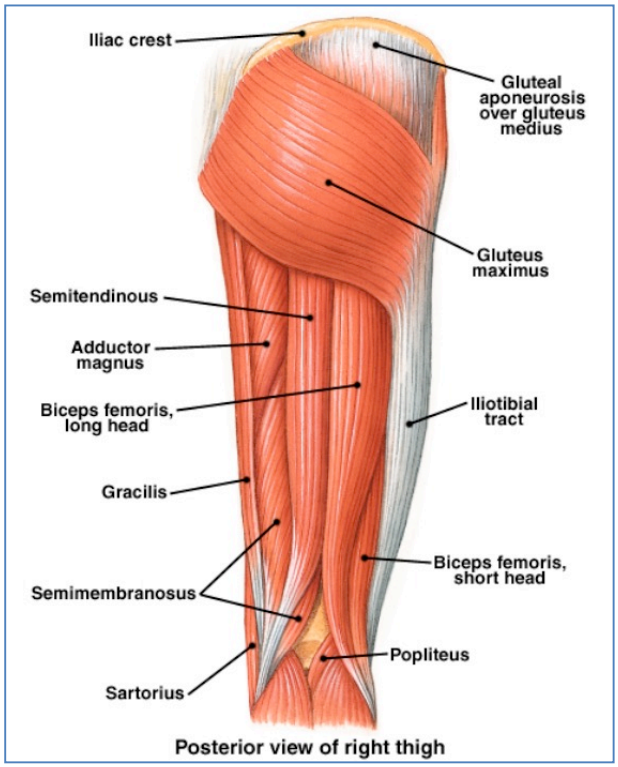
Thigh Medial:

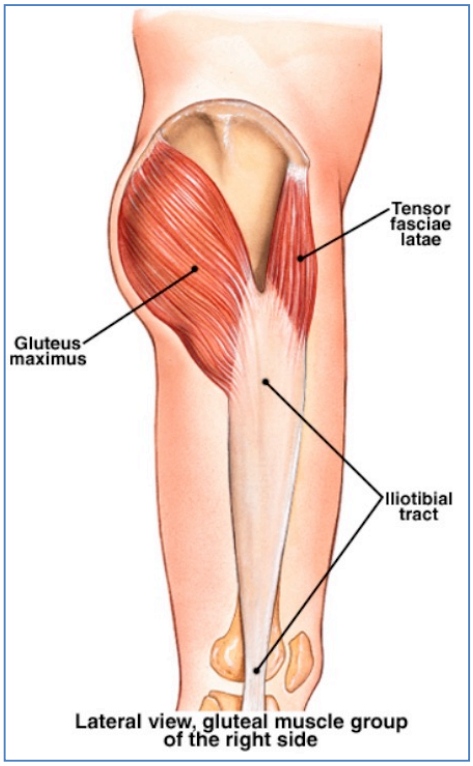
(Obturator Nerve)



Thigh Posterior & Lateral:

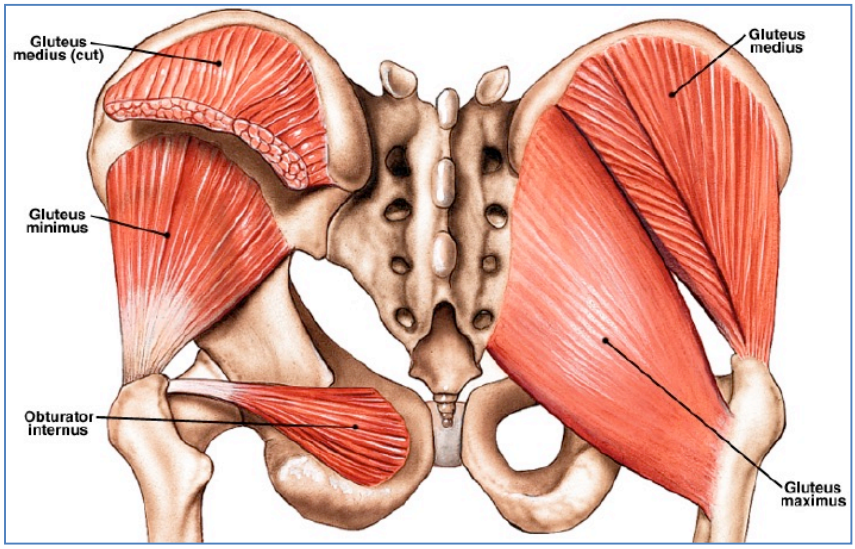
(Sciatic Nerve)





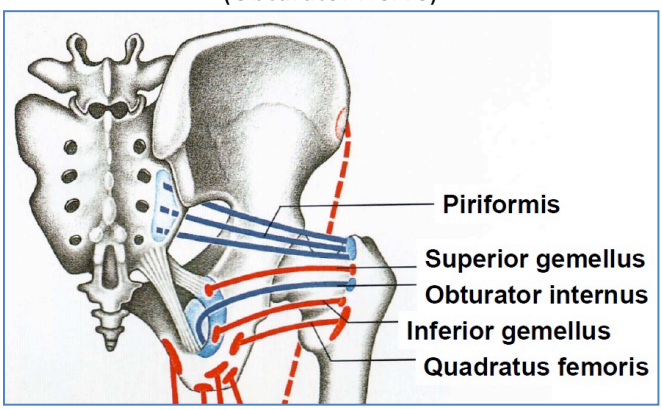
Gluteal Region:

(Sciatic Nerve)



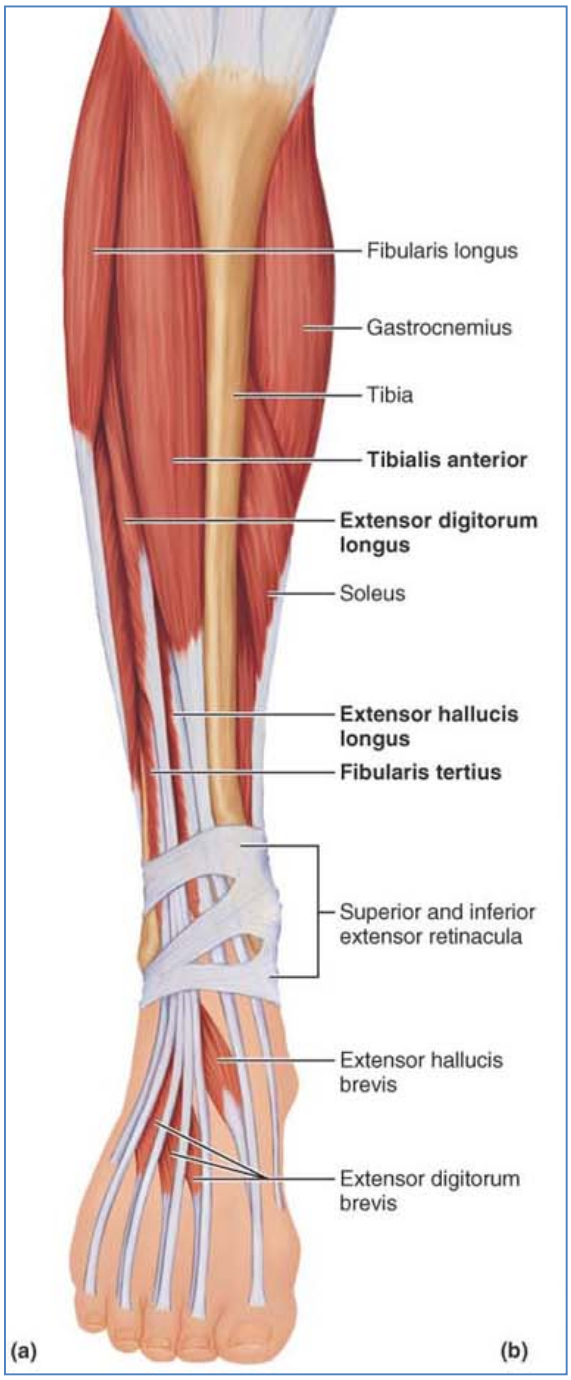
Lateral Rotators: Origins & Insertions: Posterior Aspect

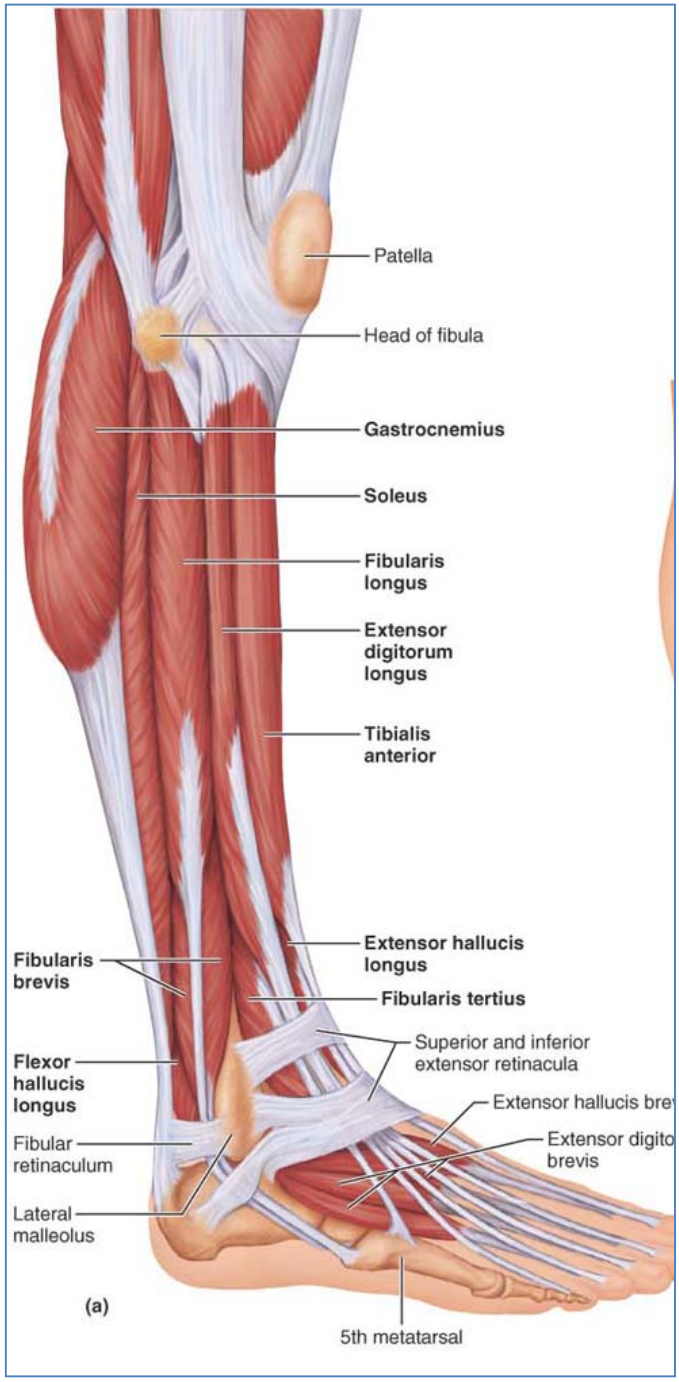
(Obturator Nerve)



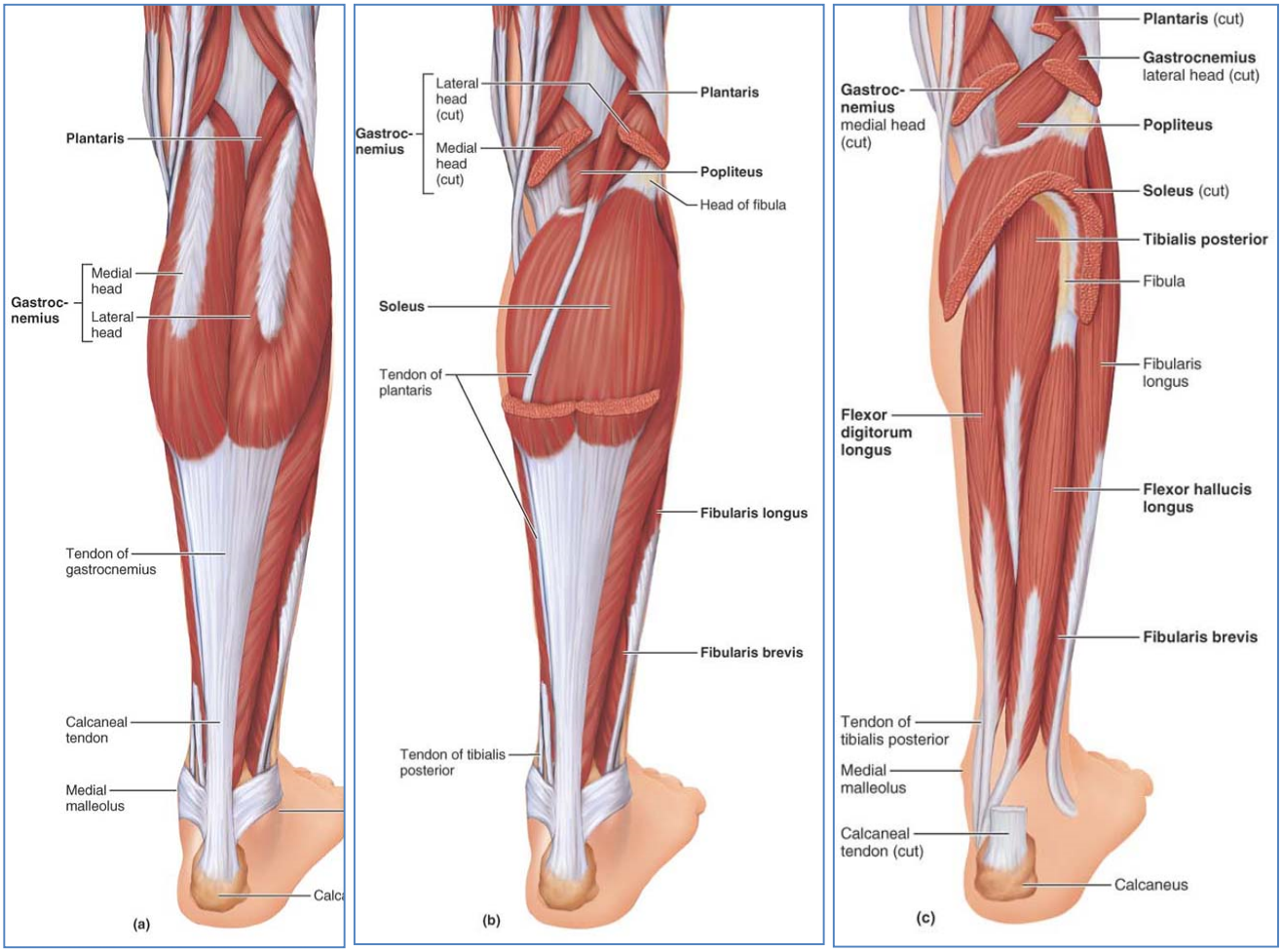
- **<u>Anterior Compartment of Leg:</u>** (Fibular Nerve)
  - **Tibialis Anterior:**
    - Dorsiflexion
  - **Extensor Hallucis Longus:**
    - Extension of Big Toe
  - **Extensor Digitorum Longus:**
    - Extension of Phalanges 2→5
  - **Fibularis Tertius:**
    - Dorsiflexion

- **<u>Lateral Compartment of Leg:</u>** (Fibular Nerve)
  - **Ankle Evertors:**
    - Ankle Eversion



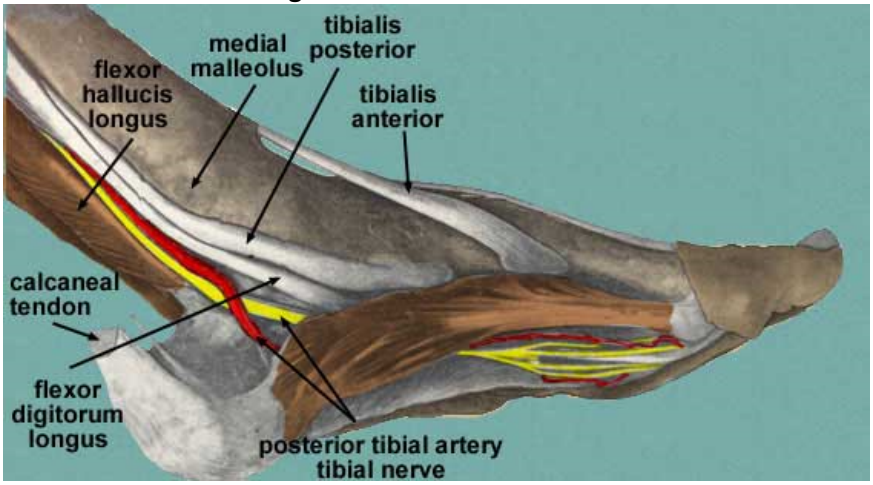


- **<u>Posterior Compartment of Leg:</u>** (Tibial Nerve)
  - **Triceps Surae (Calf):**
    - **Gastrocnemius & Soleus.**
      - Plantar Flexion
  - **& Other Plantar-Flexors:**
    - Plantar Flexion



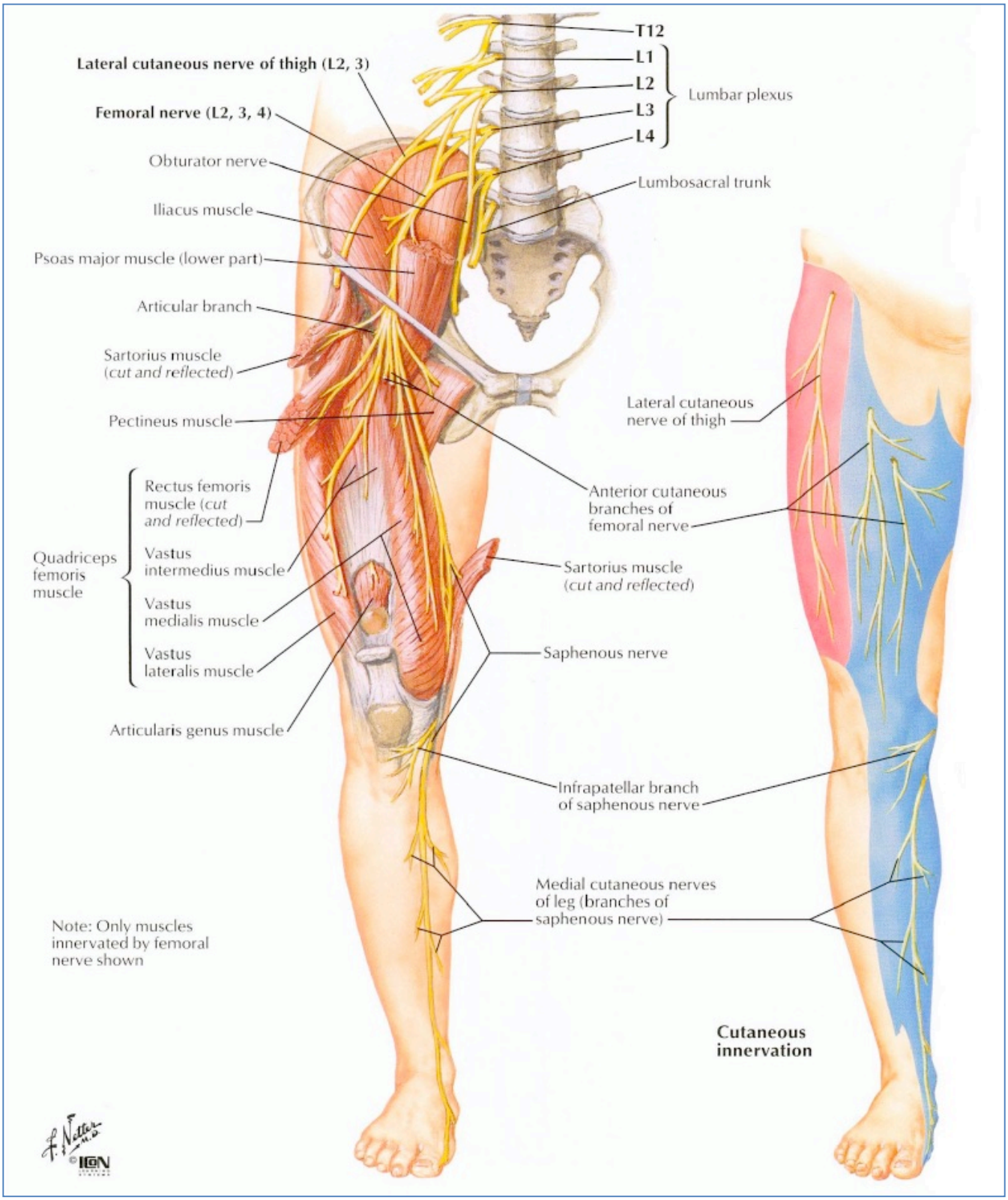
<u>Structures Passing Medial Malleolus:</u>

- **Tom** - **Tibialis Posterior**
- **Dick** - **Flexor Digitorum Longus**
- **And** - **Posterior Tibial Artery**
- **Naughty** - **Tibial Nerve**
- **Harry** - **Flexor Hallucis Longus**



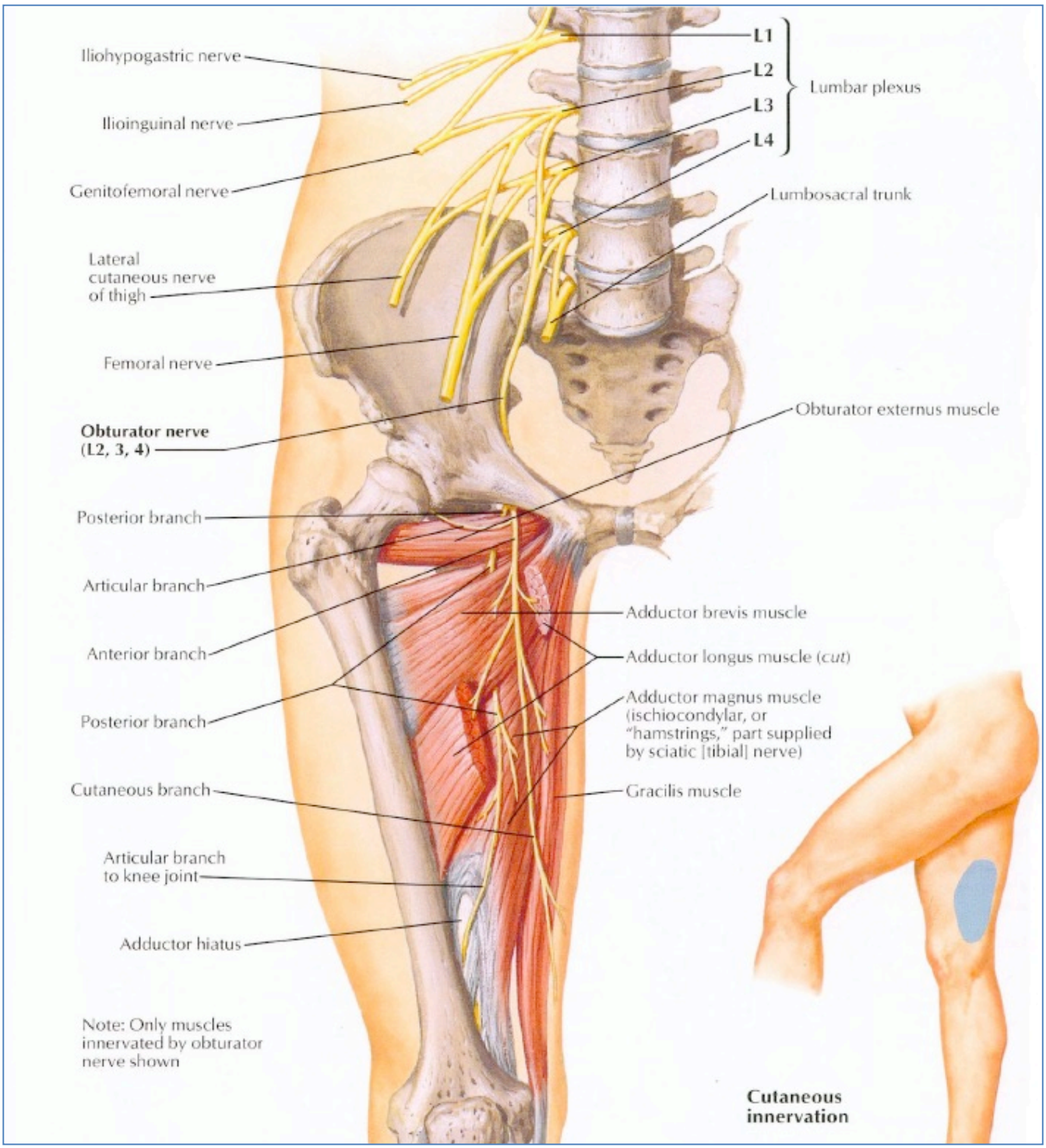
Innervation:

- **Lumbar Plexus:**
  - **Femoral Nerve:**
    - Branches off L2, L3 & L4
    - Runs between Psoas Major & Iliacus → beneath the Inguinal Ligament → Thigh.
    - Innervates:
      - Hip Flexors
      - Quadriceps (Knee Extensors)
      - Skin of Anterio-Medial Thigh & Lower Leg + Medial Aspect of Foot

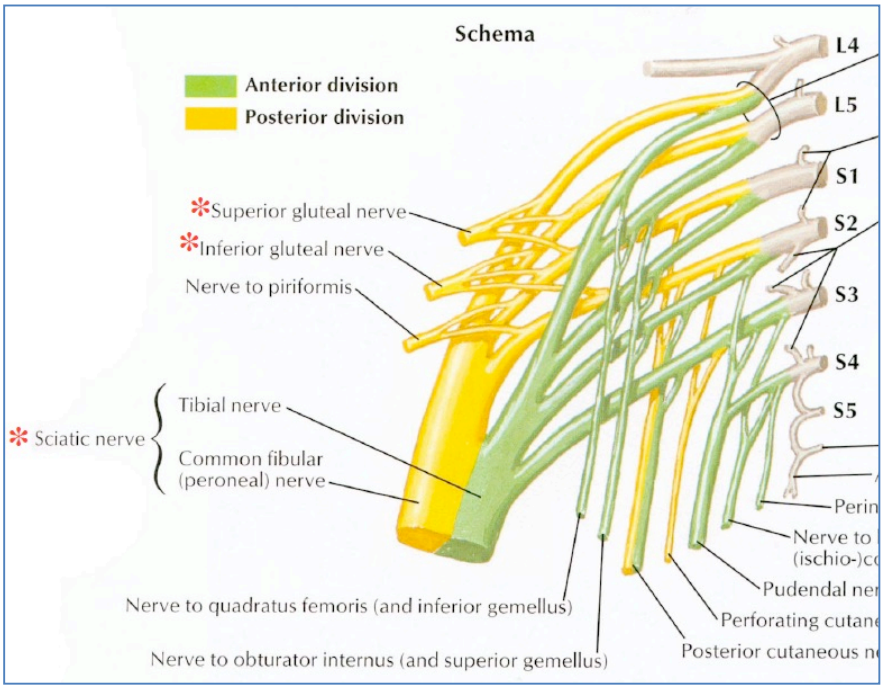


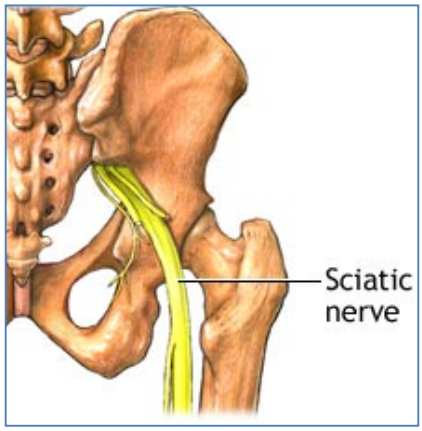
NB: Cutaneous Innervation – In blue

- **Obturator Nerve:**
  - Branches off L2, L3 & L4
  - Runs medial to Psoas Major, down along the inside wall of lesser pelvis → through Obturator Canal (in obturator membrane) through Obturator Foramen → Thigh
  - Innervates:
    - External Obturator
    - Adductor Muscles
    - Skin of medial aspect of thigh

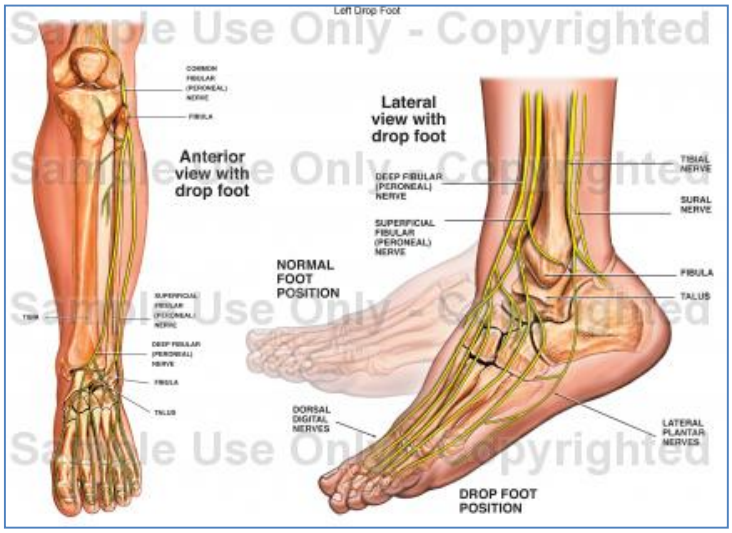


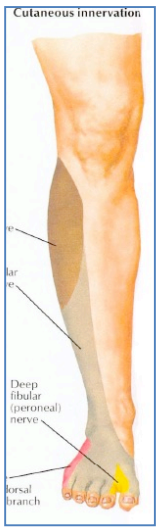
- **Sacral Plexus:**
  - **Sciatic Nerve:**
    - Branches off L4, L5, S1, S2 & S3
    - Runs from inside pelvis → through Greater Sciatic Foramen (below piriformis) → descends along the posterior thigh to about its lower third → ***Divides into 2 Branches: Tibial & Common Fibular Nerves.*** (some variation)
    - Innervates:
      - Hamstrings (Knee Flexors)
      - ½ of Adductor Magnus
      - Nearly the whole of the skin of the leg.



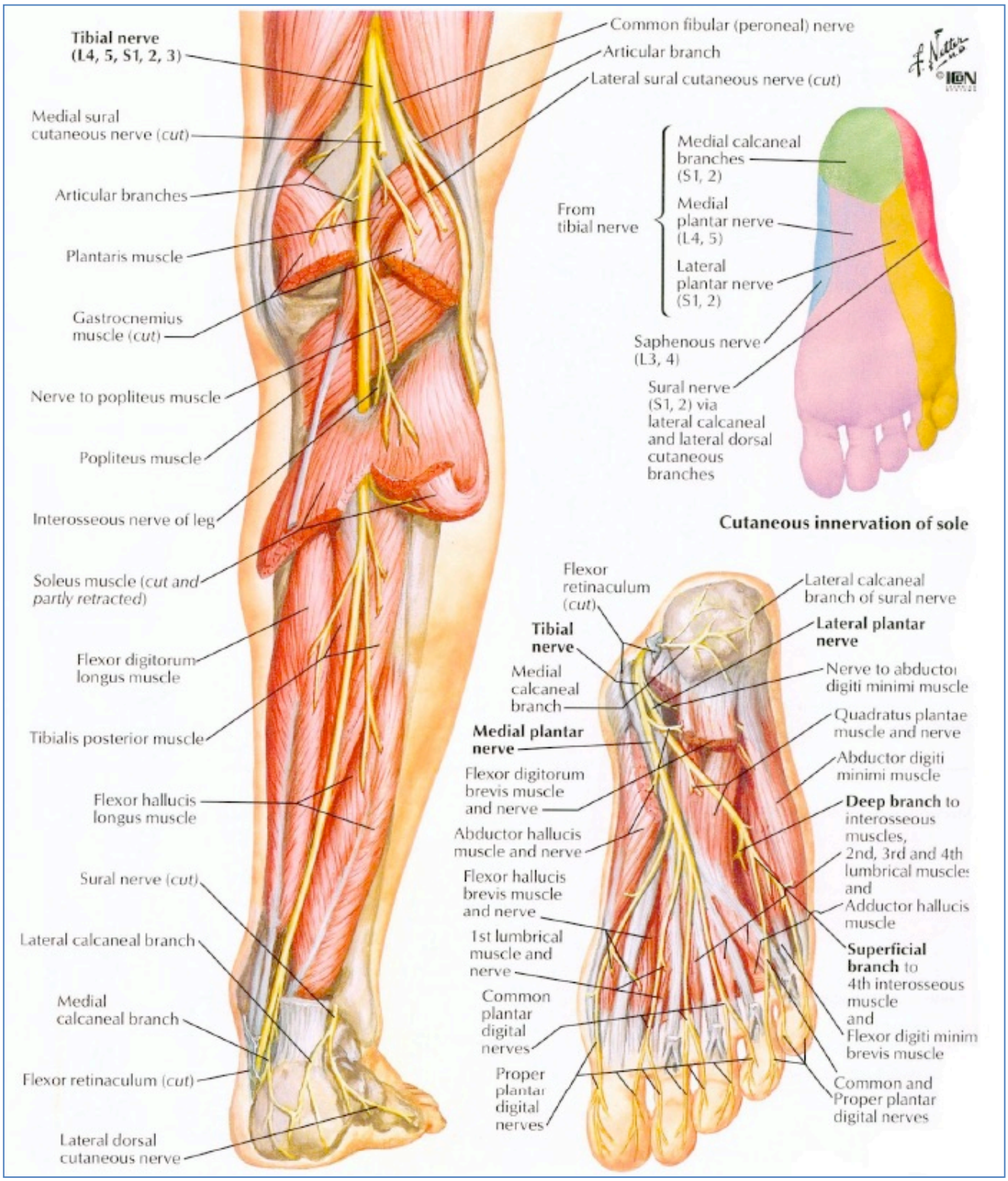


    - **Sciatic Nerve: Common Fibular (Peroneal) Nerve:**
      - Branches off Sciatic Nerve
      - Runs through the Popliteal Fossa → Winds around neck of Fibula → **divides into Deep & Superficial Fibular (peroneal) Nerves**
      - Innervates:
        - Dorsi-Flexors
        - Ankle Eversion
        - Skin of Lateral Aspect of Lower Leg
        - Skin of Dorsum of Foot





- **Tibial Nerve:**
  - Branches off Sciatic Nerve
  - Runs through Popliteal Fossa → Then follows the Tibia to the ankle → passes into Foot (below medial malleolus) → Terminates as Medial & Lateral Plantar Nerves
  - Innervates:
    - Plantar-Flexors (Calf Muscles)
    - Tibialis Posterior
    - Toe Flexors



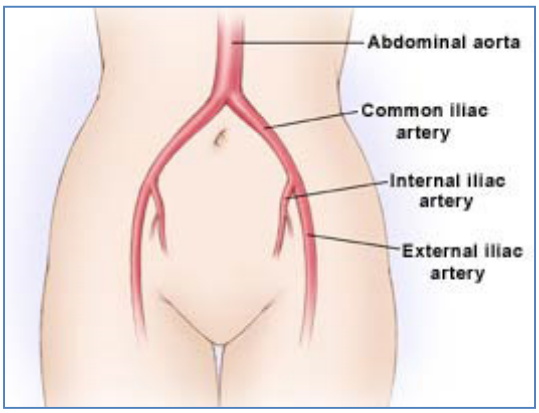
Nerve Lesions:

- Most are incomplete lesions
- Can be disabling

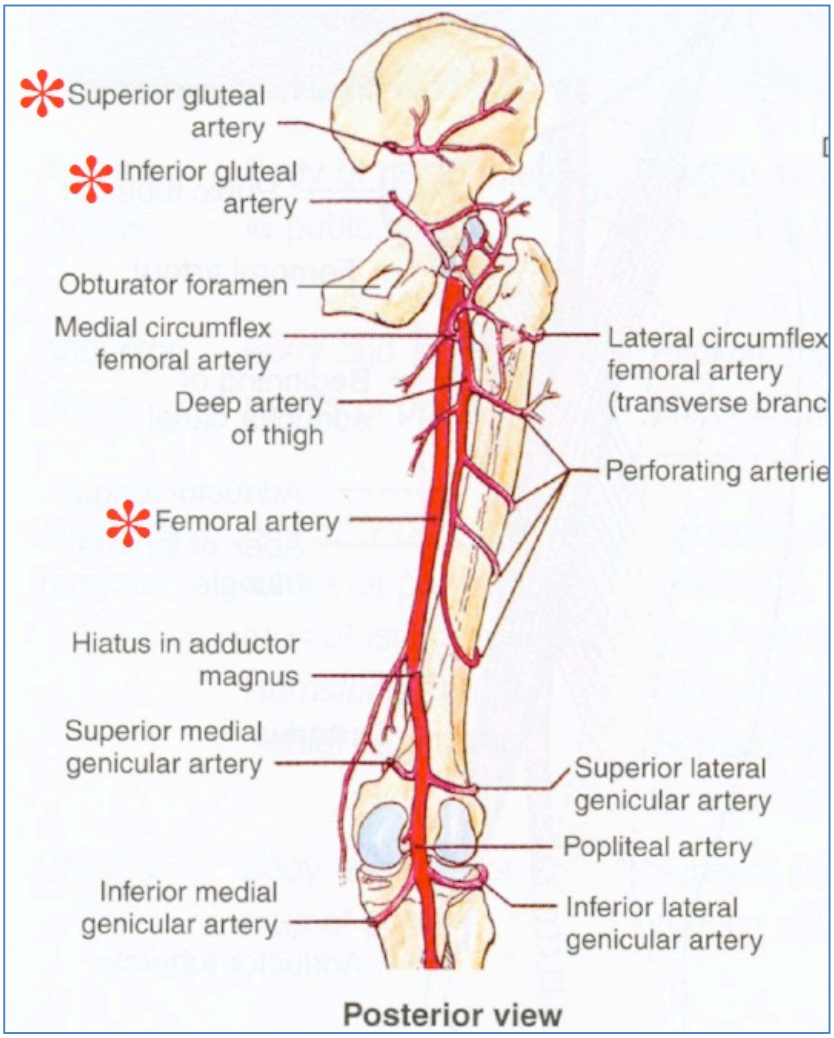
Nerve Lesion:	Functional Loss:
Femoral Nerve Lesion	○ Loss of Extension of Knee ○ Loss of Flexion of Hip
Obturator Nerve Lesion	○ Loss of Adductors of Hip ○ Weird Gait
Tibial Nerve Lesion	○ Loss of Plantar Flexion ○ Loss of Movement of Toes
Fibular Nerve Lesion	○ Loss of Dorsiflexion → 'foot drop' ○ Loss of Extension of toes ○ Loss of Eversion

Vasculation:

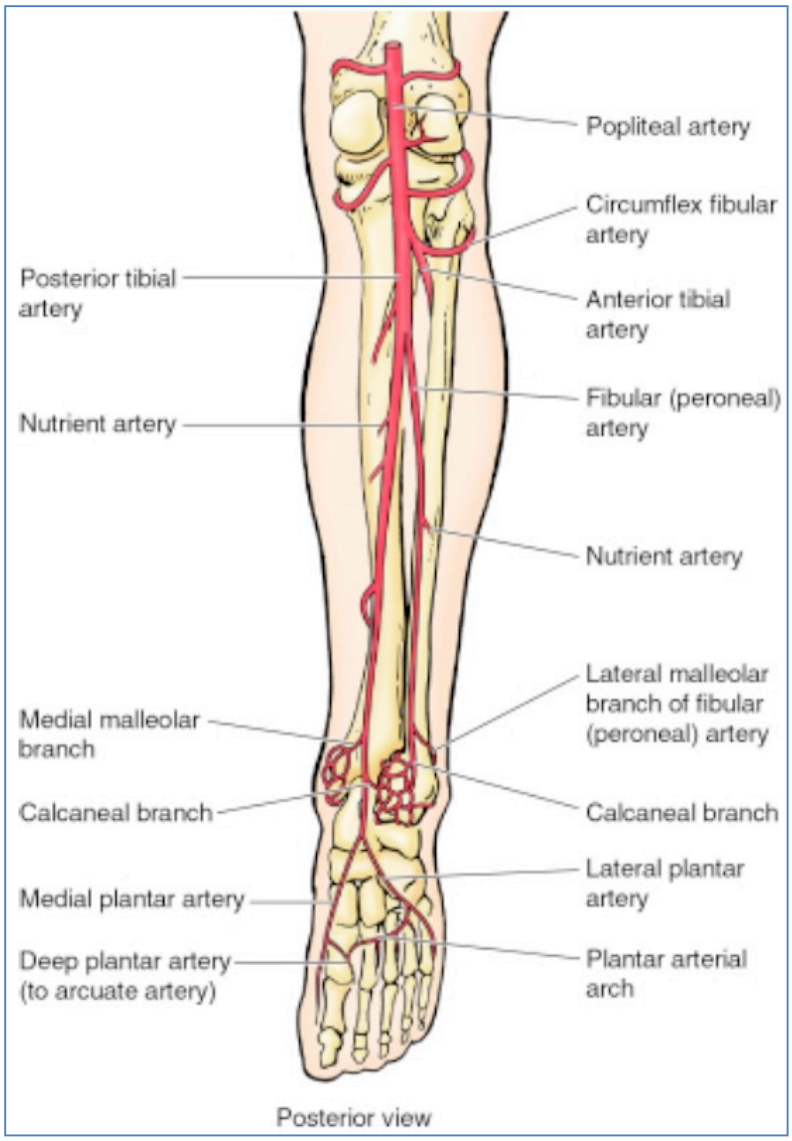
- **Arterial Blood Supply:**
  - **Thoracic:**
    - **Abdominal Aorta →**
  - **Pelvic:**
    - **Common Iliac Arteries →**
      - **Internal Iliac Artery →** Pelvic & Reproductive Organs + Buttocks + Medial Thigh
      - **External Iliac Artery →** Thigh, Leg, Foot.



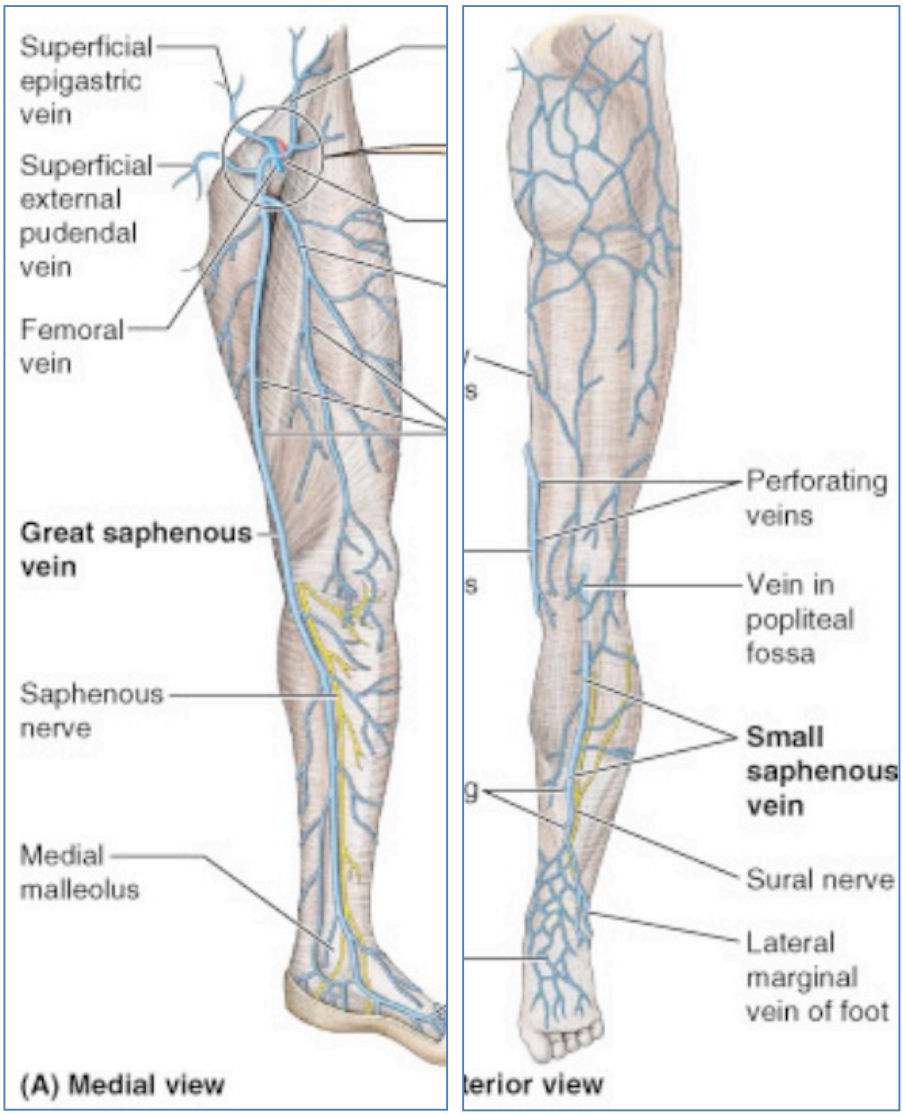
  - **Upper Leg:**
    - **External Iliac Artery →**
      - **Deep Artery of the Thigh →** branches into many perforating arteries
      - **Femoral Artery →** descends the femur, becoming more & more medial + posterior
        - **Popliteal Artery →**



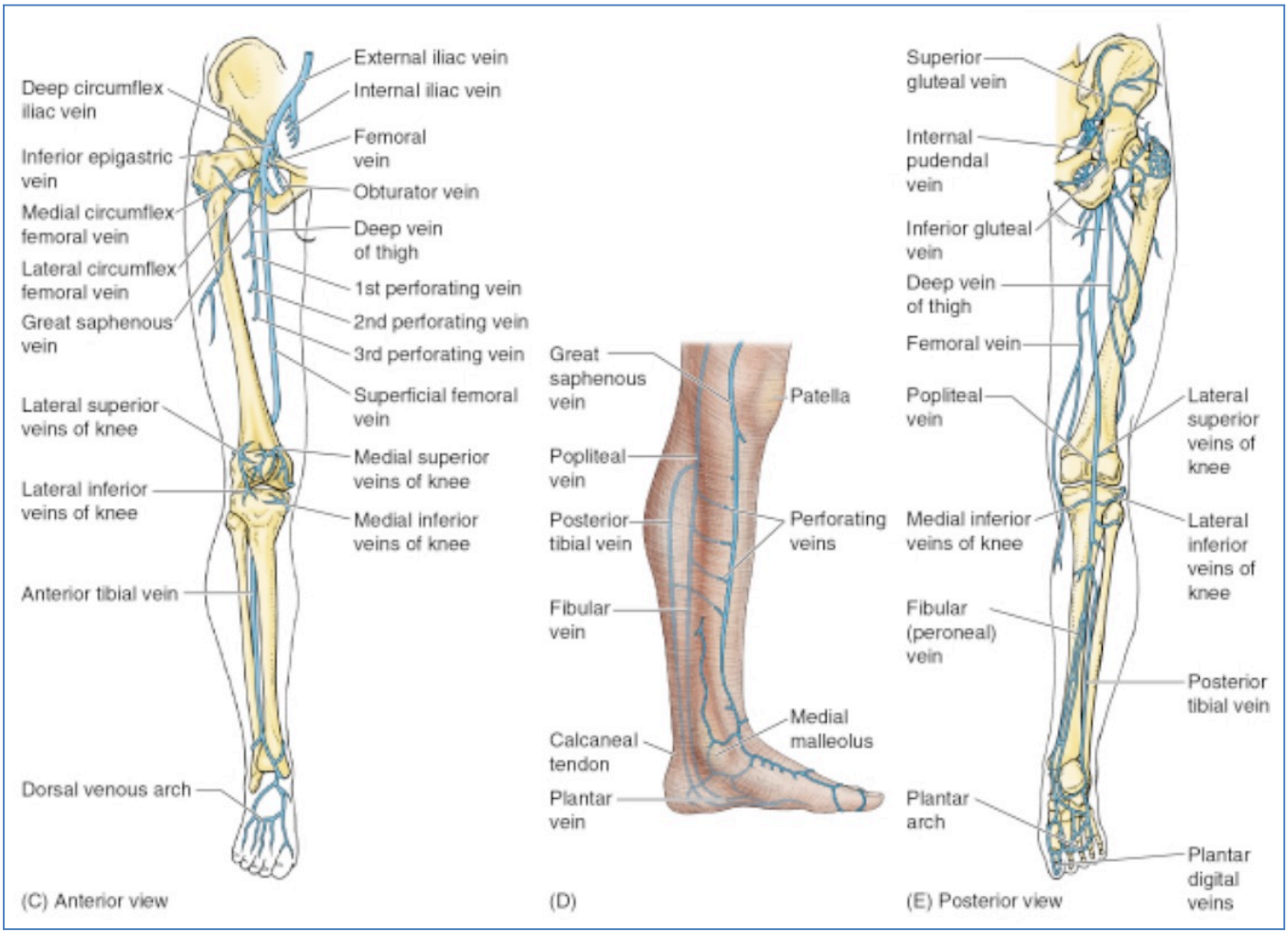
- **Lower Leg:**
  - **Popliteal Artery + Genicular Anastomosis** → Runs down 1/3 of lower leg, then splits into:
    - **Anterior Tibial Artery** -
    - **Posterior Tibial Artery** – Runs down posterior aspect of Tibia → ankle
    - **Fibular Artery** – Runs down posterior aspect of Fibula → ankle
- **Foot:**
  - **Anterior Tibial Artery →**
    - **Dorsalis Pedis Artery**
  - **Posterior Tibial Artery →**
    - **Lateral Plantar Artery**
    - **Medial Plantar Artery**



- **Venous Blood Drainage:**
  - **Foot:**
    - **Dorsal & Plantar Venous Arches**
  - **Lower & Upper Leg:**
    - **Deep:**
      - **Ant. & Post Tibial Veins → Popliteal Vein**
      - **Fibular Vein → Popliteal Vein**
    - **Superficial:**
      - **Small Saphenous Vein →** Runs up calves (posteriorly), goes deep → **Popliteal Vein**
      - **Great Saphenous Vein →** All the way up the leg (medially) → **Deep Femoral Vein**
  - **POPLITEAL VEIN → Deep Femoral Vein**
  - **Pelvic:**
    - **Deep Femoral Vein → External Iliac Vein**
  - **Thoracic:**
    - **External iliac Vein → Inferior Vena Cava**



Superficial Veins of Lower Limb:



Deep Veins of Lower Limb:

Emergency Medicine Notes
Musculoskeletal Injuries

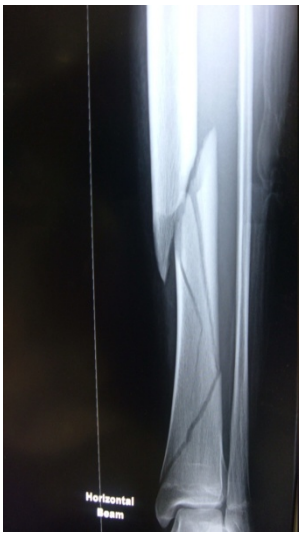
Key words + Definitions:

- **Fracture:**
  - A Break in a Bone
- **Compound fracture:**
  - An *Open* Fracture where there is broken skin.
- **Dislocation (or "*Luxation*"):**
  - The Displacement of Joint Surfaces such that Normal Articulation no longer occurs.
  - The ligaments always become damaged as a result of a dislocation.
  - A *Subluxation* is a partial dislocation.
- **Reduction:**
  - Medical procedure to restore a fracture or dislocation to the correct alignment.
- **Splint:**
  - Medical device used for immobilization of limbs or spine to prevent further injury and aid in patient retrieval.
- **Neurovascular compromise:**
  - Damage to either Vessels and/or Nerves as a result of an injury. Leads to functional impairments.
- **Compartment syndrome:**
  - With any bony injury, particularly crush-injuries, bleeding may occur into the muscle compartments. → Raises intra-compartmental pressure and compress blood vessels and nerves.

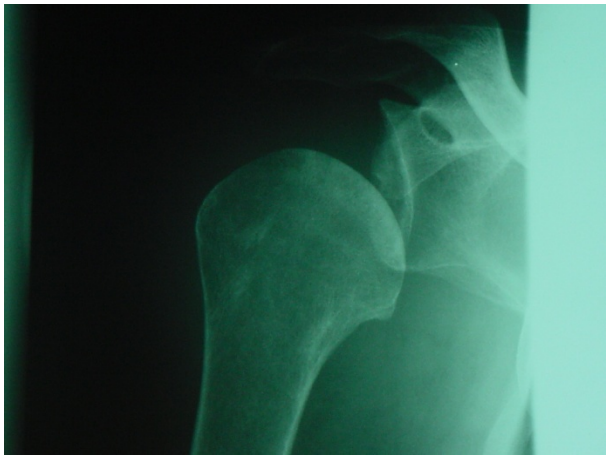
Major Concepts:

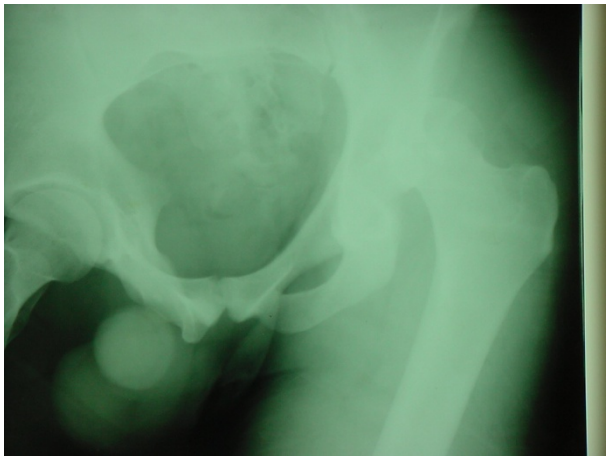
What is a Musculoskeletal Emergency and why?

- **Fractures:**
  - Breaks in Bone.
  - Emergency Because:
    - If it's an '*Open Fracture*' – Risk of Infection
    - Some fractures won't heal without treatment
    - Neurovascular compromise – can pull/tear/compress/rupture surrounding nerves/vessels.



- **Dislocations:**
  - The Displacement of Joint Surfaces such that Normal Articulation no longer occurs.
  - When forces on joint are greater than stabilizing forces of Bone, Ligament & Muscle.
  - Emergency Because:
    - The longer the delay before reduction, the more difficult it becomes, as the muscles around the joint contract.
    - Delay can also result in significant joint & ligament damage → Impairment of function.
    - Neurovascular compromise – can pull/tear/compress/rupture surrounding nerves/vessels.





- **Dismemberment:**
  - Loss of limb or Extreme Tissue-loss resulting in permanent functional impairment of that limb.

Factors Affecting the Degree of Urgency:

- Abnormal ABC
- Bleeding
- Major Vascular Compromise
- Open Vs. Closed Injury
- Neurological Compromise
- Pain
- Potential Loss of Function if Injury is Untreated.

The Basic Priorities of MSK Care:

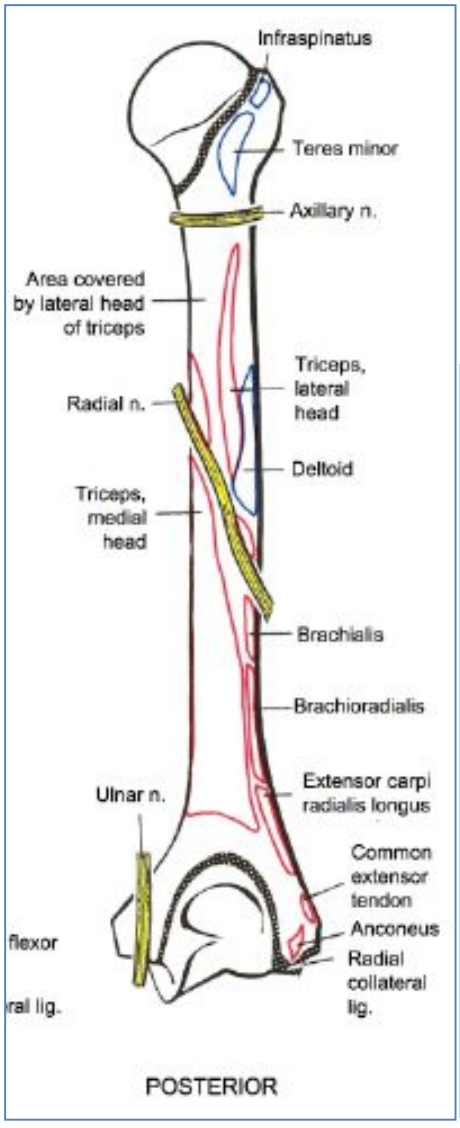
- Primary Survey – "ABC" (Life before limb)
- Identify Injury
- Analgesia
- Splint
- Prevent Infection
- Reduction (Restoring Alignment)

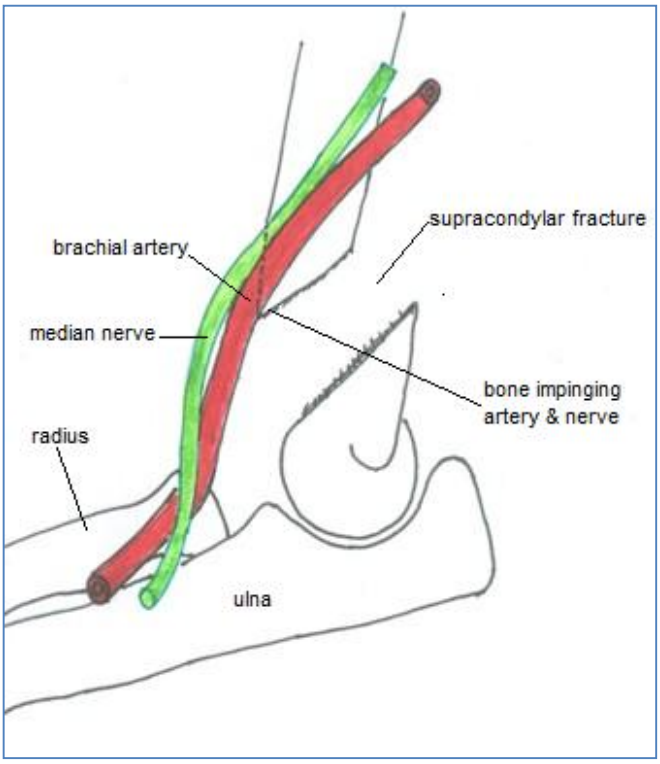
Benefits of Reduction & Splinting:

- **Splinting:**
  - Reduces Pain
  - Reduce Bleeding
  - Promote Healing
  - Reduce risk of Further Compromise (Bone/Neuro/Vascular/Functional)
- **Reduction:**
  - Reduce Pain
  - Restore Function
  - Reduce risk of Further Compromise (Neuro/Vascular/Functional)

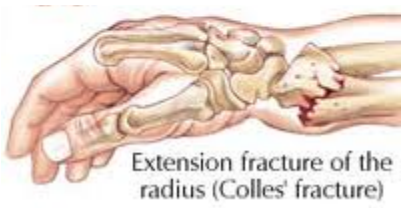
What Possible Neurovascular Compromise Can Result From:

- **Common Fractures:**
  - **Humeral #:**
    - **Neck:**
      - Axillary Nerve Damage
    - **Mid-Shaft:**
      - Radial Nerve Damage (As it closely traverses the lateral aspect of the humerus)
    - **Supracondylar:**
      - Median Nerve Damage
      - Brachial Artery.

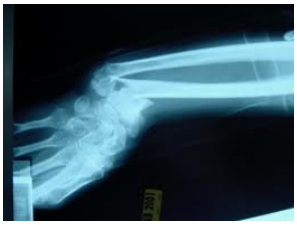




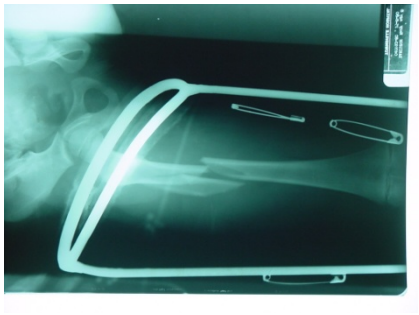
  - **Radial (Colles?) #:**
    - Median Nerve (Compression)
    - Ulnar Nerve (Compression)
    - Radial Artery


Extension fracture of the radius (Colles' fracture)

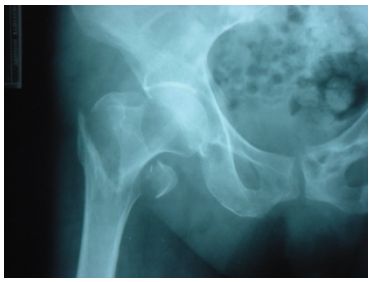
- **Wrist (Both Radius & Ulnar) #:**
  - Median Nerve Damage
  - Ulnar Nerve Damage
  - Radial Artery Laceration
  - Ulnar Artery Laceration



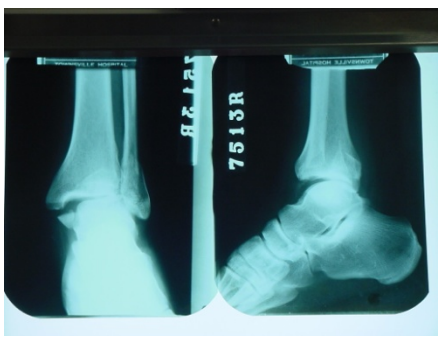
- **Femoral Shaft #:**
  - Femoral Nerve
  - Sciatic Nerve
  - Femoral Artery

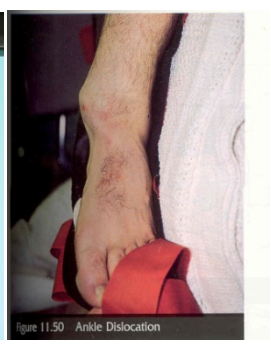


- **Neck of Femur #:**
  - Sciatic Nerve
  - Femoray Nerve
  - Femoral Artery

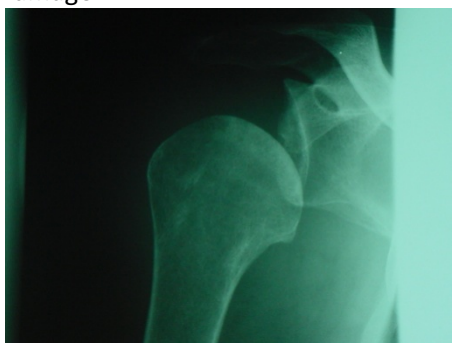


- **Ankle #:**
  - Post. Tibial Artery
  - Tibial Nerve

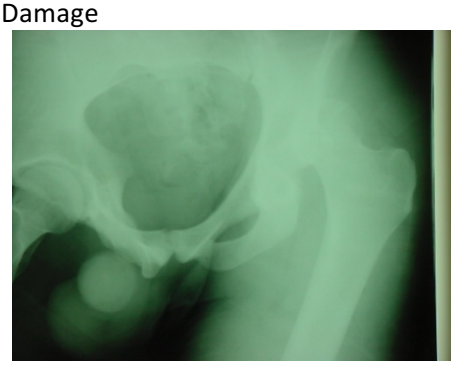




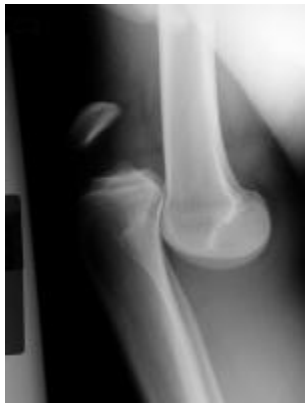
- **Common Dislocations:**
  - **Shoulder (Gleno-humoral):**
    - Axillary Nerve Damage
    - Musculocutaneous Nerve Damage
    - Radial Nerve Damage



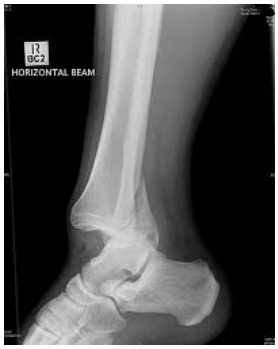
  - **Hip:**
    - Sciatic Nerve Damage



  - **Knee - Usually accompanied by Severe Ligament Damage:**
    - Tibial Nerve
    - Common Fibular Nerve
    - Popliteal Artery
    - Popliteal Vein



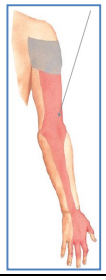
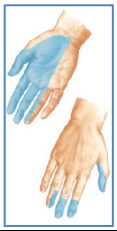
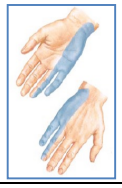
  - **Ankle - Usually accompanied by a Fracture:**
    - Post. Tibial Artery
    - Tibial Nerve



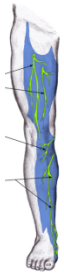
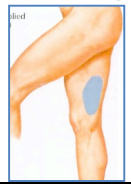
- **Laceration:**
  - **Volar (Palmar) Aspect of Wrist – (Eg. In Attempted Suicide):**
    - Median Nerve
    - Ulnar Nerve
    - Radial Artery
    - Ulnar Artery
    - Basilic Vein
    - Cephalic Vein
    - (+ Wrist Flexor Tendons)

What Functional Impairments Suggest Damage to These Nerves?:

- **Upper Limb:**

Nerve	Site of Injury	Paralysis	Motor Loss	Sensory Loss
Axillary Nerve	Axilla	Deltoid	Shoulder Abduction	Deltoid Region
Musculocutaneous Nerve	Axilla	Arm Flexors	Forearm Flexion	Lateral Forearm 
Radial Nerve	Axilla	Arm Extensors + Supinator	Elbow Extension, Supination, Wrist Extension	Lateral Dorsum of Hand, & Posterior Arm. 
	Cubital Fossa	Arm Extensors, *Except* Triceps	Supination, Wrist Extension	
Median Nerve	Elbow	Wrist Flexors, Thenar Muscles, Lateral 2x Lumbricals	Weak Wrist Flexion, Thumb Opposition, Lateral 2x Finger-Flexion	Lateral 3.5 Fingers 
	Wrist	Thenar Muscles, Lateral 2x Lumbricals	Thumb Opposition, Lateral 2x Finger-Flexion	
Ulnar Nerve	Above Elbow	Wrist Flexors, Hypothenar Muscles, Medial 2x Lumbricals	Weak Wrist Flexion, Medial 2x Finter-Flexion	Palm, Medial 1.5 Fingers 
	At Wrist	Hypothenar Muscles, Medial 2x Lumbricals	Medial 2x Finter-Flexion	

- **Lower Limb:**

Nerve	Site of Injury	Paralysis	Motor Loss	Sensory Loss
Femoral Nerve	Femoral Neck, Shaft #	Quadriceps Femori	Knee Extension, Hip Flexion	Antero-Medial Leg 
Obturator Nerve	Hip	Hip Adductors	Hip Adduction	Medial Thigh 
Sciatic Nerve	Hip, Femur #	Hamstring Function, Plantar-Flexors, Dorsi-Flexors, Intrinsic Foot Muscles.	Knee Flexion, Plantar-Flexion, Dorsi-Flexion, Ankle Eversion, Toe Movement	Most of Post.Thigh, Leg & Foot
Tibial Nerve	Knee, Tibial #, Laceration	Plantar-Flexors, Intrinsic Foot Muscles	Plantar-Flexion, Toe Movement	Posterior Leg
Fibular Nerve	Knee, Fibular # Laceration, Compression	Dorsi-Flexors, Intrinsic Foot Muscles	Dorsi-Flexion, Toe Movement, Ankle Eversion,	Anterio-Lateral Leg

- **Testing for Neurovascular Compromise:**
  - Presence of Vascular Compromise?
    - Bleeding/Haematoma → Probably
    - No Distal Pulses → Probably
    - Distal Pulses Present→ Probably Not
  - Presence of Neurological Compromise?
    - Sensory Alterations/ Loss → Probably
    - Impaired Motor Function → Probably
    - Neither of the above → Probably Not

Major Processes:

Describe the Approach to Musculoskeletal Emergencies:

1. **First, The Primary survey – ABCDE:**
    - Airway
    - Breathing
    - Circulation
    - Disability
    - Exposure

2. **Analgesia (If Necessary):**
    - **a.** Is it needed?..If So:
        - **i.** What?
        - **ii.** Which Route?
        - **iii.** What Dose?
    - **b.** **Oral Analgesia (Paracetamol/Aspirin) –** Cheap & Easy; But Weak
    - **c.** **Parenteral Analgesia (IM/IV opiates)** – Strong; But More Expensive/Complicated
    - **d.** **Regional Blocks (LA injection around a nerve) –** Only Good for Isolated Injuries

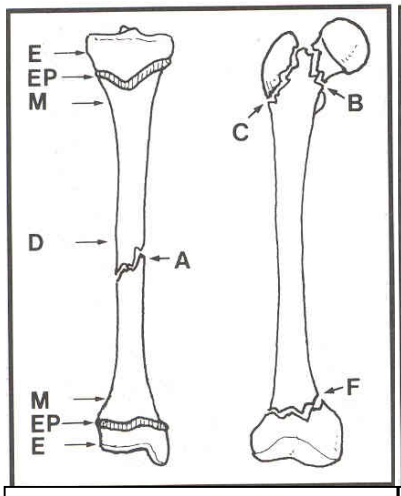
3. **Musculoskeletal Assessment:**
    - **a.** Suspicion of Injury from:
        - **i.** History
        - **ii.** Appearance
        - **iii.** Examination
        - **iv.** X-ray (Later – for Confirmatory Purposes)
    - **b.** Open Vs. Closed:
        - **i.** High Risk of Infection if Open – Requires early treatment.
    - **c.** Neurovascular Compromise?
        - **i.** Presence of Vascular Compromise?
            - **1.** Bleeding/Haematoma → Probably
            - **2.** No Distal Pulses → Probably
            - **3.** Distal Pulses Present→ Probably Not
        - **ii.** Presence of Neurological Compromise?
            - **1.** Sensory Alterations/ Loss → Probably
            - **2.** Impaired Motor Function → Probably
            - **3.** Neither of the above → Probably Not

4. **Order & Examine the X-Ray:**
    - **a.** **X-Ray Order:**
        - **i.** Include 2 Views:
            - **1.** AP (Anteroposterior)
            - **2.** Lateral
        - **ii.** Also include images of the joints Above & Below.
    - **b.** **Examining the X-Ray:**
        - **i.** Examine area of suspicion
        - **ii.** Examine joints Above & Below.
        - **iii.** Examine all other bones.
        - **iv.** → Confirms the clinical diagnosis & gives more info about injury severity.

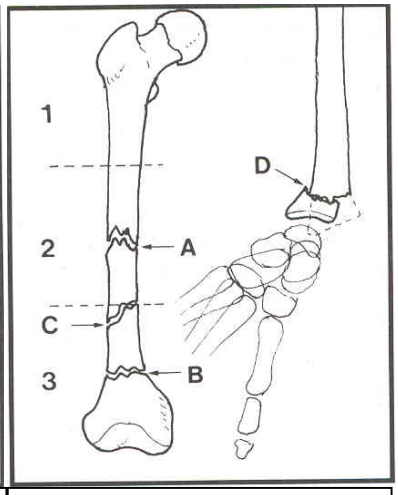
5. **Treatment:**
    - **a.** Splinting (if permanent treatment isn't immediately available)
    - **b.** Reduction
    - **c.** Surgery

***XRays* - Differentiate Normal from Abnormal and Describe an X-Ray:**

- **Describing an X-Ray:**
  - **Fracture (#) or Dislocation?**
  - **Which Bone?**
  - **Location? (Which part of the Bone?):**
    - (E) Epiphysis
    - (EP) Epiphyseal Plate
    - (M) Metaphysis
    - (D) Diaphysis (Shaft) [In 'Thirds']
      - Eg. (1) Proximal 1/3
      - Eg. (2) Diaphyseal (mid) 1/3
      - Eg. (3) Distal 1/3)



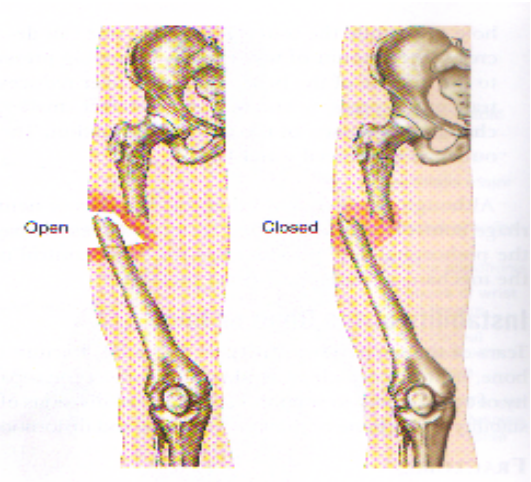
(A) = Tibial Diaphysis (B) = Femoral Neck (C) = Greater Trochanter (F) = Supracondylar #



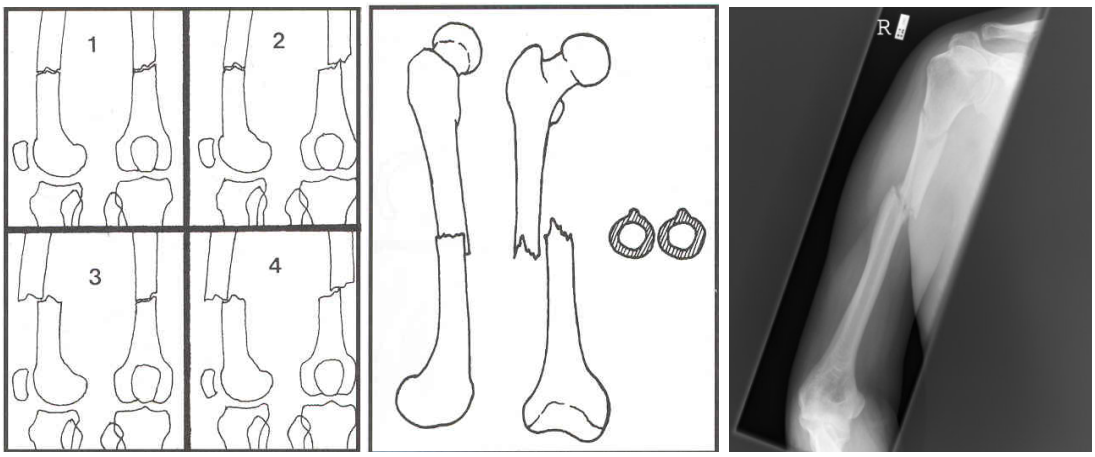
(A) Diaphyseal Femur # (B) Distal Femur # (C) Mid-Distal Femur # (D) Radial 'Colles' #

    - Epicondyle
    - Malleolus
    - Etc.

- **If It's a Fracture (#):**
  - **Properties of Fractures:**
    - **Closed** (*Simple*) or **Open** (*Compound*)?

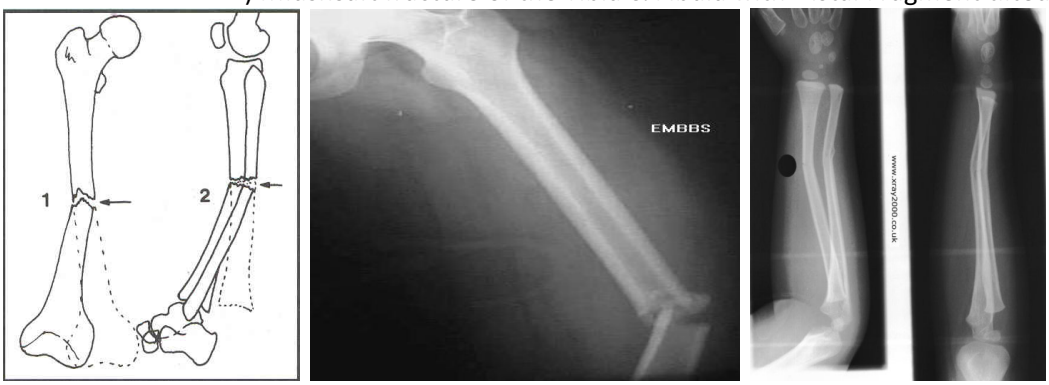


- **Displaced?** (Fracture gap) – Described as a Percentage. (Eg. 50% Displaced)

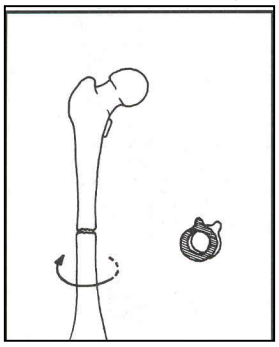


1) Aligned; 2) 50% Displaced; 3) 80% Displaced;
4) 90% Displaced; Right) 100% Displaced

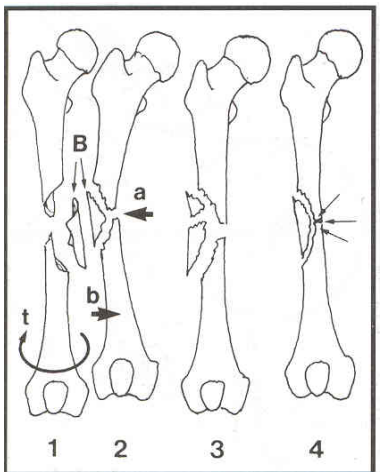
- **Angulation** – Expressed in *Degrees* relative to Each Other. (Eg. A 30^{o} Angulation)
  - 1) Fracture of the Mid 1/3 of Femur with distal fragment tilted laterally.
  - 2) Midshsaft fracture of the Tibia & Fibula with Distal Fragment tilted Anteriorly.

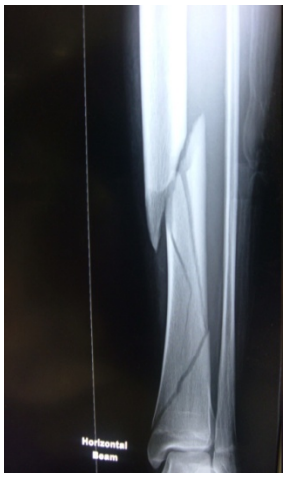


- **Rotated** – Can be hard to see on an X-Ray.



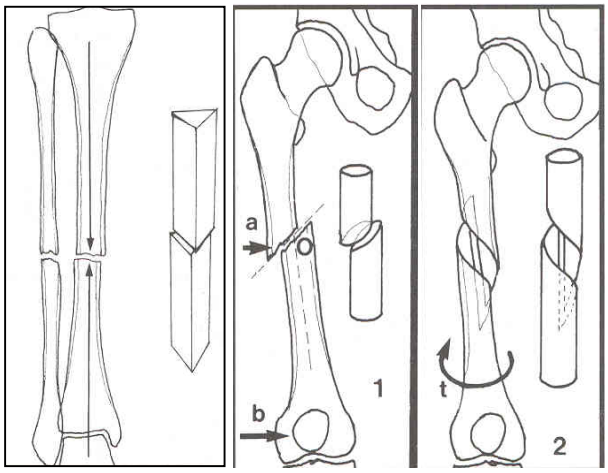
- **Types of Fractures:**
  - **Complete**: A fracture in which bone fragments separate completely.
  - **Incomplete**: A fracture in which the bone fragments are still partially joined.
  - **Greenstick**: Occurs mostly in children with non-brittle bones. (An Incomplete #)
  - **Comminuted**: 3 or More Pieces





  - **Compacted**: A fracture caused when bone fragments are driven into each other.

- **What shape is the #?**
  - **Linear**: A fracture that is parallel to the bone's long axis.
  - **Transverse**: A fracture that is at a right angle to the bone's long axis.
  - **Oblique**: A fracture that is diagonal to a bone's long axis.
  - **Spiral**: A fracture where at least one part of the bone has been twisted.



Transverse **Oblique** **Spiral**

- **If Dislocation (AKA: *"Luxation"*):**
  - **1. Which Joint?**
    - Shoulder (Gleno-humoral)
    - Fingers (Inter-Phalangeal)
    - Wrist - Usually accompanied by a fracture
    - Elbow - Usually accompanied by a fracture
    - Knees
    - Ankle - Usually accompanied by a fracture
  - **2. What Direction?:**
    - Superior/Inferior
    - Anterior/Posterior
  - **3. Is there an Associated Fracture?**
  - **4. Any possible Neurovascular Compromises?**

Emergency Medicine Notes
Neurological Emergencies

What is a Neurological Emergency?

- Involves all or part of the CNS or PNS. (We'll focus on CNS)
- Acute
- Serious or Potentially serious
- Requires urgent or emergent investigation and/or treatment

Classification of Neurological Emergencies:

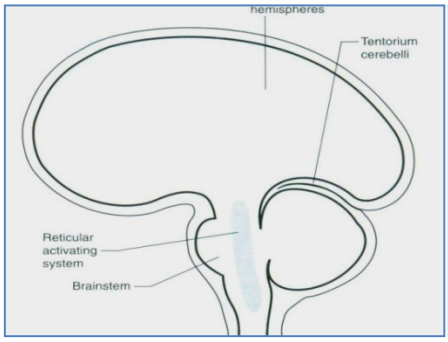
- **No Particular Method.**
- Not Very Important
- (Usually Pathology/Symptom Based)
- **Pathology based classification:**
    - Infection
    - Trauma
    - Neoplasia
    - Haemorrhage
    - Toxins/Drugs
- **Symptom-sign based Classification:**
    - **Based on anatomic/physiologic dysfunction:**
        - Loss of motor function
        - Loss of sensory function
        - Visual disturbance
        - Headache or other pain

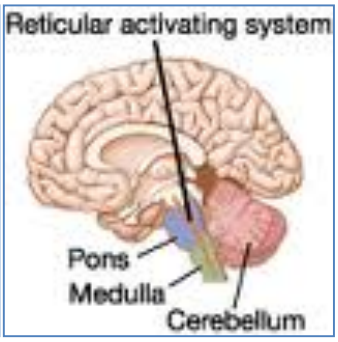
WEEK OVERVIEW - Symptoms:

- **Altered level of Consciousness (Covered Last Week)**
- **Headache**
- **Acute neurological deficit**
- **CNS infection**
- **Traumatic Brain Injury (TBI) and spine trauma**
- **Seizures**

ALTERED LEVEL OF CONSCIOUSNESS (COVERED LAST WEEK):

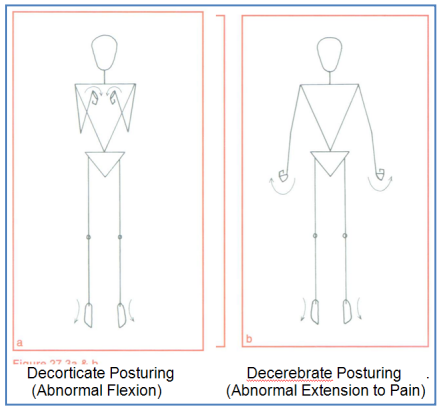
- **Two Possibilities:**
  - **Global Impairment of *Cerebral Cortices*:**
    - Possible Causes (Surgical Sieve):
      - Infection
      - Trauma
      - Neoplasia
      - Haemorrhage (Vascular)
      - Toxins/Drugs
      - Idiopathic
      - Endocrine
  - **Impairment of *Reticular Activating System* (The bit that allows consciousness) in Brainstem:**
    - Possible Causes (Surgical Sieve):
      - Infection
      - Trauma
      - Neoplasia
      - Haemorrhage (Vascular)
      - Toxins/Drugs
      - Idiopathic
      - Endocrine





- **Assessment:**
  - **Glasgow Coma Scale:**
    - Grades eye opening, verbal and motor response to stimuli and is more precise than the AVPU system.
    - (Minimum Score = 3)
    - (Comatose = < 8; Remember Airway Management)

Components:		Score:	
Eye Opening:	Spontaneously	4	
	To verbal command	3	
	To pain	2	
	Nil	1	
Verbal Response:	Orientated	5	
	Confused	4	
	Inappropriate words	3	
	Incomprehensible sounds	2	
	Nil	1	
Motor response:	Obeys command	6	
	Localises pain	5	
	Withdraws to pain	4	
	Abnormal flexion	3	(*decorticate posturing*)
	Extension to pain	2	(*decerebrate posturing*)
	Nil	1	



  - **AVPU Scale:**
    - This is the most simple and categories the patient into one of four states.
      - ***Alert*** = awake *but* may be confused
      - ***Verbal*** = responding to verbal stimulus
      - ***Pain*** = responding *only* to painful stimulus
      - ***Unresponsive*** = no response to *any* stimulus

- **CASES**
  - Give each of the following cases a AVPU rating and GCS.
  - **Case 1**
    - A 16 year old female is brought into your ED by ambulance after falling from a third floor balcony. Her eyes are closed despite painful stimulus and she's making groaning noises. She pulls her hands and feet away when painful stimulus applied to them.

    - AVPU……..
    - GCS……..

  - **Case 2**
    - A 24 year old man who is cheerfully intoxicated with alcohol is brought to your ED by Police. His speech is slurred he doesn't know what day or month it is, but is very happy to assist you with your physical examination of him.

    - AVPU……..
    - GCS……..

  - **Case 3**
    - A febrile 82 year old is brought into your ED by relatives. She is looking around the ED but every time you ask a question she tells you to "@#$% off" and this response never varies. She tries to hit you when you ask her to move her arms or legs.

    - AVPU……..
    - GCS……

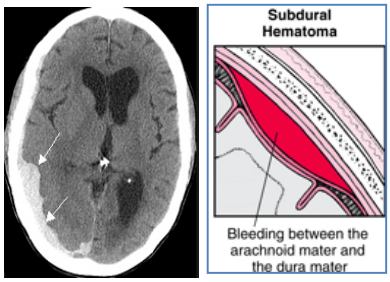
  - **Case 4**
    - A six month old is brought your ED by his parents after suffering a generalised seizure. His eyes are open but he makes no response to any stimulus.

    - AVPU……..
    - GCS…… .
    - How useful do you think the AVPU and GCS scales are in this age group? Why?

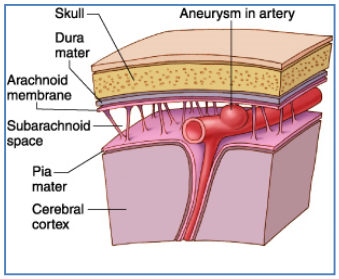
HEADACHE:

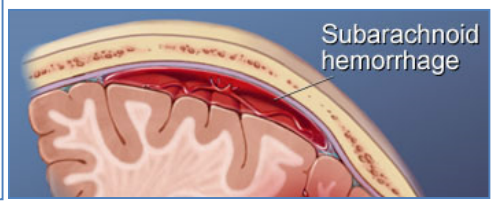
- (One of the most common presentations in ED)
- **Range of Causes:**
  - *Most are* Benign
  - *But Some are* Life Threatening
  - (NB: Headache ≠ *'Brain Ache'*)-(Many extra-cranial pathologies can cause headache)
- **Broad differential diagnosis**
- Need to understand pathophysiology
- **Classification:**
  - **Primary (Typically Benign):**
    - Migraine
    - Cluster Headache
    - Tension Headache
  - **Secondary (Due to Specific Pathology):**
    - Eg. Meningitis
    - Eg. Brain Tumour
    - Eg. Bleed/Haemorrhage
    - NB: IF it's the worst headache they've ever had, It's quite often a Sub-Arachnoid Haemorhage → Investigate.
    - If there's a Neurological Deficit + Headache → Investigate

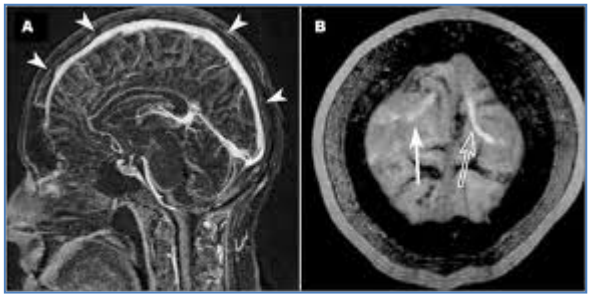
- **Some Haemorrhages can cause Unexplained Headache in Healthy People:**
  - ***Subdural Haematoma* (SDH):**
    - **Haematoma is beneath the dura**
    - **Caused by:**
      - Mostly Old people with Low force trauma → Slow Venous Bleed
        - Because their brain is shrunken
        - Brain moves significantly under mild trauma
    - **Gradual onset** (:. Is often Chronic)
    - **Wide distribution** – ie. Over the entire hemisphere
    - **Presentation:**
      - Unsteady gait
      - Other abnormalities
    - **Prognosis:**
      - **Abnormal Brain underneath** (Ie. Pt. Is often demented, or alcoholic, or have severe cerebral atrophy)
      - Therefore the prognosis is worse



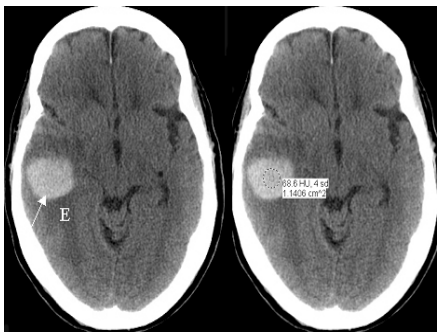
- ***Subarachnoid Haemorrhage*** **(SAH):**
  - **Caused by:**
    - Often due to Aneurysm of circle of willis
    - Hypertension is a Contributing Factor
    - (Arterial Bleed)
  - **Presentation:**
    - Typically a fit, well person who has sudden onset, severe headache (or feel like they've been hit in the head with a baseball bat)
    - Loss of Consciousness.





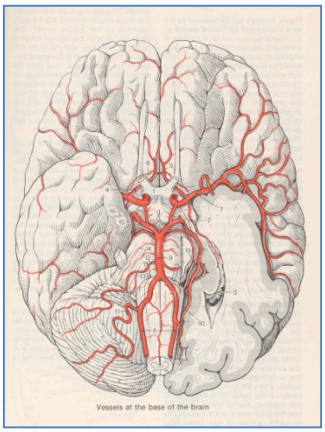


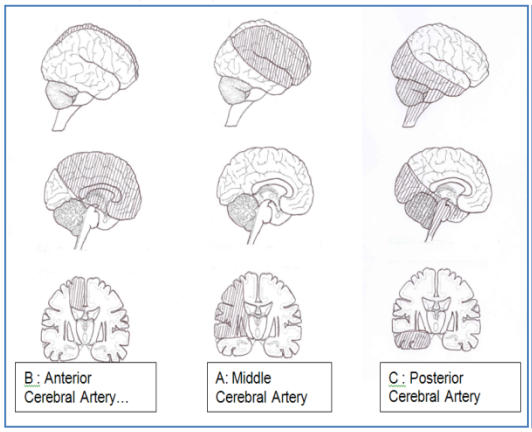
- ***Intracranial Haemorrhage*** **(ICH) ("Haemorrhagic Stroke"/CVA)**:
  - **Caused by:**
    - Non penetrating and penetrating cranial trauma.
    - →Haemorrhage directly into Brain Parenchyma → Stroke
  - **Presentation:**
    - Headache
    - Various Neurological Deficits
    - Loss of Consciousness
    - Vomiting
  - **Prognosis:**
    - 75% of Survivors are Disabled

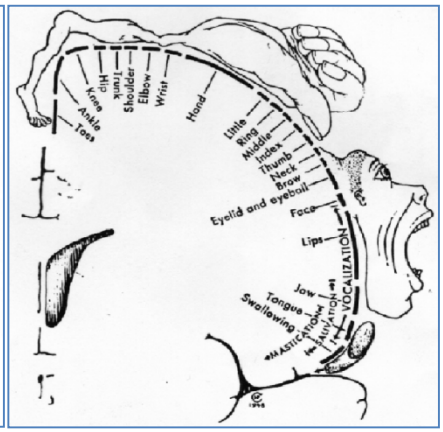


ACUTE NEUROLOGICAL DEFICIT:

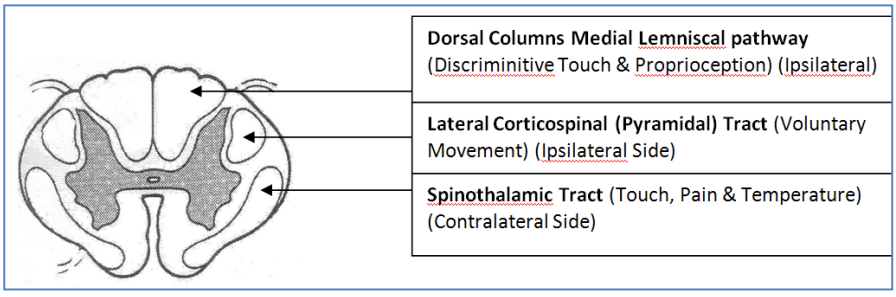
- (An Enormous & Complex Topic)
- ***EXAM* Understand the Cerebra Circulation & Deficits Result from Ischaemia of Certain Brain Regions:**
  - **Anterior Cerebral Arteries:**
    - Fed by the Internal Carotids
    - Legs & Arms Affected by Compromise
  - **Middle Cerebral Arteries:**
    - Fed by the Internal Carotids
    - Hands, Fingers & Face Affected by Compromise
  - **Posterior Cerebral Circulation:**
    - Fed by the Basilar Artery
    - Vision Affected by Compromise





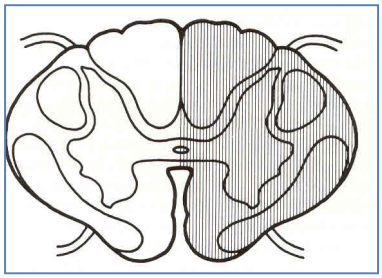


- **Spinal Cord Tracts:**
  - **Main Spinal Cord Tracts and their Functions:**
    - **Dorsal Column Medial Lemniscal Tract (Afferent):**
      - Ascends in the Dorsal Aspect of the Spinal Cord
      - Functions:
        - Fine/Descriminitive Touch
        - Proprioception
      - Decussates in the Medial Lemniscal Tract in the Medulla
        - Sensory Deficits will be Ipsilateral to the Spinal Lesion
    - **Spinothalamic (Afferent):**
      - Ascends in the Lateral Aspects of the Spinal Cord
      - Functions:
        - Touch
        - Pain
        - Temperature
      - Decussates @ The Level of the Spinal Cord
        - Sensory Deficits will be Contralateral to the Spinal Lesion
    - **Corticospinal (Efferent):**
      - Descends from the Motor Cortex through the Lateral Aspects of the Spinal Cord
      - Function:
        - Voluntary Movement
      - Decussates in the Pyramidal Tracts (In the Brain)
        - Motor Deficits will be Ipsilateral to the Lesion

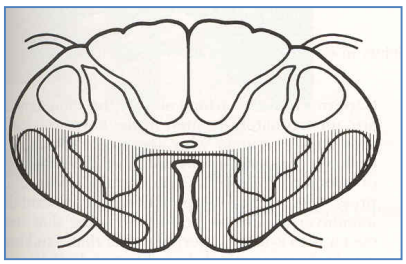


- **Changes in Motor Reflexes with Spinal Cord Injury:**
  - **Immediate Consequences:**
    - Reflexes are conserved since they aren't mediated by the brain.
    - (NB: Reflexes are only lost if the lesion is @ the level of that reflex)
  - **Consequences Over Time:**
    - Muscle movement diminishes over a period of time
    - Due to Progressive Muscle Atrophy (not Nerve Atrophy)

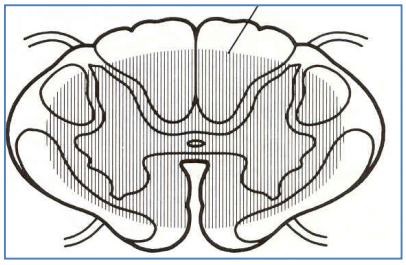
- **Different Spinal Cord Lesions (Pathways Affected & Clinical Consequences):**
  - **'Brown-Sequard' Syndrome:**
    - (seen if someone is stabbed in the back with a knife or shot with a handgun causing a hemi-transection of the spinal cord)
    - **Pathways Affected & Clinical Consequences:**
      - Dorsal Column Medial Lemniscal Pathway
        - Loss of Discriminative Touch & Proprioception
        - (Ipsilateral to & below the level of the lesion)
      - Spinothalamic Tract
        - Loss of somatosensation (Touch, Pain, Temperature)
        - (Contralateral to & below the level of the lesion)
      - Lateral Corticospinal:
        - Loss of voluntary movements
        - (Ipsilateral to the side of the lesion. Because it decussates in the pyramidal tracts)



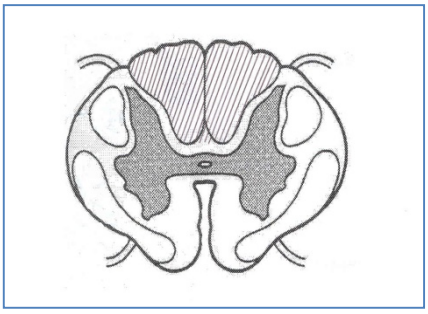
  - **Anterior Spinal Artery Syndrome:**
    - (Due to Lesion of the Anterior Spinal Artery. Eg. Diving injury)
    - **Pathways Affected & Clinical Consequences:**
      - Doesn't affect the Dorsal Column Medial Lemniscal pathway.
        - No loss of Discriminitive Touch & Proprioception
      - Doesn't Affect the Corticospinal Tract
        - Conserves Motor Function)
      - Affects Spinothalamic Tract
        - Loss of Somaosensation Contralateral to & below the level of the lesion.



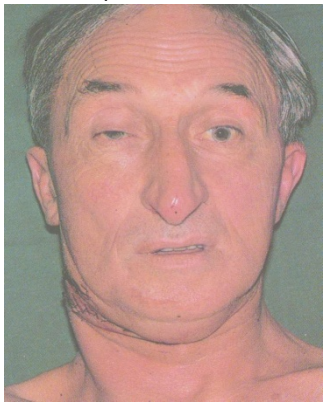
- **Central Cord Syndrome:**
  - (Usually secondary to spinal trauma and, affects the centre of the spinal cord.)
  - **Pathways Affected & Clinical Consequences:**
    - Mainly Corticospinal Tracts
      - → Motor Impairment (Mostly in Upper Extremities)
      - (Why? Motor Fibres supplying Upper limbs tend to be more Central than those supplying the lower limbs)
    - Dorsal Column & Spinothalamic Tracts:
      - Variable sensory losses below the Lesion.



- **Dorsal Column Syndrome:**
  - (Very Unusual)
  - **Pathways Affected & Clinical Consequences:**
    - Dorsal Column Medial Lemniscal Tracts:
      - Ipsilateral Loss of Discriminitive Touch & Proprioception Below the Lesion
    - Doesn't Affect Spinothalamic Tract:
      - Somatosensation (Touch, Pain, Temperature) Unaffected.
    - Doesn't Affect Corticospinal Tract:
      - Motor Functions Conserved

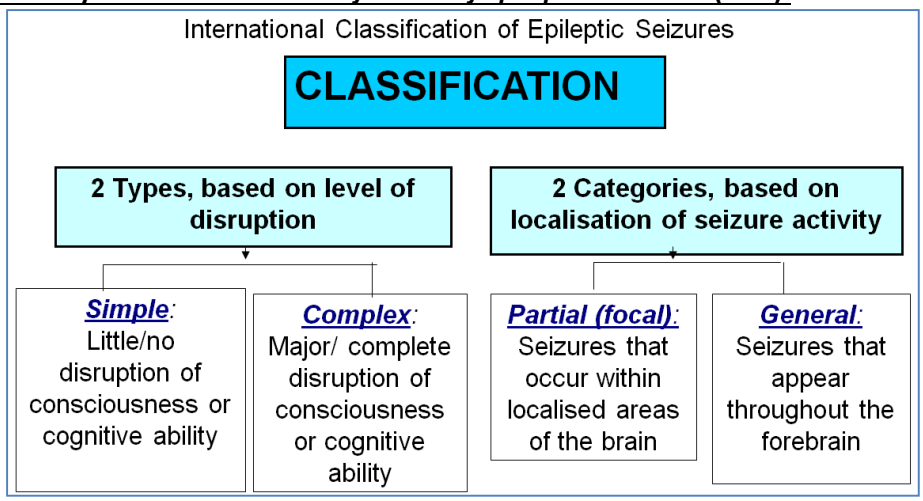


- **Central/Peripheral:**
  - **Central:**
    - Due to Need to a lesion into cerebral cortex, brain stem and spinal cord
    - Need to be able to localize the lesion
  - **Peripheral:**
    - (Mostly due to Trauma)
    - – Not dealt with this week. (See Week 1 – MSK Emergencies)



SEIZURES:

- **Classification of Seizure Disorders:**
  - **NB: Old-School Nomenclature:**
    - **Grand Mal ("Big Bad") Seizure:**
      - Eg. Full 'Tonic Clonic' seizures.
    - **Petit Mal ("Little Bad") Seizure:**
      - Eg. Absence seizure
      - NB: Some old doctors regard Petit Mal seizures as any seizure that is not Grand Mal.
  - **Currently: "*International Classification of Epileptic Seizures*" (ICES):**



- **Pathophysiology of Seizure Activity:**
  - ****Neurons are Hyperexcitable**; 3 Possibilities:**
    - **1.**Resting Membrane Potential has been Altered in a Subset of Neurons**:**
      - Pushes Neurons *Closer to Threshold* → Spontaneous Activity
    - **2.**Ion-Channelopathy → Decrease in Threshold of Voltage-Gated Channels**:**
      - Eg. Mutation in Amino Acid sequence in Voltage-Gated Channels → Channel Responds to Lower Voltage (Ie. More Negative Potentials) → Hence, are More Easily Activated.
    - **3.**Neurotransmitter Imbalance**:**
      - Inappropriate Activity of the Epileptic Focus can be due to Excess of Glutamate Activity or a Deficit of GABA Activity:
      - **↑Excitatory (Glutamate= Primary Excitatory Neurotransmitter):**
      - **Or ↓Inhibitory (GABA= Primary Inhibitory Neurotransmitter):**

- **Types of Seizures:**
  - **"Simple Partial Seizure":**
    - Ie. Conscious & Localised Seizure.
    - **Symptoms Depend on Cortical Region Affected:**
      - Typically – Small, Rapid Muscle Movements
    - **Duration:**
      - Very Short Duration (Less than 1min)
    - **NB:** Preservation of Consciousness & Memory is Key.

  - **"Complex Partial Seizure":**
    - Ie. Impaired Consciousness & Localised Seizure.
      - NB: '*Impaired* Consciousness' = Dazed / Vague / Dream-like / Inability to Respond / Amnesia.
    - **Symptoms Often Associated with Purposeless Movements:**
      - Hand-Wring
      - Pill-Rolling
      - Face-Washing
      - Fidgeting
      - Mumbling
    - **Duration:**
      - Less than 2 min
    - **NB:** Impaired Consciousness → Little/No Memory of Seizure.

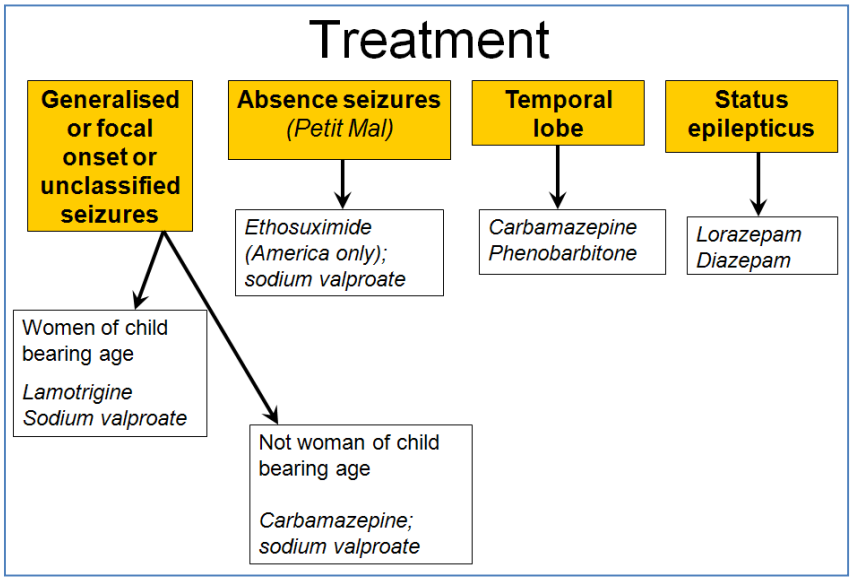
  - **"Partial with 2° Complex Generalised Seizure" ("Tonic-Clonic"/"Grand-Mal"):**
    - Ie. Simple *or* Complex Partial Seizure, Progressing to Complex (Unconscious) Widespread (Generalised) Seizure.
      - **4 Phases:**
        - **1. Pre-Seizure Period:**
          - Often involving Aura
        - **2. Tonic Phase:**
          - Sustained Tonic Contraction ('Rigid Extension') of all Muscles of the Body.
        - **3. Clonic Phase:**
          - Repetitive Synchronous Jerks ('Clonic') of all Muscles of the Body.
        - **4. Post-Ictal Coma:**
          - Patients may not regain consciousness for a while.
    - **Duration:**
      - 1-2mins

  - **"Myoclonic":**
    - Brief, Marked Contraction of Muscles (Ie. A "Shock-Like Jerk" or a "Startle")
      - Contraction may be restricted to a Specific Muscle Group or a number of Muscle Groups.
      - Typically Upper Body Muscles.
      - Generally Bilateral
    - **Duration:**
      - Typically 1-5sec

- **"Temporal Lobe Epilepsy":**
  - Unique type of Seizure.
  - Typically Manifests as some type of Behavioural Alteration or Complex Activity.
  - **Symptoms:**
    - **- Automatic Activity but Without Conscious Awareness:**
    - **- Sexually Inappropriate Behaviour:**
    - **- Aggression:**
    - **- Relived Experiences:**
  - **Treating Temporal Lobe Epilepsy - *Carbamazepine* (AKA: Tegretol)**is 1st Line**:**

- **"Absence Seizures" (The *Classic* "Petit Mal"):**
  - Ie. Abrupt Onset of Impaired Consciousness
  - **Duration:**
    - Up to 30sec
  - **NB:** Impaired Consciousness → Little/No Memory of Seizure.
  - **Treating Absence Seizures – *Ethosuximide*.**

- **"Status Epilepticus" – The Epileptic Emergency:**
  - **A term that describes An Episode of Seizures of Any Type that have 1 of 2 Properties:**
    - 1. Seizures Don't Stop Spontaneously.
    - 2. Seizures Occur in Rapid Succession Without Recovery.
  - **A *Status Epilepticus* Seizure = Absolute Neurological Emergency:**
    - High Risk of Cerebral Hypoxia
    - High Risk of Permanent Brain Damage
    - Often Results in Permanent Loss of Neurons due to Excito-Toxicity.
  - **Treating Status Epilepticus:**
    - ***Benzodiazepines* are the 1st Line Drugs:**
      - **The Drugs:**
        - ****Diazepam*** – (Generally #1; But Short Acting)
        - ***Lorazepam*** – (Some argue that it's #1 due to Higher Seizure-Termination Rate)
        - ***Midazolam***
      - **Mechanism of Action:**
        - ↑Frequency of GABA-Channel Opening (Agonist-Like but NOT an Agonist)
        - → Increased General Inhibition.
        - → Inhibits spread of Signals from Epileptic Focus



- **Anti-Epileptic Drugs:**
  - **VG-Na^+ Channel Blockers:**
    - **Phenytoin:**
      - (Use Dependent VG-Na^+ Channel Blockers)
        - (→ ↑Time of Recovery of Voltage-Gated Na^+ Channels from *Inactive* to *Resting* States)
      - All Seizures EXCEPT Absence Seizures
      - **An Adjunct to Benzodiazepines in Status Epilepticus.**
    - **Carbamazepine (Tegretol):**
      - (Use Dependent VG-Na^+ Channel Blockers)
        - (→ ↑Time of Recovery of Voltage-Gated Na^+ Channels from *Inactive* to *Resting* States)
      - All Seizures EXCEPT Absence Seizures
    - **Lamotrigine (Lamictal):**
      - (Use Dependent VG-Na^+ Channel Blockers)
        - (→ ↑Time of Recovery of Voltage-Gated Na^+ Channels from *Inactive* to *Resting* States)
      - All Seizures EXCEPT Absence Seizures

  - **VG-T-Ca^+ Channel Blockers:**
    - **Ethosuximide:**
      - Blocks VG-T-Ca^+ Channels in the Thalamus → Prevents Propagation of Seizure Activity through the Thalamus.
      - Used ONLY in Absence Seizures

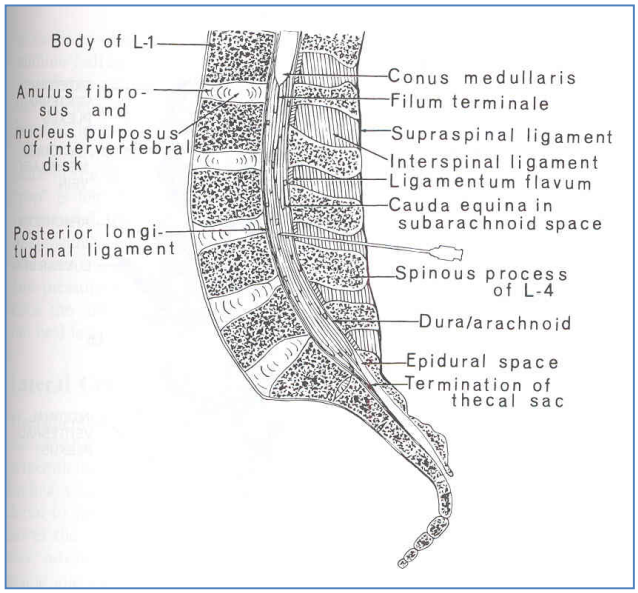
  - **GABA Channel Modulators:**
    - **Barbiturates (Primidone, Phenobarbitone):**
      - → Prolongs Opening of GABA Channel (But Not Frequency) → ↑Cl^- Influx.
        - (Like a chemical stent → Potentiates Channel Activation→ ↑Cl^- Influx.)
        - Ie. ↑Cl^- Influx → Hyperpolarisation →Stabilises Membranes of Neurons.
      - All Seizures EXCEPT Absence Seizures
      - **An Adjunct to Benzodiazepines in Status Epilepticus.**
    - **Benzodiazepines (Diazepam/Lorazepam):**
      - →Agonist-Like Effects → Causes Conformational Change in GABA Channel
      - →↑Frequency of GABA-Channel Opening:
        - Ie. ↑Cl^- Influx → Hyperpolarisation →Stabilises Membranes of Neurons.
      - **1ST LINE FOR STATUS EPILEPTICUS**

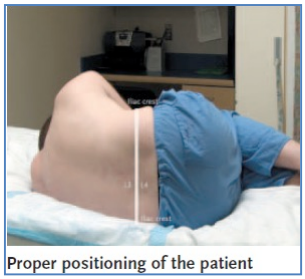
  - **GABA Analogues:**
    - **Gabapentin (Neurontin):**
      - A GABA-Analogue (But NOT a Receptor Agonist)
      - → General Neuronal Inhibition of the Brain.
      - Used for Partial Seizures

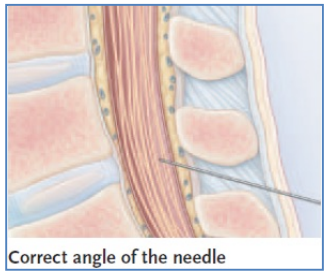
  - **Na^+ Channels, Ca^+ Channels & $GABA_A$ Receptors:**
    - **Valproate:**
      - **3 Mechanisms of Action:**
        - 1. Delays Recovery Time of Na^+ Channels
          - → ↓Repetitive Firing of Neurons.
        - 2. Inhibits Thalamic Ca^+ Channels
          - → Prevents Spread of Signals from Epileptic Focus.
        - 3. ↑GABA *Production* & ↓ *Breakdown*→ ↑ GABA (Inhibitory) Signalling.
          - → General Neuronal Inhibition of the Brain.
      - Effective Against ALL Seizure Types

CNS INFECTION:

- ***Meningitis*** **= Inflammation of the Meninges**
  - **Presentation:**
    - Meningism:
      - Headache
      - Photophobia
      - Neck Stiffness (Due to Inflammation of the Meninges; Bending Stretches Meninges → Pain → Neck Stiffness)
    - Nausea/Vomiting.
    - May eventually have loss of consciousness. (Rare)
  - **Typical Organisms:**
    - **Adults:** Nesseria Meningiditis.
    - **Neonates:** E.Coli
    - **Immunocompromised:** M.Tuberculosis, Toxoplasmosis
    - **Can also be Viral:** Typically Herpes Virus (Treatable by Aciclovir)
  - **Diagnosis:**
    - ***Treat on Suspicion***
      - Don't wait for lab results!
    - **Lumbar puncture for Definitive Diagnosis:**
      - **Performed @ L3-L4 (or L4-L5):**
        - Because the spinal cord Terminates at this level
        - → Becomes the Cauda Equina below the level of L3.
      - **Anatomical Landmarks:**
        - Draw a line visually between the Superior Iliac Crests
        - This line intersects @ the L4 Spinous Process.
      - **Flex the Spine as Much as Possible:**
        - To Maximise the space between the Spinous Processes → Provides a space to insert the Needle.
      - **Structures Between Skin & CSF (Focus on Ligaments):**
        - Skin
        - Subcutaneous Tissue
        - Supraspinous Ligament
        - Interspinous Ligament (Between the Spinous Processes)
        - Ligamentum Flavum
        - Epidural Space (Including the Internal Vertebral Venous Plexus, Dura, and Arachnoid)
        - Subarachnoid Space (CSF should flow once this has been entered)
      - **NB: DON'T do a Lumbar Puncture if Intracranial Pressure is High:**
        - If ICP is high, and you drain CSF → Can cause "Cerebral Herniation" (Aka: Cistern Obliteration)
        - → Brain can herniated through the foramen magnum → Puts extreme pressure on parts of the brain and thereby cuts off their blood supply.
        - Is often Fatal
      - **Signs of Raised Intracranial Pressure:**
        - ***Cushings Response/Reflex (Cushing's Triad):***
          - *Hypertension*
          - *Bradycardia*
          - *Irregular Breathing*
        - **Also Presents with:**
          - *Abnormal Posturing*
          - *Altered level of Consciousness (GCS 3-5)*
          - *Pupils may be Dilated & Fail to Constrict in response to light.*
          - *Vomiting*




Proper positioning of the patient


Correct angle of the needle

- **Good prognosis** with Aggressive Treatment.
    - **Treatment on Suspicion:**
        - Empirical Antibiotics (or Antivirals).
    - **Role of the *'blood brain barrier'* to CNS infection and its implication for treatment:**
        - NB: Most antibiotics aren't lipid soluble → don't cross the bbb.
        - However, in inflammation, the BBB permeability is increased.

- ***Encephalitis*** = **Inflammation of the Brain Parenchyma**
    - **Always Viral**
    - **Typical Presentation:**
        - *Altered Mental State*. (Or other neurological deficit)
        - Seizures
        - Fever
    - **Treatment on Suspicion:**
        - Empirical Antivirals.
    - **Poor prognosis:**
        - Once symptomatic, rapid inflammation & necrosis → Brain-Death or Neurological Deficit

- ***Meningo-encephalitis*** = **Inflammation of Both**
- ***Brain abscess*** = **Abscess in Brain**
    - Typically following Otitis Media

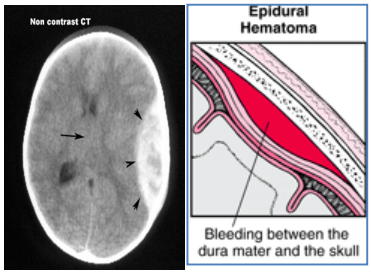
- **Diagnostic Distinctions Between the 3 CNS Infections:**

	Normal	Bacterial Meningitis	Viral Meningitis (Usually Herpes Virus)	Encephalitis (typically viral)
CSF Pressure	Normal	Normal-Raised	Normal-Raised	Markedly Raised
White Cell Count	Normal	Raised (Polymorphs)	Raised (Lymphocytes)	Raised (Lymphocytes)
Glucose	Same as Serum	Lower than Serum (Hungry Bacteria)	Normal	Normal
Protein	Normal	Raised (produced by the organisms)	Raised (produced by the organisms)	Raised
Gram Stain	None	Presence of Bacteria	Nothing ("Aseptic Meningitis")	Nothing

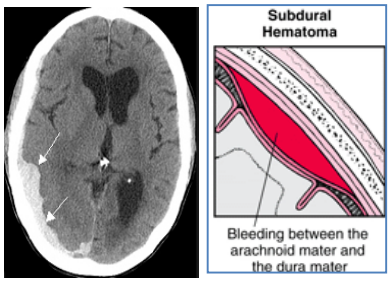
TRAUMATIC BRAIN INJURY (TBI) AND SPINE TRAUMA:

- (NB: Spinal trauma may or may not involve spinal cord injury)
- ***Mild*** = GCS 14 or 15
  - Likelihood of +ve CT scan = 10% and 1% will require neurosurgery
- ***Moderate*** = GCS 9-13
  - Likelihood of +ve CT scan = 40% and 8% will require neurosurgery
- ***Severe*** = GCS < 9
  - Mortality around 40%!

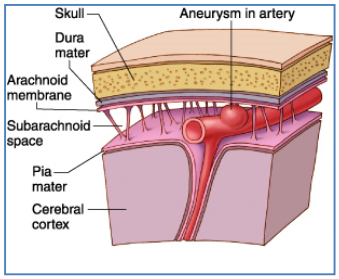
- **HAEMORRHAGES: Understand the Differences: YOU COULD GET A CT PICTURE OF ONE IN AN EXAM:**
  - ***Extradural Haematoma* (EDH):**
    - **Caused by:**
      - **Impact/Cranial Fracture** → Arterial Laceration →Separation of Dura from the Skull → Haematoma in this seperated region = EDH
      - Or Blood vessel bursts spontaneously
    - **Leads to:**
      - → Dissects the dura from the skull → ↑Intracranial Pressure
      - →Sudden collapse with a severe headache, vomiting and altered consciousness.
      - (NB: Normal brain underneath)
    - **Rapid progression** (due to arterial source of blood)
    - **Prognosis:**
      - If not treated →↑ICP →Cerebral Herniation (Coning), brainstem compression →Cerebral Infarct/Death
      - Treatable Via Craniotomy to Relieve the ↑ICP → Good Prognosis

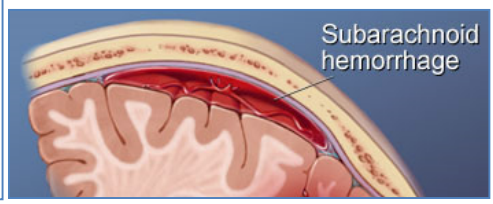


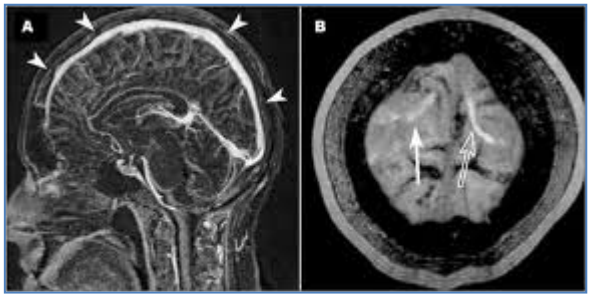
  - ***Subdural Haematoma* (SDH):**
    - **Haematoma is beneath the dura**
    - **Caused by:**
      - Mostly Old people with Low force trauma → Slow Venous Bleed
        - Because their brain is shrunken
        - Brain moves significantly under mild trauma
    - **Gradual onset** (:. Is often Chronic)
    - **Wide distribution** – ie. Over the entire hemisphere
    - **Presentation:**
      - Unsteady gait
      - Other abnormalities
    - **Prognosis:**
      - **Abnormal Brain underneath** (Ie. Pt. Is often demented, or alcoholic, or have severe cerebral atrophy)
      - Therefore the prognosis is worse



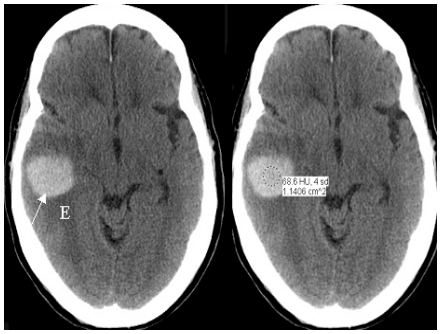
- ***Subarachnoid Haemorrhage*** **(SAH):**
  - **Caused by:**
    - Often due to Aneurysm of circle of willis
    - Hypertension is a Contributing Factor
    - (Arterial Bleed)
  - **Presentation:**
    - Typically a fit, well person who has sudden onset, severe headache (or feel like they've been hit in the head with a baseball bat)
    - Loss of Consciousness.







- ***Intracranial Haemorrhage*** **(ICH) ("Haemorrhagic Stroke"/CVA)**:
  - **Caused by:**
    - Non penetrating and penetrating cranial trauma.
    - →Haemorrhage directly into Brain Parenchyma → Stroke
  - **Presentation:**
    - Headache
    - Various Neurological Deficits
    - Loss of Consciousness
    - Vomiting
  - **Prognosis:**
    - 75% of Survivors are Disabled



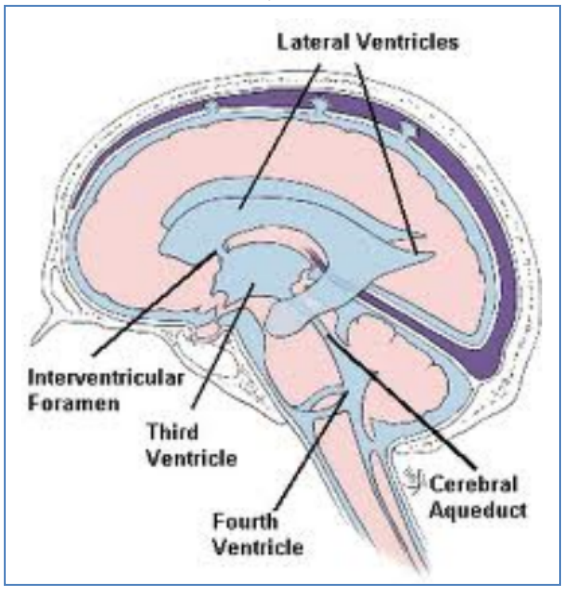
ASSESSMENT OF NEUROLOGICAL EMERGENCIES:

- ***Clinical:***
  - Try to determine ***nature*** and ***location*** of lesion (Which bit of the brain is affected)
- ***Laboratory investigation:***
  - Haematology (WBC count)
  - Biochemistry
  - Blood Culture (If suspect infection)
  - CSF (Via Lumbar Puncture)
- ***Radiology:***
  - CT
  - MRI

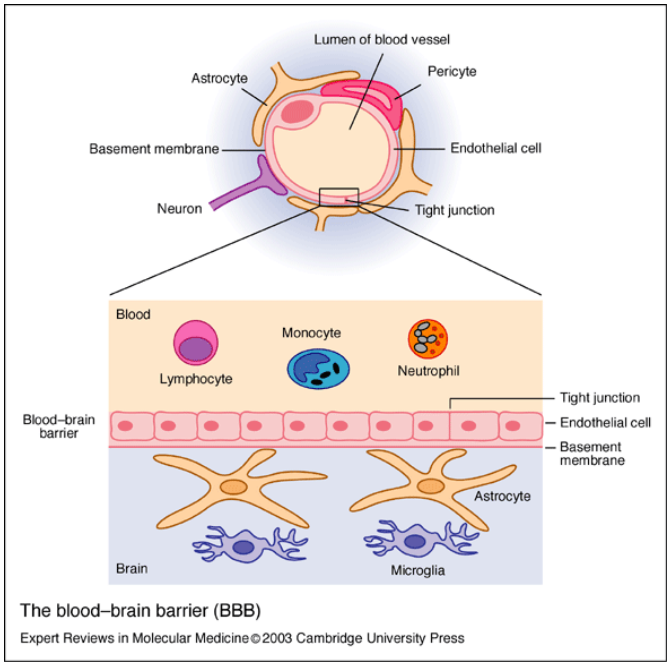
- **THE EMERGENCY-DEPARTMENT APPROACH:**
  - **Primary Assessment:**
    - Airway + Cervical Spine
    - Breathing
    - Circulation (Haemorrhage Control) & BP (Hypertensive Crisis or Shock)
    - **Check Glucose (exclude hypoglycaemia)
  - **Management Principles:**
    - **Objective:**
      - Primary injury has already happened :. prevent secondary injury.
    - **Ensure adequate:**
      - Oxygenation
      - cerebral perfusion
    - **C-Spine Precautions (If Trauma)**

CSF, CEREBRAL BLOOD FLOW, THE BLOOD BRAIN BARRIER AND INTRACRANIAL PRESSURE:

- **CerebroSpinal Fluid:**
  - **Functions Of CSF:**
    - Provides Buoyancy (Suspends the brain & displaces most of its mass)
    - Protection (Protects the brain tissue from injury when jolted or hit.)
    - Chemical Stability (In addition to the BBB) → Homeostasis of Neuro-Endocrine Factors.
  - **Formation Of CSF And Its Passage Within The Brain:**
    - **Site of CSF Formation:**
      - Produced by modified Ependymal Cells in the ***Choroid Plexus***.
    - **Site of CSF Absorption:**
      - It is reabsorbed into venous sinus blood via ***Arachnoid Granulations***.
    - **What happens if CSF Production Exceeds Absorption?**
      - →Hydrocephalus
      - → Increased Intracranial pressure
    - **Path of CSF passage between those two points:**
      - Produced in Choroid Plexus
      - →Circulates from the Lateral Ventricles
      - →**Through the Interventricular Foramen**
      - →Third Ventricle
      - →**Through the Cerebral Aqueduct**
      - → Fourth Ventricle
      - → **Through the Median Aperture** under the Cerebellum
      - → Into the Subarachnoid Space over the Brain & Spinal Cord.
    - **Point(s) of Potential Obstruction to CSF Flow → Resulting in Hydrocephalus?**
      - (Hydrocephals = abnormal accumulation of CSF in the Ventricles)
      - Due to blockage of CSF outflow in the ventricles or the Subarachnoid Space over the brain.
        - @ The Interventricular Foramen
        - @ The Cerebral Aqueduct (Between the Third & Fourth Ventricle)
        - @ The Median Aperture (Between the Fourth Ventricle & the Subarachnoid Space)

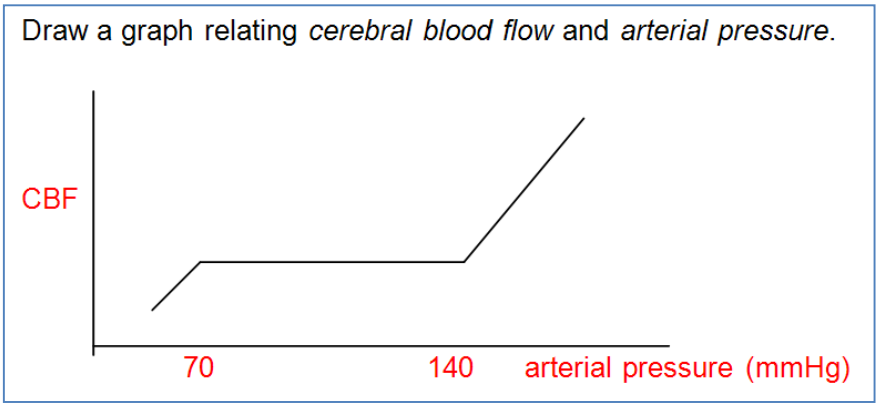


- **<u>The Blood Brain Barrier:</u>**
  - **What is it?**
    - Separation of the Circulating Blood & CSF in the CNS.
    - Tight Junctions between Endothelial Cells in CNS Capillaries.
  - **Where is it?**
    - It occurs along all the capillaries in the CNS.
  - **Functions?**
    - Restricts Diffusion of Microscopic Objects (eg. Bacteria) and Large &/or Hydrophillic molecules into the CSF.
    - Allows Diffusion of Small, Hydrophobic (lipophilic) Molecules (Oxygen, Hormones, CO_2).
    - NB: Metabolic products (Eg. Glucose) are actively transported by the Endothelial Cells.
  - **Implications in terms of Drug Treatment of CNS Pathology (e.g. treatment of meningitis)?**
    - Only Lipophilic Drugs can diffuse through the BBB.
    - NB: Most antibiotics aren't lipid soluble → don't cross the bbb.
    - However, in inflammation (Eg. Meningitis), the BBB permeability is increased anyway.



The blood–brain barrier (BBB)
Expert Reviews in Molecular Medicine ©2003 Cambridge University Press

- **<u>Cerebral Blood Flow And Intracranial Pressure:</u>**
  - Cerebral blood flow is carefully regulated under normal conditions.
  - **<u>Cerebral Blood Flow:</u>**
    - **What percentage of cardiac output goes to the cerebral circulation *at rest*?**
      - 750ml/min (15% of cardiac output)
    - **Relationship Between Cerebral Blood Flow & Arterial Pressure:**



- **Autoregulation of Cerebral Blood Flow:**
  - **What effect does a *high* P_{CO2} have on cerebral blood flow?**
    - Hypercarbia → Vasodilation → ↑Cerebral Blood Flow.
  - **What effect does a *very low and very high* P_{O2} on cerebral blood flow?**
    - Very High O2 → Vasoconstriction
    - Very Low O2 → Vasodilation
  - **What implication does this have for the *management* of a patient with an acute head injury/cerebral oedema?**
    - You want to prevent any hypercapnia because any vasodilation will → Takes up more room → ↑Intracranial Pressure.
    - You want to maintain PO2

- **Kelly-Monroe Doctrine:**
  - States that the Cranial Compartment is Incompressible, and the Volume is Fixed.
  - The Cranial Constituents (Blood, CSF, and Brain Matter) create a state of Volume Equilibrium:
    - Any increase in Volume of one of the constituents must be compensated by a decrease in volume of another.
  - **Volume Buffers:**
    - Both **CSF and**, to a lesser extent, **Blood** Volume.
    - (Eg. In Extradural Haematoma → CSF & Venous Blood Volumes are Decreased)
    - → Maintain normal ICP
    - Buffer Capacity ≈ 100-120mL

- **Intracranial Pressure:**
  - **What is Normal ICP?:**
    - 10mmHg
  - **When ICP is Rapidly Increased, what happens to the following:**
    - **Cerebral Venous Pressure?**
      - Remains same or slight Increase.
    - **Cerebral blood flow?**
      - Decrease (Due to reduced perfusion pressure)
      - (Perfusion Pressure = Sys.BP – Intracranial Pressure)
        - (NB: Perfusion only occurs when Perfusion Pressure is *Positive*)
    - **What Happens when *ICP* = Arterial Pressure?**
      - If Arterial Pressure = ICP…then Perfusion Pressure = 0.
      - Nil Perfusion
  - **Signs of Raised Intracranial Pressure:**
    - ***Cushings Response/Reflex (Cushing's Triad):***
      - *Hypertension*
      - *Bradycardia*
      - *Irregular Breathing*
    - **Also Presents with:**
      - *Abnormal Posturing*
      - *Altered level of Consciousness (GCS 3-5)*
      - *Pupils may be Dilated & Fail to Constrict in response to light.*
      - *Vomiting*
  - **Treating Raised ICP:**
    - **Osmotic Diuretics** (Eg. Mannitol) → ↑Plasma Osmolarity → Extracts Water from Brain Tissue.
    - **Hyperventilation** → Hypocapnia → Vasoconstriction of Cerebral Vessels
    - **Continuous CSF Drainage/Surgical CSF Shunt**

- **NB: DON'T do a Lumbar Puncture if Intracranial Pressure is High:**
  - **If ICP is high, and you drain CSF → Can cause "Coning":**
    - Aka. "Cerebral Herniation" (Aka: Cistern Obliteration)
    - → Brain can herniated through the foramen magnum → Puts extreme pressure on parts of the brain and thereby cuts off their blood supply.
    - **Is often Fatal**
    - **Can cause 3rd Nerve (Oculomotor) Palsy:**
      - Ptosis = Unable to Open Eyelid (Levator Palpebrae Superioris)
      - Pupil Unresponsive to Light
      - Eye faces *Downwards & Outwards*

Emergency Medicine Notes
Paediatric and Obstetric Emergencies

Overview:

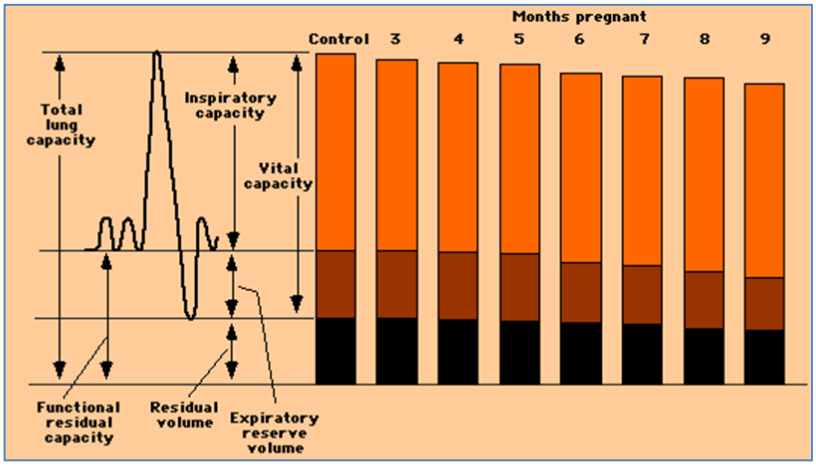
- What is normal and what is not?
- What is sick and what is not?

Exam Questions:

- **Physiological differences:**
  - Pregnancy
  - Childs Vs Adult (Airway, Breathing, Circulation)
  - Childs Vs Adult ECG
- **Given a case, Find abnormal parameters:**
  - Sick child
  - Pregnancy
  - Umbilical height
- NB: The care of a pregnant female is a priority because there are 2 lives at stake.

OBSTETRICS: CHANGES IN PREGNANCY:

- **(For Each of the systems):**
  - 2 things up
  - 1 thing down
  - And something else
- **Airway**
  - ↑Breasts
  - ↑Abdomen
  - ↓Airway Patency
  - Reflux
  - Facial Oedema
- **Respiratory:**
  - ↑ Tidal Volume
  - ↑ Respiratory Rate
  - ↓ Residual Volume
  - Alkalosis (Respiratory alkalosis due to Hyperventilation)
  - Dyspnoea
  - Foetal haemoglobin has a higher affinity for O_2 Than Mum's.
    - (Why? Foetal O2 is @ 30mmHg from the Placenta)



- **Cardiovascular:**
  - ↑ HR + SV
  - ↑ CO
  - ↓ Blood pressure
  - ECG – Left Axis Deviation (Due to Mechanical presence of baby → pushing heart out of the way)

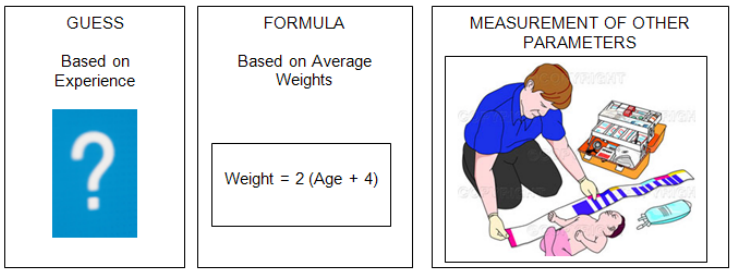
- **Haematological:**
  - ↑ Blood volume by 40% (But ↓in BP)
  - ↑ RBC Count
  - ↓ Hb (Dilutional Anaemia due to ↑Volume)
  - WBC (Marginal Increase)
- **GIT:**
  - ↑ Weight gain
  - ↑ Metabolic rate
  - ↓ Gut Tone & Motility (→ Constipation)
    - ↓Gastric Emptying + Abdominal Pressure→ Reflux
  - Diaphragm Raised
  - GI Contents Displaced Upward (Including Appendix)
- **Renal:**
  - ↑ Renal blood flow
  - ↑ Frequency of Urination (Due to Pressure on the Bladder)
  - ↓ Decreased Ureteric motility & peristalsis
  - Bladder displaced by uterus (NB: @ 20wks, the Uterus is at the Level of the Umbilicus)
- **Musculoskeletal:**
  - ↑Width of Pubis Symphysis & Flexibility of Sacroliliac Joints (Due to Relaxin)
  - ↑ Increased Foot Size (Due to Relaxin)
  - ↓ Ligament Tension (Due to Relaxin)
  - Unstable Gait

Relevance of Changes of Pregnancy:

- **Airway & Breathing:**
  - **Breathing:**
    - ↑Tidal Volume….↑Resp.Rate…..↓Residual Volume…..
    - →Burns oxygen MUCH faster because the Baby is breathing too
    - :. Very little time for Intubation. (Maternal O2 Sats drop *RAPIDLY* during apnoea)
  - **Airway:**
    - Facial changes
    - Large breasts and abdomen
    - Reflux
    - → Makes Intubation Difficult & ↑Risk of Aspiration
  - **MANAGEMENT:**
    - Intubation is Difficult
    - Give High [Oxygen]
    - Avoid Maternal Hypoxia (Avoid Foetal Hypoxia)
    - Early & RAPID Intubation
    - Full gut
- **Circulation:**
  - **They have More to lose (↑Blood Volume):**
    - :. By the time they're Tachycardic with Signs of Shock, they've lost a LOT (30%) of Blood.
    - Signs to look for include Urine Output, Capillary Return, Skin Colour/Temperature.
      - NB: Uteroplacental circulation Shuts down first (To Protect the Mother)
  - **May have Supine hypotension syndrome:**
    - = When they lie down, the weight of the baby blocks off the IVC.
      - → Poor venous return from the legs → Hypotension.
    - Prevented by Elevating the *RIGHT* Hip.
  - **Shock:**
    - Shock is **very** bad
    - → Utero-Placental Circulation Shuts Down →85% foetal mortality
    - **Treated by:**
      - **AGGRESSIVE Fluids (Hartman's Solution) and/or Blood**

PAEDIATRICS:

- **Definitions:**
  - **Neonate =** birth to 4wks
  - **Infant =** 4wks to 1yr
  - **Child =** Over 1yr to adolescence
- **What's Important?**
  - **Weight:**
    - **Determines everything:**
      - Drug Dos
      - Selection of Equipment
      - Fluid Resuscitation
    - **Methods of Estimating Weight:**
      - Broselow Tape Method (A plastic strip with different sized-colour-codes)
      - Formula (Based on Average Weights @ Age)
      - Guess
      - **Disadvantages of Methods:**
        - Access – do you have a Broselow Tape?
        - Reliability – can you do maths when stressed ? (or remember the formula)
        - Accuracy – some are rough measures only
        - Accuracy across cultural groups – are methods always reliable e.g. malnourished child in Sudan vs a well nourished child in Australia



  - **Child's Airway Vs Adult's Airway:**
    - Head (Much Bigger & Lolls about)
    - Neck (Relatively Short)
    - Nose (Children are Nasal Breathers :. Give O2 through nose rather than mouth)
    - Teeth (Always Loose – Easy to knock out with Laryngoscope)
    - Tongue (Relatively big – Makes intubation harder)
    - Tonsils (Huge, Often Inflamed/Infected)
    - Epiglottis (Long, Floppy & hangs out the front)
      - (:. You have to lift the whole epiglottis with the laryngoscope when intubating)
    - Larynx (More Anterior)
    - ***Cricoid (The Narowest part of a Child's Airway)(Good MCQ question)***
    - Trachea (Quite Short)(:. Don't feed the laryngoscope in too far)
  - **Child's Breathing Vs Adult's Breathing:**
    - Lower Respiratory Surface Area
    - Have a Flat Diaphragm and can't increase their tidal volume.
    - **If Hypoxic, What can they increase?**
      - Can ONLY increase their Resp. Rate
      - :. If Resp Rate is High – Give them Oxygen!!!
  - **Child's Circulation Vs. Adult's Circulation:**
    - Larger circulating volume/kg
    - Lower Stroke Volume :. Require Higher Heart Rate to maintain CO.
    - CO=SV x HR
    - **If ↓CO, What can they increase?**
      - They can *ONLY* increase Heart Rate.
      - :. If they're tachycardic, give them fluids!!!

- **Primary Paediatric Assessment (ABC):**
  - Observe from a Distance.
  - Remain Calm
  - Get Parents Help.
  - **Fix A before B before C!!**
    - Give O2 before IV access.
  - **Airway**
    - Observe
    - Speech = Patent Airway
    - Look for Chest Movements
    - Listen for Chest Sounds
    - Feel for Breath
  - **Breathing**
    - Normal Resp Rate decreases by 10 each 2yrs of life until 8 yrs.

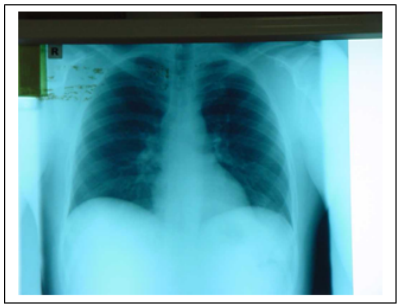
Age (years)	Respiratory Rate (bpm)
<1	<50
2	<40
4	<30
8	<20

    - Look for:
      - Recession and accessory muscles
      - Abnormal Respiratory rate
      - Inspiratory/Expiratory Noises (Stridor)
      - Grunting and sitting
      - Nasal flare
      - chest expansion
      - HR (Tachy)/skin colour(Blue)/mental state(Agitated/Drowsy)
      - Oxygen Sats
  - **Circulation:**
    - Focus on Heart Rate (Goes down 20 every 2yrs of life)

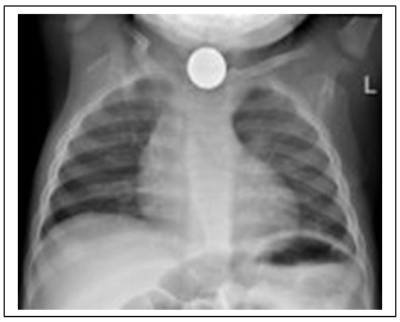
Age (years)	Heart Rate	Blood pressure
1	<160	>70
2	<140	>75
4	<120	>80
8	<110	>90

      - BP isn't really relevant in young kids
    - If Abnormal:
      - Needs IV access
      - ?Dehydration
        - Tachycardic
        - Dry mouth
        - ↓Urine Output
      - →Give Bolus amounts of fluid (10mls/kg)
- **Secondary Paediatric Assessment:**
  - Focused history, exam, investigation
  - Glucose
  - Close monitoring
  - Is the child ill or not
  - Take parents concerns seriously
  - Play, Observe, Respect

- **Paediatric Radiology:**
  - (1) ribs more horizontal
  - (2) thymus enlarged in childhood so may have bigger mediastinum
  - (3) more likely to have prominent gas bubble under diaphragm
  - (4) bigger right ventricle (compared to adult) means different heart silhoutte



Adult Chest XRay



Child Chest XRay

- **Paediatric ECG:**
  - **NB:** The Right Ventricle is Dominant in Utero, but the Left Ventricle is Dominant in Adults.
    - :. Babies initially have a larger Right-Heart → Causes an apparent Axis Shift (Right Axis Deviation) in Early Childhood.
  - **↑Heart Rate**
  - **T-Wave Inversion**

- **Practice MCQ Questions:**

1. Infants differ from adults in that they:
    a. have a relatively smaller tongue
    b. the ribs contribute more to chest expansion.
    c. have a higher proportion of HbA
    d. have a higher circulating volume per kilogram.
2. Upper airways obstruction:
    a. is less likely in the small child because of the large cross sectional area
    b. can be assessed by the intensity of the stridor
    c. is usually due to foreign body inhalation
    d. may be due to epiglottitis
3. Cardiac arrest in children:
    a. is usually due to primary myocardial disease
    b. usually presents in ventricular fibrillation
    c. can be due to shock
    d. has a very good prognosis
4. Treatment of shock in children involves all the following except:
    a. High flow oxygen by face mask
    b. Correction of metabolic abnormalities
    c. Reduction of low body temperature
    d. Optimisation of intravascular volume
1. The tidal volume in pregnant women
    a. increases by 20%
    b. increases by 40%
    c. increases particularly in women with asthma
    d. increases proportionally to the women's weight
2. The cardiac output of the average size healthy woman in pregnancy is
    a. increases to 8L/min
    b. *increases to 6L/min
    c. increases to 4.5L/min
    d. increases to 4l/min
3. The haemoglobin of the average healthy woman in pregnancy is
    a. 16 g/dL
    b. *13g/dL
    c. 9g/dL
    d. resistant to hypoxia
4. Foetal oxygen from the placenta is
    a. 90mmHg
    b. 70mmHg
    c. 50mmHg
    d. *30mmHg
5. All the following are true except
    a. Pulmonary embolus is more common in pregnancy
    b. Hyperemesis gravidarum is more common in twin pregnancies
    c. *Acute appendicitis is more common in pregnancy
    d. Urinary tract infections are more common in pregnancy
6. The most common causes of death in childhood in western countries are
    a. congenital
    b. infections
    c. cancers
    d. *trauma
7. The following are true of children's airways except
    a. They have short necks
    b. Their epiglottis is more anterior
    c. Their larynx is higher
    d. *They are narrowest at the vocal chords

8. Flow is inversely proportional to
    a. r
    b. r2
    c. r3
    d. *radius^4
9. Normal capillary refill is
    a. less than 1 second
    b. *less than 2 seconds
    c. less than 4 seconds
    d. less than 5 seconds
10. Children with inadequate circulation should initially be given
    a. 20ml/kg 5% glucose
    b. 10ml/kg 5% glucose
    c. *20ml/kg normal saline
    d. 10ml/kg normal saline
11. A woman comes into the emergency department who is 32 weeks pregnant. She has been involved in a motor vehicle collision. She was wearing her seatbelt. Her pulse is 100 bpm. Her blood pressure is 110/75. All the following are true except:
    a. She should have high flow oxygen applied.
    b. She should have a large bore cannula inserted.
    c. She should have cardiotochographic monitoring for 4 hours.
    d. *She should have an urgent foetal ultrasound.

A pregnant woman has respiratory acidosis – T/F?
- F

Differences between child & adult:
- Child has more horizontal ribs
- Child has flatter diaphragm
- Bigger heart
- Short trachea

At how many weeks is the uterus palpable during pregnancy?
- 12 weeks

Children have a relatively small head compared to the body – T/F?
- False

By the 3rd trimester, blood volume has increased by:
- 750mls
- 1L
- 1.5L

The narrowest part of the child's airway is:
- The Cricoid Cartilage

Residual Volume in a pregnant female increases/decreases?
- Decreases

In a 2 yr old, a resp rate of what amount would worry you?
- 40

Maternal ruptured appendicitis has a foetal death rate of?
- 35%

2 ways of estimating weight?
- Broselow
- 2x Height + 8

Being pregnant is an independent risk factor of domestic violence – T/F?
- T

A HR of 120 in a 1yr old is normal/abnormal?
- Normal

3 Reasons for difficulty intubating a heavily pregnant female?

- Large Breasts
- Facial Oedema
- Aspiration from reflux
- Quick Hypoxia

List 5 differences between adult's and child's airway?

- **Child's Airway is:**
    - Larger tonsils
    - Nasal Breathers
    - Epiglottis is floppy and more anterior
    - Trachea is shorter
    - Big Tongue

An ECG of a pregnant female @ 36weeks shows R/L Axis Deviation?

- L

How much should a 4yr old weigh?

- 16kg

You should put a block under which hip of a supine pregnant woman?

- Right

3 clinical features of a dehydrated child:

- Tachycardia
- Dry mouth
- Loss of skin elasticity
- Slow cap return
- Tachypnoea
- Concentrated Urine

What is the appropriate bolus amount of fluid resus for a dehydrated child?

- 20mls/kg

Post Op Complications

Early – (In Recovery):

- **Nausea/Vomiting:**
  - <20% of All Patients
  - Risk Factors = Young, Female, Motion Sickness
  - Causes = Nitrous Oxide & Opioids.
  - Rx: Antiemetics (Odansetron/Metaclopromide)
- **Respiratory:**
  - Resp. Depression (Opioids/Residual Neuromuscular Block)
  - Aspiration (Gastric Contents/Loss of Cough Reflex)
  - Airway Obstruction (Laryngospasm/Foreign Bodies/Clot)
  - (Atelectasis/Pneumothorax/PE)
- **CVS:**
  - Hypotension (Hypovolaemia/Haemorrhage/3rd Space) (LV Failure/IHD) (ACE-Is)
  - Hypertension (Pain/Stress/Hypoxia/Overhydration) (Pre-Eclampsia/↑Thyroid/↑ICP)
  - Arrhythmias (Electrolytes/Drugs)
- **Pain:**
  - **Rx.** Opioids
- **Hypothermia:**
  - **Rx.** Warming, Oxygen, IV Pethidine.

Late – (In Ward/Post Discharge):

- **Wound Complications:**
  - Haematoma (Poor Haemostasis) (Predisposes to Infection) (BAD in the Neck!!)
  - Infection [Day 5-10] (Diabetic/Steroids/Smoker, etc) → Cellulitis. **Rx.** Fluclox/Gent/Vanc
  - Dehiscence (Infection/Poor Closure/Suture #) → Hernias
- **Respiratory:**
  - Atelectasis [Day 2] (Elderly/Obese/Smokers) → Fever, Tachypnoea, Tachycardia. **Rx:** Spiro
  - Pneumonia (Atelectasis/Aspiration) → 30% Mortality. **Rx.** O2 + Cefalexin
- **Vascular:**
  - DVT (Stasis + Vascular Injury + ↑Coagulation) (Age/Ca./Surgery/OCP/HRT/CVD) → Pain & Oedema. **Ix.** Doppler US. **Rx.** Heparin + Warfarin.
  - PE (DVT → Pulmonary Arteries) → RVF → Dyspnoea/CP/Tachy/↓BP. **Ix.** CTPA **Rx.** **Thrombolysis** + Heparin. Or Pulmonary Embolectomy
- **CVS:**
  - MI (Vascular Surg/↓BP/Hypoxia) → Usually Silent + ST-Dep. **Ix.** Tn-I. **Rx.** O2 + Morphine + GTN + Aspirin.
- **Abdo:**
  - Ileus (Physical Bowel Handling/Opioids/AAA Surg). **Rx.** NBM +/- NG-Tube
- **CNS:**
  - Confusion (Elderly) (Hypoxia/Drugs/Alcohol Withdrawl/Sepsis/UTI/Full Bladder/↓BSL)
- **Fever:**
  - See "Post-Op Fever +/- Infections"

Post Op Fever +/- Infections

The 5 W's:

- Day 1: ***Wind*** (Post-op Atelectasis/Pneumonia)
- Day 2: ***Water*** (UTIs from catheters)
- Day 3: ***Walking*** (DVT – can → Fever/PE)
- Day 4: ***Wounds*** (Wound Infections)
- Day 5: ***Wonder Drugs*** ??

Prevention of the 5 W's:

1. **Wind:**
    - **a.** Early Ambulation (Atelectasis)
    - **b.** Spirometry (Atelectasis)
    - **c.** Antibiotics (Pneumonia)
2. **Water:**
    - **a.** IDCs out ASAP
3. **Walking:**
    - **a.** DVT Prophylaxis
        - **i.** Heparin/LMWH
        - **ii.** Graduated Compression Stockings
    - **b.** Early Ambulation
4. **Wounds:**
    - **a.** Antibiotic Prophylaxis (Typically Cefalexin / Gentamicin / Metronidazole)
        - **i.** Or Triple Therapy: Ampicillin + Gentamicin + Metronidazole
    - b. Dressings/IDC/IVC/Drain Surveillance (Keep Clean & Dry)
5. **Wonder Drugs:**
    - **a.** Analgesia
    - **b.** NB: Some drugs can cause fever

****NB: Low-Grade Fevers are common Post-Op...Why?**

- Atelectasis
- Tissue Damage/Necrosis
- Blood Transfusions
- DVT
- *Or Infection...*

Infection Screening:

- **Common Sites of Infection:**
    - Abdo → Peritonism
    - Chest → Cough/↓Sats/Crackles
    - UTI → Dysuria/Frequency/Urgency/Pelvic Pain
    - Wound/Cannula/Drain Site
    - Meningism → Headache/Photophobia/Neck Stiffness
    - Endocarditis → Splinters/Janeways/Oslers
- **Phys Examination:**
    - Vital Signs (Fever, Tachycardia, Hypotension)
    - Listen to Chest (Crepitations, Wheezes)
    - IDC/IVC/Drains (Erythema, Pain, Pus, Heat, Oedema)
        - NB: Superficial → Cellulitis
        - NB: Deep → Fluctuant Abscess
    - Wounds (Erythema, Pain, Pus, Heat, Oedema)
    - Drug Chart
- **Investigations:**
    - FBC (WBC count)
    - Cultures (Blood/Wound/Sputum)
    - Urine MCS
    - CXR (Consolidation/Collapse)

Treatment:

	Egs:	**Effective Antibiotics:**
G. Positives ("-cocci") (Skin / Throat)	Enterococcus Spp. Staphylococcus Spp. Streptococcus Spp.	**Penicillins** (Benz-Pen-G, Amoxicillin, Ampicillin, Flucloxacillin) - (NB: **Augmentin** for β-lactamase resistant bacteria = Amoxil + Clavulonate) **Cephalosporins** (Ceftriaxione[3], Cefipime[4], Cepfalexin[4]) **[Vancomycin** (For resistant G-Pos/ if Penicillin Allergy)]
G. Negatives (GI / UTI)	E. Coli Neisseria Spp. Pseudomonas Haemophilus Spp. Klebsiella Spp. Enterobacter Spp.	**Aminoglycosides** (**Gentamicin**, Tobramycin, Streptomycin) - (NB: Used with Penicillins/Cephs for *Synergy*) **Tetracyclines** (Tetracycline, Doxycycline) **Macrolides** (Erythromycin, Azithromycin) **Quinolones** (Ciprofloxacin, Norfloxacin) **Cephalosporins** (Ceftriaxione[3], Cefipime[4], Cefalexin[4]) **[Benz-Pen-G** (For Neisseria Gono/Mening)]
Anaerobes	Bacteroides Spp. Clostridium Spp.	**[Metronidazole** (For Bacteroides)] **[Vancomycin** (For C.Diff)]
Atypicals	Mycoplasma Legionella	**Tetracyclines** (Tetracycline, Doxycycline) **Macrolides** (Erythromycin, Azithromycin)

Specific Infections & Rx:

- **Cellulitis/Wound Infections:**
  - **G-Pos's** (GABH-Strep/Staph. Aureus)
    - **Rx: Penicillin (Flucloxacillin;** Gentamicin if Penicillin Allergic)
    - **(Rx: Vancomycin** if MRSA)
- **Abdominal Infections:**
  - **G-Negs** (E.Coli, Klebsiella, Enterobacter)
    - **Rx: Gentamicin / Doxycycline**
  - **Anaerobes** (Bacteroides, Clostridium)
    - **Rx: Metronidazole** (For Bacteroides)
    - **Rx: Vancomycin** (For C.Diff)
  - **G-Pos** (Enterococci)
    - **Rx: Ampicillin**
- **UTIs:**
  - **G-Negs** (E.Coli, Klebsiella, Enterobacter, Pseudomonas)
    - **Rx: Ciprofloxacin**
- **Bone (Osteomyelitis/Septic Arthritis):**
  - **G-Pos** (Staph. Aureus/Strep)
    - **Rx: Penicillin (Flucloxacillin;** Gentamicin if Penicillin Allergic)
    - **(Rx: Vancomycin** if MRSA)

NB: Triple Therapy: –Ampicillin, Gentamicin, Metronidazole = 'Broad Cover'. (Remember by AGM – Annual General Meeting...If you want to be around next year, take these 3)

Emergency Medicine Notes
Psychiatric Emergencies

Overview of Psychiatry in Emergency Medicine:

- Presentation
- Initial assessment
- Implications, to individuals and community, of:
  - mental illness
  - suicide
  - violence
- ''Medical Clearance''
  - Organic vs non-organic mental health illness
- Mental Health Act
  - Deprivation of liberty vs. protection/safety of Community

Definition of Mental Illness:

- "A condition characterised by a clinically significant disturbance of thought, mood, perception or memory"
- Emergency: indicates that assessment and intervention are required within a short timeframe

Illness Detection:

- **Physical/ "Organic":**
  - Hx, Physical Exam, Investigations
- **Mental:**
  - Interpret speech and behavior to establish abnormal thought paterns
  - (In addition to the above)

Possible Presenting Symptoms:

- **Behaviours:**
  - Self Harm
  - Aggression
  - Bizzare Actions
- **Emotions:**
  - Distress
  - Anger
  - Worry
- **Thoughts:**
  - Suicidal Ideation
  - Delusions
- **Physical:**
  - Agitation
  - Overactivity

Multi-Axial (DSM) Assessment of Psychiatric Illness:

- **Axis 1:**
  - Clinical Disorders (Other conditions that may be a focus of clinical attention)
- **Axis 2**
  - Personality disorders
  - Mental retardation
- **Axis 3**
  - General medical conditions
- **Axis 4**
  - Psychosocial and environmental
- **Axis 5**
  - Global assessment of functioning state

Mental Health Assessment has 4 parts:

1. History and Corroborative History
2. Mental State Examination (MSE)
3. Physical Examination
4. Investigations

Safety:

- Can I safely interview this patient on my own, or do I need backup?
- Where is the most appropriate place to interview the patient given their level of arousal/agitation?
- Is the Patient going to be safe.
- Take away sharp objects
- Metal detectors
- Can they be left alone safely?
- What degree of observation do they need?

Mental State Examination:

- **Appearance & Behaviour:**
  - Clothing – Appropriate, Clean, Torn?
  - Grooming – Unkempt, well groomed, Smelly?
  - Posture – Slumped, Rigid, Upright?
  - Eye Contact – Good, Avoiding, Limited?
  - Facial Expression – Sad, Animated, Anxious?
  - Motor Activity – Decreased, Increased, Lethargic?
  - Reaction to Interviewer – Hostile, withdrawn, operative, guarded, uncommunicative?
- **Speech:**
  - Rate – Normal, fast, slowed, pressured
  - Volume/Tone – Quiet, loud, Whispered
  - Quantity – Poverty of speech, Monosyllabic, excessive
  - Continuity – Can they maintain normal progression from 1 stream of thought to the next?
- **Mood & Affect:**
  - **Mood** (Patient Reported) – Depressed, Euphoric, Suspicious, Elated, Angry, Anxious, Perplexed
  - **Affect** (Clinician Assessed) – Restricted, Flattened, Inappropriate, Blunted, Anxious
  - **NB: Does *Mood* Match *Affect*?**
- **Thought Form:**
  - Continuity of Ideas
  - Disturbance in language or meaning
  - Goal-directed
  - Flight of ideas
  - Thought blocking
  - Preoccupied
- **Thought Content:**
  - Delusions
  - Overvalued Ideas
  - Pre-Occupations
  - Phobias
  - Suicidal Ideas
- **Perception:**
  - Hallucinations (Auditory/Visual)
  - Derealisation
  - Depersonalisation
  - Illusions
- **Insight & Judgement:**
  - Partial Insight – Aware they have a problem, but believe someone else is responsible
  - No insight
  - Judgement – person's ability to measure the consequences of their actions and how their behavior may affect themselves/others.

- **Components of Cognition:**
    - **Mental Status Questionnaire (MSQ)**
        - Orientation
        - Score out of 10 (8-10 = normal)
    - **Folstein's Mini-Mental State Exam:**
        - Orientation
        - Registration
        - Attention & Calculation
        - Recall
        - Language
        - Score out of 30 (27-30 = Normal)

Interaction of *Organic Disease* & Mental Illness:

- Organic diseases can cause apparent Mental Illness
- Examples of Organic causes of Mental "Illness":
    - Hypoxia
    - Hypoglycaemia
    - Trauma
    - Sepsis
    - Metabolic/Endocrine Disorders
    - CNS Infection/Trauma/CVA
    - Intoxication/Withdrawal
    - Medications (Side Effects/Toxicity)

TYPES OF EMERGENCIES:

Suicide & Self-Harm:

- **Number of factors have increased risk of suicide:**
  - Eg. Feelings of hopelessness, worthlessness, isolation
  - Impulsiveness
  - Psychodymanics/Psychological Vulnerability
  - Life Stressors
  - Access to Weapons
- **Who is At Risk?**
  - Male
  - Youth
  - Rural
  - Aboriginal
  - Depressed
  - Shizophrenics
  - Other Mental Illness
  - Substance Abusers
  - Personality Disorders
- **Males Vs Females:**
  - More males than females
  - ≈20/100,000 population
- **Assessment of Suicide/Self-Harm Assessment:**
  - **"Sadperson's Scale":**
    - Number Scoring System
    - Higher Score = Higher Risk.
    - Pneumonic:
      - Sex
      - Age
      - Depression
      - Psychiatric Care
      - Excessive Drug Usage
      - Rational Thinking Loss
      - Separated/Single
      - Organised Attempt
      - No life Supports
      - Stated Future Intent

RISK FACTOR	POINT	HIGH RISK CRITERIA	SCORE
Sex	1	Male gender associated with higher risk	
Age	1	Young and old are associated with higher risk (<19 yrs or >45 yrs)	
Depression	2	Feelings of worthlessness, hopelessness, despair especially associated with dysfunction of sleep, libido, eating etc.	
Psychiatric care	1	Previous suicide attempts or a past history of inpatient or outpatient psychiatric care, severe personality disorder	
Excessive drug usage	1	History of alcohol or other drug abuse	
Rational thinking loss	2	Association with severe depression or psychotic illness or organic brain syndrome associated with high risk. Delusions - high risk.	
Separated, single	1	Single, separated, divorced or widowed increases the risk	
Organised Attempt	2	Serious life-threatening suicidal attempt or suicidal ideation with a specific well organised plan are at very high risk of "success"	
No Life Supports	1	Socially isolated with no supportive family, close friends or religious affiliation, unemployed, NFA etc	
Stated Future Intent	2	Stated determination to repeat the attempt when possibly is a very high risk.	
Total Score	14	< 6 - low risk; 6-8 psych. review; > 8 high risk.	

  - **Psychiatric Examination:**
    - Risk level Assessment: (Risk Factors Vs. Protective Factors)
      - Low/Med/High
    - Specific Suicide Inquiry

Violence:

- **Many Causes (not just Psychiatric):**
  - Medical
  - Situational
  - Criminal
- **Affects All:**
  - Patients,
  - Families,
  - Health Workers
- **Why are ED's at High Risk?**
  - Open 24hrs
  - Easy access
  - Patients in crisis situations
  - Long waiting times
  - Overcrowding
  - Inadequate security
- **Management:**
  - Staff must Anticipate potential violence.
  - Staff must act in defensive manner prior to the violence.
  - Patient Placed in Quiet/Secure area
  - Verbal Intervention
  - **Use of Restraints (Eg. Verbal/Oral Sedatives/Physical Restraints) if:**
    - Imminent harm to Patient or others
    - To allow evaluation/Investigation/treatment of patient
    - At patient's request
    - To prevent harm to the environment of the ED
    - **Legal Considerations:**
      - Adult Guradianship laws
      - Pt/Staff/Visitor Protection Vs. Pt Freedom and Autonomy.

IMPACT OF MENTAL HEALTH EMERGENCIES:

On Individuals:

- Stigma/Discrimination
- Affects Employment
- Relationships (Family/friends)
- Finances
- Self-Esteem
- Lifestyle (eg. Drug Use)

On Families:

- Time off work
- Grief
- Loss of income
- Role of Carer
- Stigma of mental illness

On Healthcare Workers:

- Burnout
- Suicide
- Stressors

GLS – Grief & Loss:

- **Grief:**
  - Determined by Culture, Age, Gender, Physical & mental Health
  - Everyone Grieves differently.
- **Dimensions & Manifestations of Grief:**
  - **Physical:**
    - Pains
    - Sleep disturbance
    - Changes in eating patterns + Weight-Loss/Gain.
    - Stomach aches/headaches
    - Extreme fatigue
    - Chest pains
    - Breathlessness
  - **Emotional:**
    - Self-blame, Guilt
    - Sadness, Numbness
    - Loneliness, Yearning
    - Fear, Crying
    - Anxiety
    - Numbness
    - Ager
    - Helpless/hopelessness
    - Sometimes Relief (if the loss has been a long time coming – Eg. Terminal Illness)
  - **Cognitive:**
    - Nightmares/dreams
    - Memory loss
    - Decreased attention span
    - Disbelief and confusion
    - Preoccupation with the event
    - Magical thinking
    - Wishing to die
  - **Behavioural:**
    - Regressive/Aggressive
    - Withdrawal
    - Overactivity
    - Self-destructive behaciours
    - Obsessive Acitivity
  - **Spiritural:**
    - Utilization of spiritural beliefs
    - Abandonment of spiritual beliefs

- **Secondary Sources of Loss/Stress:**
  - **Financial problems**
  - **Legal issues**
  - **Single parent**
  - **Loss of role (**I've been a husband for 10yrs. Who am I now?**)**.

- **Models of Understanding Grief:**
  - (Attempt to explain the complex process experienced after significant loss & change.)
  - Eg. Sigmund Freud
  - **Currently: "The Dual Process Model (DPM):**
    - Acknowledges importance of Grief Work
    - **- A *Stressor-Specific* Model of Coping that focuses on 2 Types of Stressors:**
      - 1. Loss Orientation
      - 2. Restoration Orientation

Loss Orientated (Early Phase)	Restoration Oriented (Late Phase)
Grief work	Attending to Life Changes
Intrusion of Grief	Doing New Things
Denial/Avoidance of Restoration Changes	Distraction from Grief

- **Adjustment:**
  - Adjustment to loss is a natural & normal process.
  - Generally, people rely on Informal Resources for Support (Family, Friends, Colleagues, etc)
  - Generally, they do Not require Formal Supports (Counselling & Medication)
- **Resilience:**
  - The most Common Trajectory
  - Multiple Pathways to resilient outcome
    - Some Suffer chronic grief symptoms after the 1st year
    - Recovery trajectory
    - Very rare to have no symptoms at all
    - Doesn't seem to be delayed grief reactions.
  - Pragmatic Coping:
    - Resilient people tend to express less negative emotion while discussing loss compared to others.
    - Capacity to minimize the expression of grief-related emotions may help to minimize grief.
  - Protective Factors:
    - Large Social Network
    - Positive Emotion
- **When To Seek Support:**
  - YOU feel you need it
  - NB: Just because they're sad, doesn't mean they need a social worker.
  - Maladaptive coping strategies (eg. Alcohol/drugs/compulsivity/etc)
  - Thoughts of suicide or self destruction
  - Feeling alone/helpless
- **What does a Bereaved Person need from you?**
  - Generally, all you need to do is listen to how much it hurts.
  - Sometimes, just your presence is enough

Emergency Medicine Notes
An Approach to Respiratory Emergencies & Their Assessment.
(+ Blood Gasses & Chest Xrays)

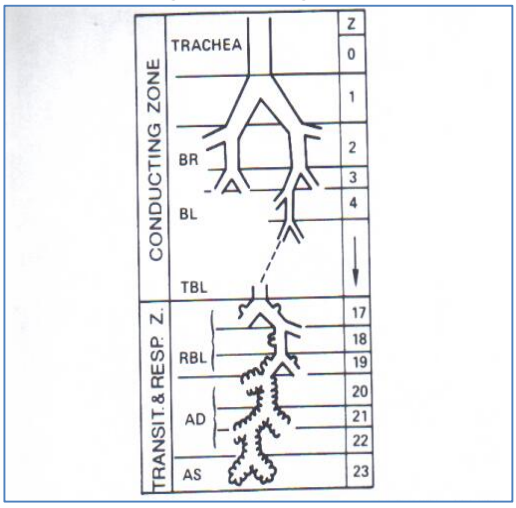
Overview

- **Keywords:**
  - **Pulmonary Embolus:**
    - A blockage of the main artery of the lung by an embolism. (Usually a thrombus)
    - The obstruction of the blood flow →↑Pressure on the R-Ventricle→ PE Symptoms.
      - Dyspnoea, Chest Pain, Palpitations, Low Sats, Cyanosis, Tachypnoea, Tachycardia.
  - **Pulmonary Oedema:**
    - Fluid Accumulation in the lungs → Collects in Air Sacs →Dyspnoea
      - →Impaired gas exchange
      - →Possible respiratory failure
  - **Pulmonary Contusion:**
    - A contusion (bruise) of the lung, caused by chest trauma →Accumulation of Blood and other fluids in the lung tissue.
    - The excess fluid interferes with gas exchange, potentially leading to hypoxia.
  - **ARDS – Acute Respiratory Distress Syndrome:**
    - A serious reaction to various forms of injuries to the lung.
    - Characterized by Inflammation of the lung →Impaired gas exchange + systemic inflammation
      - →Hypoxemia
      - →Frequently results in multiple organ failure.
    - (Often fatal, usually requiring mechanical ventilation for survival)

LECTURE:

Revision of Respiratory System:

- **Conducting Vs. Respiratory Zones:**
  - **Conducting Zone:**
    - The airways
    - Extend to the terminal bronchioles
    - They lead air to the Respiratory Zone (gas exchanging region)
    - Responsible for the anatomical dead space (volume 150 ml)
  - **Respiratory Zone:**
    - The *Respiratory Unit* (Respiratory Bronchioles + Alveoli)
    - Where gas exchange occurs



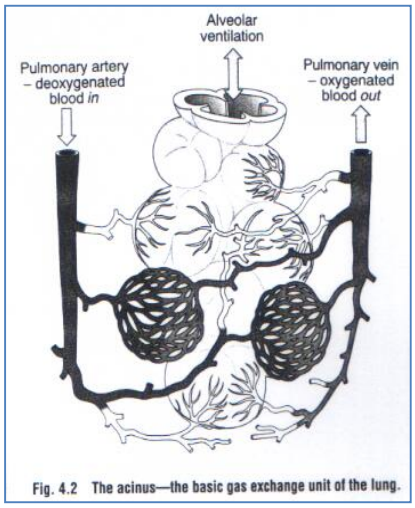


Fig. 4.2 The acinus—the basic gas exchange unit of the lung.

- **Lung Volumes & Blood Flow:**
  - **Anatomical Dead Space =** Total Ventilation – Alveolar Ventilation (= 150mL)
  - **Distribution of Ventilation:**
    - Maximum in Upper Lung
    - Minimum in Lower Lung
  - **Distribution of Perfusion:**
    - Maximum in Lower Lung
    - Minimum in Upper Lung
  - **Ventilation:Perfusion Ratio (VQ-Ratio):**
    - Approaches Infinity in Upper Lung (Zone 1)
      - = "***Physiological Dead Space***"
    - Approaches Zero in Lower Lung (Zone 3)
      - = "***Physiological Shunt***"
    - *NB: VQ Mismath is the Most Common Cause of Hypoxia*.

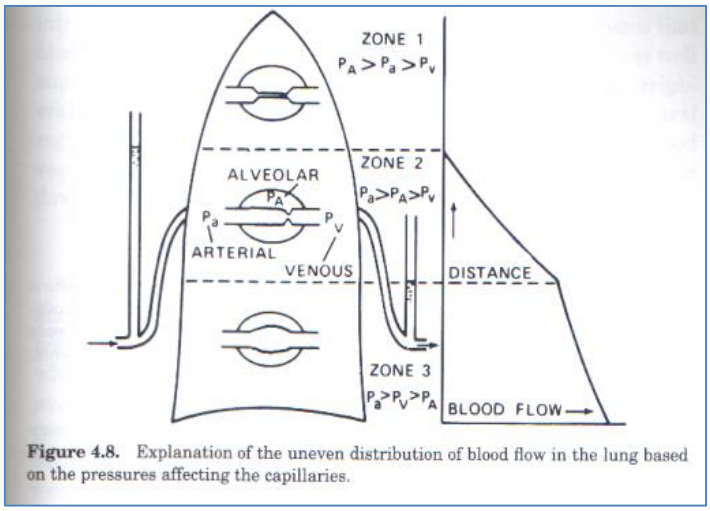


Figure 4.8. Explanation of the uneven distribution of blood flow in the lung based on the pressures affecting the capillaries.

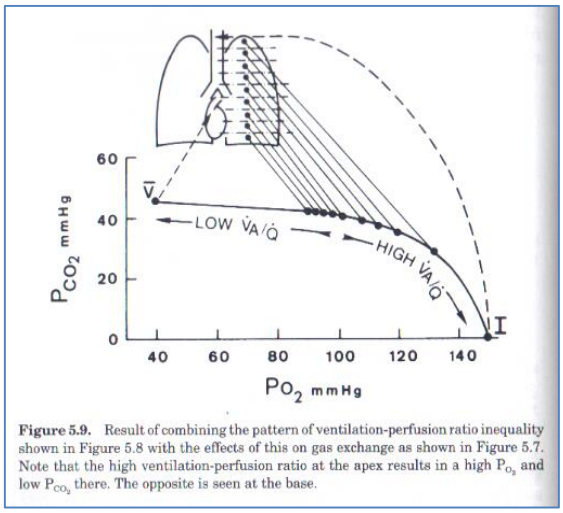


Figure 5.9. Result of combining the pattern of ventilation-perfusion ratio inequality shown in Figure 5.8 with the effects of this on gas exchange as shown in Figure 5.7. Note that the high ventilation-perfusion ratio at the apex results in a high P_{O_2} and low P_{CO_2} there. The opposite is seen at the base.

- **Effects of Disease on Gas Exchange:**
  - Obstruction to Airflow (Eg. Aspirate → Airway Obstruction)
  - Impairment of Diffusion across Alveolar Membrane (Eg. Pneumonia → "Shunt")
  - Loss of Surface Area (Eg. Emphysema → ↑ "Dead Space")

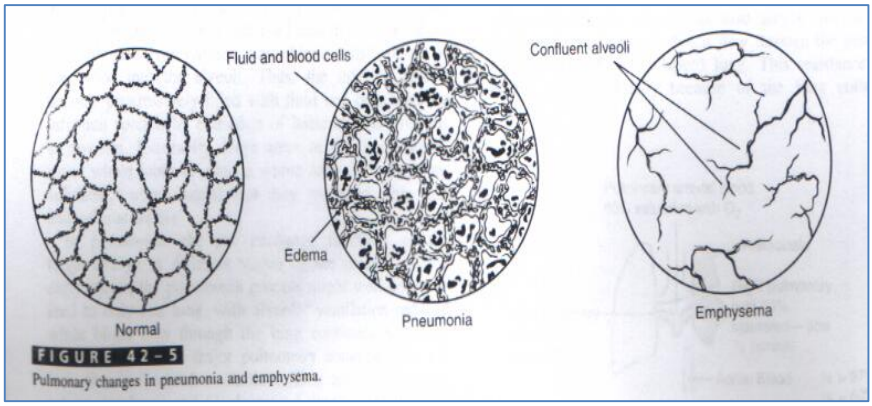


FIGURE 42-5
Pulmonary changes in pneumonia and emphysema.

- **Gas Transfer & Transport – (GLS QUESTIONS):**
  - **By what process is oxygen taken up into the pulmonary capillary from the alveolus?**
    - Diffusion
  - **What is the pressure gradient associated with this?**
    - Partial pressure gradient
  - **How far has the blood moved through the capillary before the PO2 of the capillary has almost equalled that of the alveolus? Does this offer any advantages?**
    - Under Non-Exercising conditions, blood is already O_2-saturated within the first 1/3 of the Alveolar Capillary. Therefore, during exercise (Even with shortened time of exposure in the capillaries) the blood can still become fully Oxygenated.
  - **Only 98 % of the blood that enters the left atrium has come from the alveolar capillaries. Where does the other 2 % come from?**
    - The other 2 per cent of the blood has passed through the ***Bronchial Circulation*** (from the aorta, through the deep tissues of the lungs) & *isn't* exposed to lung air.
    - This is called "shunt flow," – (blood is shunted past the gas exchange areas)
    - The Po2 of shunt blood is similar to Venous Blood (**40mm Hg**)
  - **How does this affect Left-Ventricular PO2?**
    - Po2 of blood entering the Left Heart (& pumped into the aorta) falls to about **95mm Hg**.
  - **What is the PO_2 of Venous Blood?**
    - 40mmHg

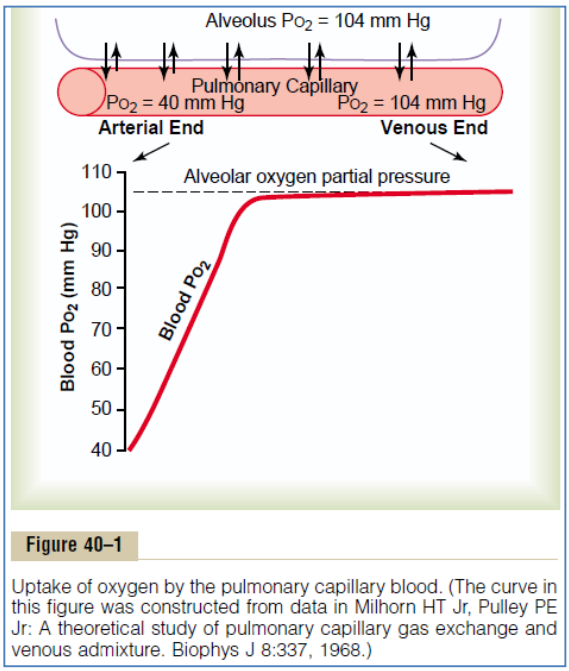


Figure 40–1

Uptake of oxygen by the pulmonary capillary blood. (The curve in this figure was constructed from data in Milhorn HT Jr, Pulley PE Jr: A theoretical study of pulmonary capillary gas exchange and venous admixture. Biophys J 8:337, 1968.)

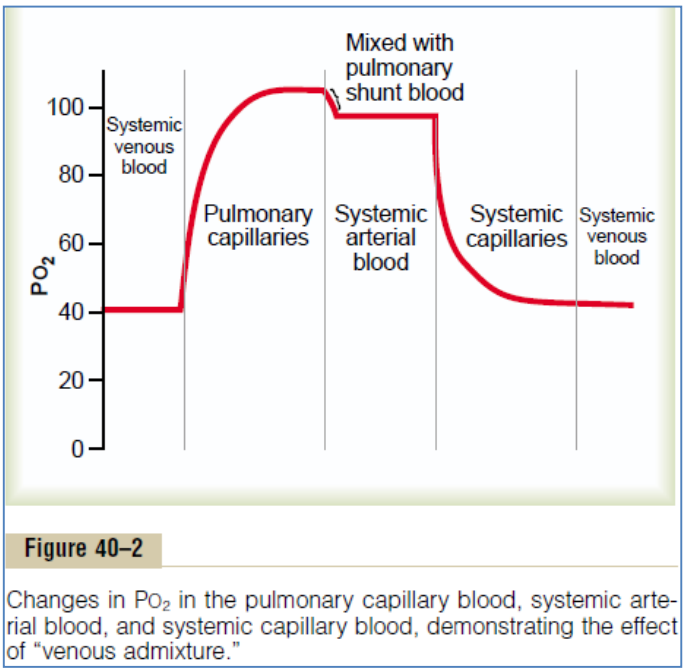
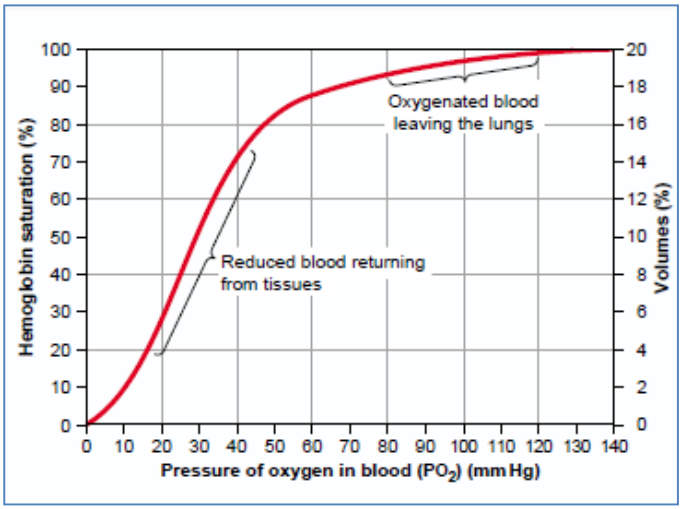


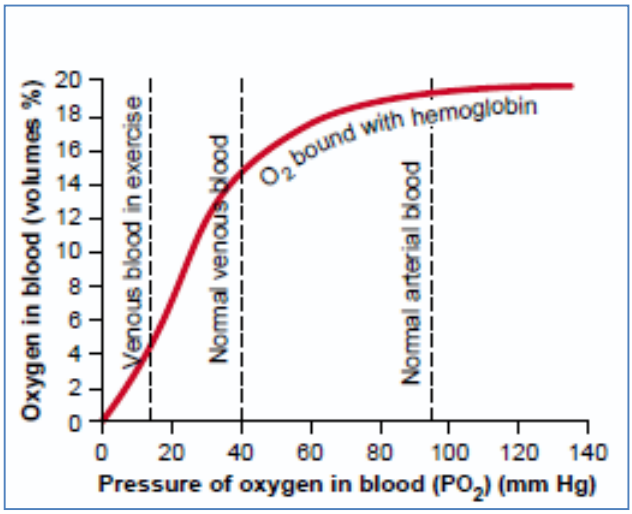
Figure 40–2

Changes in P_{O_2} in the pulmonary capillary blood, systemic arterial blood, and systemic capillary blood, demonstrating the effect of "venous admixture."

- **By what 2 mechanisms is oxygen transported in the blood?**
  - 3% Dissolved in Plasma
  - 97% Bound to Haemoglobin
- **How much oxygen can haemoglobin carry ?**
  - Each Hb molecule can hold 4x O2 molecules

- **<u>Oxygen-Hb-Dissociation Curve:</u>**
  - **Why does the oxygen-haemoglobin dissociation curve have a sigmoid shape?**
    - 1. To favour O2 loading in high PO2 environments (Lungs)
    - 2. To favour O2 unloading in low PO2 Environments (Tissues)
    - - *Acts as an Oxygen "Buffer"*:
      - Notice that the Hb Concentration is maintained despite wide alterations in Alveolar PO_2 (60mmHg – 130mmH).
      - Therefore, there is a wide margin for error when it comes to maintaining a constant supply of O_2 (Ie. High Hb-Saturations) to the Tissues.
      - (Thus, the tissue Po2 hardly changes, despite the marked fall in alveolar Po2.)





  - **What Factors Shift the Curve?**
    - **pH –** (Acid → Right-Shift → Favours O_2 Unloading)
    - **P_{CO2}** – (High P_{CO2} → Right-Shift → Favours O_2 Unloading) ("Bohr effect")
    - **BPG** (Bisphosphoglycerate) – (Hypoxia → ↑BPG → Right-Shift → Favours O_2 Unloading)
    - **Temperature –** (Exercise → ↑2-3°C → Right-Shift → Favours O_2 Unloading)

Classifications of Respiratory Emergencies:

- **Upper Airway – (Typically Obstructive):**
  - Eg. Anaphylaxis (with Laryngeal Oedema)
  - Eg. Foreign Body Aspirate
  - Eg. Epiglottitis (Swelling of the Epiglottis from Haemophylus Influenza B Virus)(Rare due to HIB Vaccine)
  - Eg. Trauma to Neck → Swelling/Deformity
- **Lower Airway – (Gas Exchange Problem/Mechanics of Breathing Problem):**
  - Eg. Pneumonia – (3 Cardinal Signs = Fever, Cough & Tachypnoea)
  - Eg. Pulmonary Oedema – (Eg. From Congestive Heart Failure)
  - Eg. Asthma – (Bronchoconstriction)
  - Eg. Pneumothorax/Haemothorax – (→Disruption of Negative Intrapleural Pressure → Disrupts Mechanics of Breathing)
  - Eg. Flail Chest – (Where broken ribs disconnect from the ribcage & are sucked inward on inspiration; instead of outward → No Ventilation in that lung)
- **Central – (Control of Breathing):**
  - Eg. Any Decreased Level of Consciousness:
    - Trauma/Drug Overdose (Eg. Opioid)/Intracerebral Event
  - Eg. Any cause of Paralysis
    - Eg. Myasthenia Gravis/Guillian Barre Syndrome

Common Outcomes of Respiratory Emergencies:

- **Hypoxia:**
  - **Types:**
    - **Hypoxic Hypoxia:**
      - Most common type.
      - Result of Insufficient oxygen available to the lungs (Eg. Obstruction/Drowning/Altitude)
    - **Stagnant Hypoxia:**
      - Not enough Cardiac Output → ↓Tissue Perfusion
    - **Anaemic Hypoxia:**
      - Not enough Haemoglobin → ↓O_2-Carrying Capacity of Blood.
    - **Histotoxic Hypoxia:**
      - Toxin which prevents Oxidative Metabolism @ the Cellular Level
      - Eg. Cyanide/Oligomycin
  - **Effects:**
    - Reduced work Capacity of Muscles
    - Depressed Mental Capacity
  - **Treatment:**
    - Supplemental O_2
- **Hypercapnia:**
  - **= Excess CO_2:**
    - Typically caused by Hypoventilation
    - (Normal pCO2 Range = 35 - 45mmHg)
  - **Effects:**
    - If pCO2 > 60mmHg → Severe Dyspnoea
    - If pCO2 > 80mmHg → Lethargy & Coma
    - If pCO2 > 120mmHg → Anaesthesia, Respiratory Depression & Death
  - **NB:**
    - CO2 Diffuses 20x faster than O_2
    - CO2 is a *More Potent* Respiratory Stimulus than O_2
    - Blood capacity for CO2 is 3x More than O_2
  - **Treatment:**
    - Encourage Hyperventilation
    - Assisted Breathing (if Unconscious)
    - NB: Supplemental O_2 can → Suppress Central Control of Breathing →Respiratory Arrest.

Common Signs &Symptoms of Respiratory Emergencies:

- **Dyspnoea:**
  - The "Feeling of not being able to breathe enough" (Shortness of Breath)
  - Associated with:
    - Hypercapnia or Hypoxia
    - Respiratory Effort
    - State of Mind
- **Cyanosis:**
  - = Blueness of the skin
  - Caused by Excessive Deoxygenated Hb in the Blood.
    - NB: Anaemic Pts. don't get Cyanotic because there's not enough Hb.
  - May be Central or Peripheral.

How to Assess a Respiratory Emergency:

- **Aims:**
  - Determine Cause of Abnormality
  - Determine Severity
- **Methods:**
  - **Primary Survey** – ABC
  - **In Depth:**
    - **Appearance (The "End-of-the-Bed –ogram"):**
      - Consciousness
      - Sweating
      - Agitation
      - Cyanosis/Pallor
      - Urticaria (Skin rash)/Angioedema
    - **History:**
      - Nature/Onset/Progression of Symptoms
      - Associated Symptoms
      - Treatment So Far
      - Previous Episodes
      - Other significant past history
    - **Examination (*Accurate* Resp. Rate; + Pulse):**
      - Airway Patency – (Swelling/Injury?; Stridor?)
      - Resp. Rate/Depth
      - Work of Breathing
      - Auscultation – (Air Entry?; Wheeze?)
      - Percussion
      - Tracheal Deviation
      - Check JVP
      - Pulse & BP.
    - **Monitoring:**
      - O_2 Sats
      - Vitals
    - **Investigations:**
      - Arterial Blood Gases
      - Ch.XR
      - ECG
      - Spirometry
      - Sputum Culture

PULSE OXIMETRY - *KNOW*:

- **Relationship Between O2 Sats & pO2:**
  - Higher the O2 Sats, the Higher the pO2
- **Shape of the Oxygen-Hb-Dissociation Curve:**
  - Plateau favours O2 loading @ High pO2
  - Steep part favours O2 Unloading @ Low pO2
- **Shifting the Oxygen-Hb-Dissociation Curve:**
  - **pH –** (Acid → Right-Shift → Favours O_2 Unloading)
  - **P_{CO2}** – (High P_{CO2} → Right-Shift → Favours O_2 Unloading) ("Bohr effect")
  - **BPG** (Bisphosphoglycerate) – (Hypoxia → ↑BPG → Right-Shift → Favours O_2 Unloading)
  - **Temperature –** (Exercise → ↑2-3°C → Right-Shift → Favours O_2 Unloading)
- **Why is Oxygen Important?**
  - Essential for aerobic cell function
  - Some cells (with no aerobic capacity – Neurons/Myocytes) are damaged very quickly if hypoxic.
- **Symptoms @ Different Arterial PO_2's:**
  - PaO2 of 90 mmHg - normal person with no symptoms
  - PaO2 of 55 mmHg - short term memory loss, euphoria, impaired judgement
  - PaO2 of 30 -55 mmHg - progressive loss of cognitive and motor function
  - PaO2 < 30 mmHg - loss of consciousness

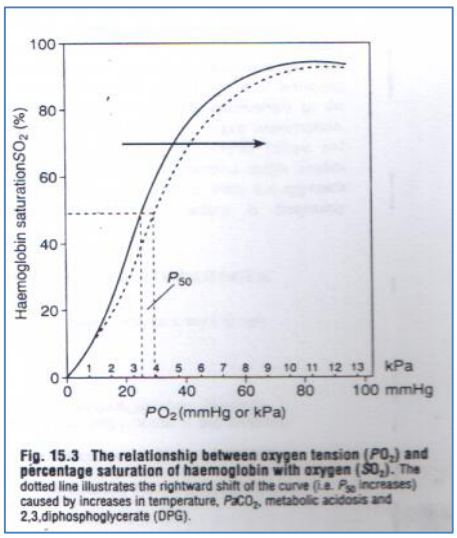


Fig. 15.3 The relationship between oxygen tension (PO_2) and percentage saturation of haemoglobin with oxygen (SO_2). The dotted line illustrates the rightward shift of the curve (i.e. P_{50} increases) caused by increases in temperature, $PaCO_2$, metabolic acidosis and 2,3,diphosphoglycerate (DPG).

O2-Haemoglobin dissociation curve

Saturation %	PaO2 mmHg	Significance
97%	100	normal
90%	60	"cusp"
75%	40	venous
50%	26	

Be able to reproduce both of the above.

Arterial Blood Gas:

- **Provides Information on:**
  - Oxygenation & Ventilation (pO2 and pCO2)
    - *i or Insp* = Inspired gas
    - *a or Art* = Arterial blood
    - *A or Alv* = Alveolar gas
    - *No Prefix* = Arterial Blood
  - Acid/Base Disturbance
  - Hb
- **Significant Measurements:**
  - pH
  - pCO_2
  - pO_2
  - HCO_3
  - Base Excess
  - A-a Gradient
- **Normal Values:**
  - pH : 7.35 – 7.45
  - pO_2 : 70-100 mmHg
  - pCO2 : 35 – 45 mmHg
  - HCO3 : 22 – 26 mmol/L (arterial) and 24 – 28 mmol/L (venous)
  - BE : -3 to +3
    - The Base Excess = The amount of base needed to be Added/Removed to restore the pH to 7.4 with the pCO2 held constant at 40mmHg. It is a representation of the metabolic component of any acid base disturbance.
- **What Specific Abnormal Values Tell Us:**
  - **Acidosis:**
    - pH less than 7.35
    - **Can be Respiratory or Metabolic:**
      - **Respiratory** – Due to Alveolar Hypoventilation (→↑$paCO_2$)
        - Compensated for by Metabolic Mechanisms (Ie. Retaining Base)
      - **Metabolic** – Due to Gain of Acid *OR* Loss of Base
        - Compensated Rapidly by Respiratory Mechanisms (Ie. Blowing off CO_2)
  - **Alkalosis:**
    - pH more than 7.45
    - **Can be Respiratory or Metabolic:**
      - **Respiratory** – Due to Alveolar Hyperventilation (→↓$paCO_2$)
        - Compensated by Metabolic Mechanisms (Ie. Excreting Base)
      - **Metabolic** – Due to Loss of Acid (Eg. Acute Vomiting)
        - Compensated Rapidly by Respiratory Mechanisms (Ie. Retaining CO_2 Hypoventilation)
  - **Compensation?:**
    - Remember the Bicarbonate Buffer system:
      - H+ + HCO3- ⇔ H2CO3 ⇔ CO2 + H2O
    - **Acidosis** (more H+) can be buffered by forcing the equation to the right:
      - Adding HCO3-
      - or Lowering CO2
    - **Alkalosis** (less H+) can be buffered by forcing the equation to the left:
      - Adding H+
      - or Hypoventilating (to raise CO2)

- **Anion Gap:**
  - (The difference between Measured Cations and Unmeasured Anions (including acids))
  - Anion gap is used to narrow down the causes of metabolic acidosis:
    - High Anion Gap Metabolic Acidosis is due to ↑Concentration of Unmeasured Anions:
      - Lactic Acidosis
      - Ketoacidosis
      - Renal Failure (Uraemia)
    - Normal Anion Gap Metabolic Acidosis is due to Loss of HCO_3 from the body.
      - Renal Losses (Eg. Renal Tubular Acidosis)
      - GIT Losses (Eg. Diarrhoea)
- **A-a Gradient:**
  - Gap between the Calculated Alveolar pO2 and the Measured Arterial pO2.(taken from the arterial blood gas)
  - (A-a) Gradient = pAlvO2 – pArtO2
  - **Normally less than 12**
  - Abnormal (A-a) gradient = V/Q Mismatch (Ie. Lungs aren't exchanging air)

- **6 Steps to Interpretation of Arterial Blood Gas (ABG) Results:**
  1) **What is the pH**
     - Is this an acidosis or an alkalosis ?
  2) **Is Co2 Responsible**
     - Is the change in pCO2 consistent with the dominant acid base disturbance ?
       - If it is then this is a respiratory acidosis or alkalosis.
       - If it is not then it is a metabolic acidosis or alkalosis.
  3) **Is HCO3 responsible?**
     - Is the change in H2CO3 consistent with the dominant acid base disturbance ?
  4) **State the primary Disturbance**
     - Is this an acidosis or an alkalosis ?
  5) **Look for compensation**
     - **Look at the Base Excess.**
       - (Base Excess = the amount of base that you'd need to add to make the pH normal if he CO2 was normal in that patient)
       - (*Abnormal Base Excess = Metabolic Component*)
       - If it is > +3 then it is a metabolic alkalosis.
         - (Excess Base or Deficit of Acid)
       - If it is < -3 then it is a metabolic acidosis.
         - (Deficit of Base or Excess Acid)
     - **Look at the pCO2 & H2CO3**
       - Is there any respiratory compensation? (signs of Hyper/Hypo-Ventilation?)
       - Is there any metabolic compensation? (signs of H2CO3 Excretion/Retention?)
  6) **Final analysis**
     - State your findings

- **Arterial Blood Gas Example Cases:**
  - **CASE 1** – A student practising arterial blood gas sampling on another student.
    - PH 7.40
    - PCO2 40 mmHg
    - PO2 95 mmHg
    - HCO3- 27 mmol/l
    - Base Excess -1
  - Normal

  - **CASE 2** – A 38 year old male who has been found unconscious after taking an overdose.
    - PH 6.95
    - PCO2 85 mmHg
    - PO2 40 mmHg
    - HCO3- 33 mmol/l
    - Base Excess +2
  - 1. It is an Acidosis
  - 2. PCO_2 is elevated (Consistent with Acidosis) → Respiratory Acidosis
  - 3. Base Excess is Normal → No Metabolic Component

  - **CASE 3** – A 17 year old who has become very upset after a fight with friends.
    - PH 7.7
    - PCO2 10 mmHg
    - PO2 110 mmHg
    - HCO3- 24 mmHg
    - Base Excess -2
  - 1. It is an Alkalosis
  - 2. PCO_2 is Low (Consistent with Alkalosis) → Respiratory Alkalosis
  - 3. Base Excess is Normal → No Metabolic Component

  - **CASE 4** – A 27 year old female diabetic with vomiting and feeling unwell.
    - PH 7.2
    - PCO2 25 mmHg
    - PO2 98 mmHg
    - HCO3- 14 mmol/l
    - Base Excess -12
  - 1. It is an Acidosis
  - 2. PCO_2 is Low (*Not* Consistent with Acidosis) → Metabolic Acidosis
  - 3. Base Excess is Abnormal → Metabolic Component
    - -12 → Metabolic Acidosis

  - **CASE 5** – A 54 year old male with diarrhoea.
    - PH 7.6
    - PCO2 46 mmHg
    - PO2 74 mmHg
    - HCO3- 39 mmol/l
    - Base Excess + 10
  - 1. It is an Alkalosis
  - 2. PCO_2 is Normal (Not Consistent with Alkalosis) → Metabolic Alkalosis
  - 3. Base Excess is Abnormal → Metabolic Component
    - + 10 → Metabolic Alkalosis

- **CASE 6** 70 year old man, short of breath
  - pH 7.25
  - CO2 90
  - O2 60
  - HCO3 38
  - Base Excess +10
- 1. It is an Acidosis
- 2. PCO_2 is High (Consistent with Acidosis) → Respiratory Acidosis
- 3. Base Excess is Abnormal → Metabolic Component
  - +10 → A Metabolic Alkalosis (Compensating for the Respiratory Acidosis; Confirmed by the high HCO_3)
  - :. Respiratory Acidosis with Metabolic Compensation

- **More Arterial Blood Gasses Cases:**
  - Ph 7.3
  - CO2 – 70 (Resp Acidosis)
  - HCO3 – 30
  - Base excess = +8 (Metabolic Alkalosis)
  - Therefore – Respiratory acidosis with Metabolic compensation

  - Ph 7.2
  - O2 – 105
  - CO2 – 16 (Respiratory Compensation)
  - HCO3 – 10
  - Base excess = -16 (Metabolic Acidosis)
  - Metabolic Acidosis with Respiratory compensation

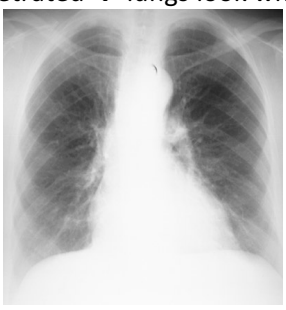
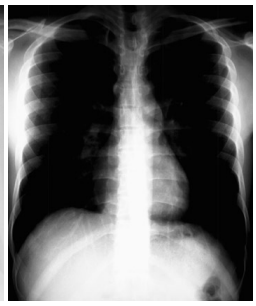
  - Ph 7.2
  - O2 - 60
  - CO2 - 80 (Respiratory Acidosis)
  - HCO3 - 28
  - Base Excess = -1 (No metabolic compensation)
  - Acute Respiratory Acidosis with no Metabolic Compensation

  - pH 7.6
  - O2 - 97
  - CO2 - 15
  - HCO3 - 20
  - Base excess = +2
  - Respiratory Alkalosis due to Hyperventilation

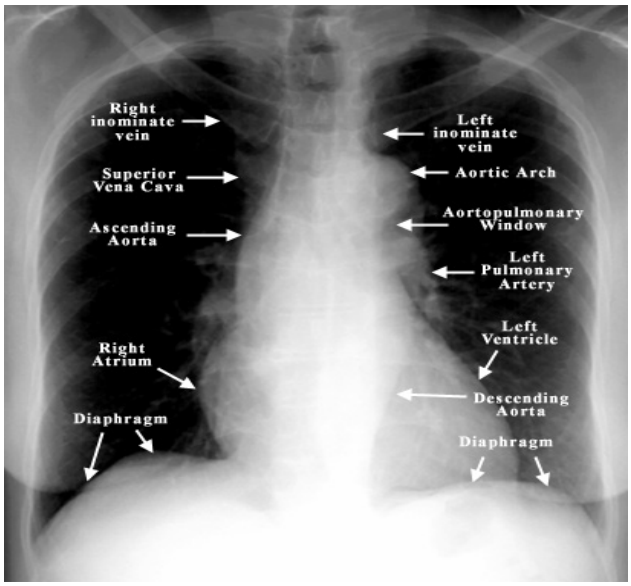
  - 7.13 Acidosis
  - CO2 - 43
  - HCO3 - 18
  - Base Excess = -31 (Metabolic Acidosis)
  - Metabolic Acidosis (However he isn't compensating by breathing(Which he should be)) → About to have a respiratory arrest (Very sick)

System for Examining Chest X-Rays:

1. **Check the patient ID/Date/Orientation.**
2. **Check the projection of film (AP, PA, portable, supine, lateral)**
    a. (Usual films are a PA and a Lateral)
    b. **AP = ANTERO POSTERIOR:** Taken with beam going from front to back.
        i. The heart is further from the plate → Magnifies the size of the heart. (Can't comment on cardiomegaly)
    c. **PA = POSTERO ANTERIOR:** Taken with beam going from back to front.
        i. Pt usually stands with chest against the xray cassette.
        ii. :. The heart is very close to the plate (negligible magnification of heart) (Can comment on cardiomegaly)
    d. **S = SUPINE:** Taken with the patient lying flat.
    e. **ERECT:** Taken with the patient standing/sitting.
    f. **LAT = LATERAL:** Taken from the patient's side.
    g. **MOBILE:** Portable xray was used while the patient was in their bed. (Therefore supine & AP)
3. **Technically Adequate:**
    a. Adequate Inspiration
        i. Need Full inspiratory film (Sometimes this isn't possible)
        ii. Should see at least 8 posterior ribs
    b. Adequate penetration (you should be able to see the bones of the vertebral column through the heart)
        i. Overpenetrated → Lungs look black
        ii. Underpenetrated → lungs look white

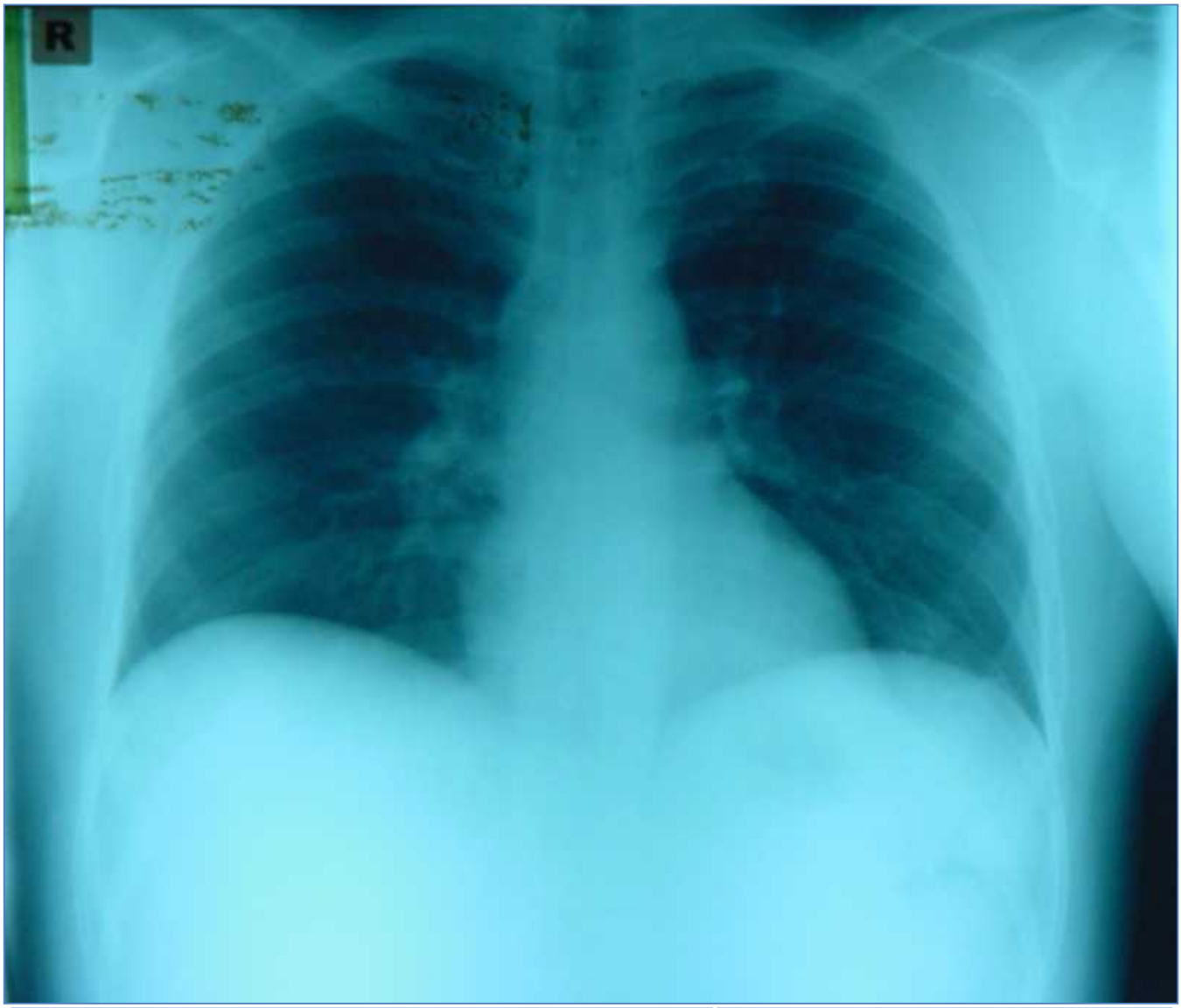



    c. Rotation (Clavicles should have the spine in the middle)
        i. Ie. Distance between spines of the vertebra and the sternal ends of each clavicle are the same.
4. **Look at all structures in some order**
    a. Heart
    b. Mediastinum (Diaphragm, Right Atrium, Ascending Aorta, SVC, Pulmonary Vessels, Left Ventricle, Descending Aorta)
    c. Airways (Trachea (midline/deviated))
    d. Carina
    e. lungs
    f. diaphragm
    g. bones and soft tissues



5. **Look at specific areas that may be more difficult to see clearly**
    a. Apices
    b. Hilar
    c. Behind heart
    d. Costophrenic angles

- **What is Normal & Abnormal?**
  - **The Trachea (& isn't Deviated)**
    - May be Deviated in Tension Pneumothorax/Haemothorax/Collapse and Consolidation on opposite side.
  - **The Carina (The bifurcation of the 2 Primary Bronchi)**
    - Carina Angle may be Widened/Distorted by Cancer of Surrounding Lymph Nodes (Serious)
  - **Left & Right Bronchus (Right is Shorter & more Verticle)**
    - Significant as solid aspirates tend to lodge in Right Bronchus.
  - **Sharp, clear Costo-Phrenic Angles (Where the diaphragm meets the ribs)**
    - **Pleural Effusion:**
      - Collection of fluid in the pleural space
      - → Blunts or totally Obscures Costophrenic Angles.
      - A Meniscus is sometimes visible
  - **Well Defined Diaphragmatic Border (Right dome higher due to liver)**
  - **Normal Lung Markings**
    - Extra lung markings (White) = Increased Density of the lung parenchyma – Eg. Fibrosis, Oedema, Exudate, Malignancy, Pneumonia
  - **Hilar Shadows (Radiographic Hilum of the Lung – Incl. Pulmonary Vessels, Bronchial Walls, Lymph Nodes)**
    - Enlarged Hilar Shadows - Dilated Pulmonary Vessels or Enlarged Hilar Lymph Nodes.
  - **Well Defined Heart Borders (Offset to the Left)**
    - Can be obscured by pneumonia
  - **Cardiothoracic ratio = Heart Width/Chest Width**
    - If greater than 50% = Cardiomegaly
    - Cardiomegaly suggests:
      - Heart Failure
      - Pericardial Effusion
      - L/R Ventricular Hypertrophy
  - **Pneumonia**
    - Infection in the lung tisues
    - Air spaces filled with bacteria & pus
    - X-Ray Signs are Opacification (Consolidation)
  - **Pulmnoary Oedema:**
    - Kerly-B Lines (Thin white lines extending to the periphery)
    - Due to increased interstitial fluid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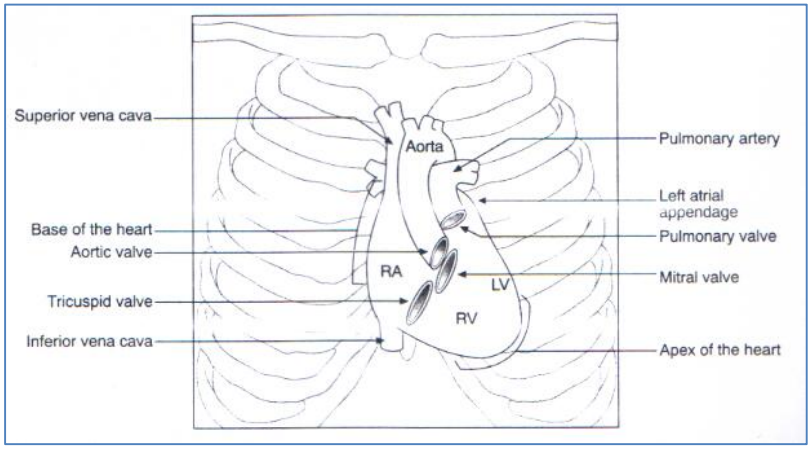
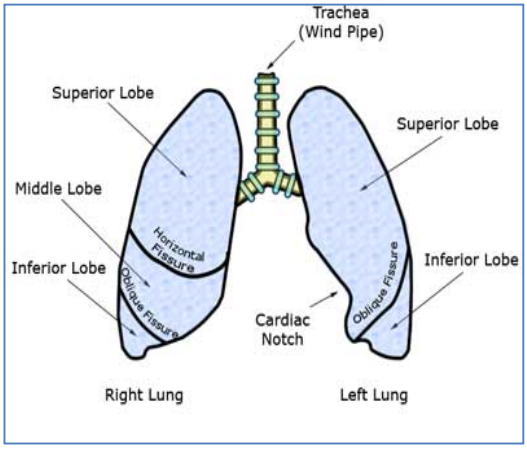

Superior vena cava
Aorta
Pulmonary artery
Left atrial appendage
Base of the heart
Aortic valve
Pulmonary valve
RA
LV
Mitral valve
Tricuspid valve
RV
Inferior vena cava
Apex of the heart


Trachea
(Wind Pipe)
Superior Lobe
Superior Lobe
Middle Lobe
Horizontal Fissure
Inferior Lobe
Oblique Fissure
Inferior Lobe
Cardiac Notch
Oblique Fissure
Right Lung
Left Lung

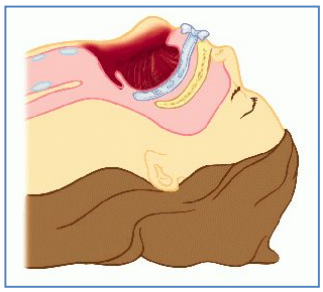
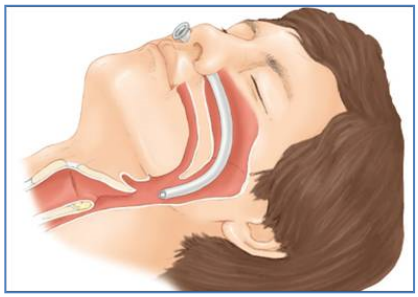
TREATING RESPIRATORY EMERGENCIES

Definitive Treatments: (The simple Equations):

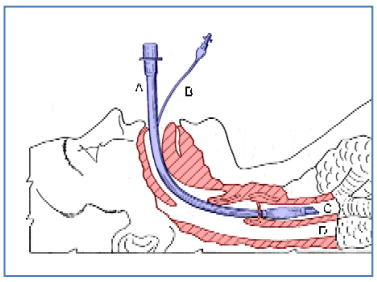
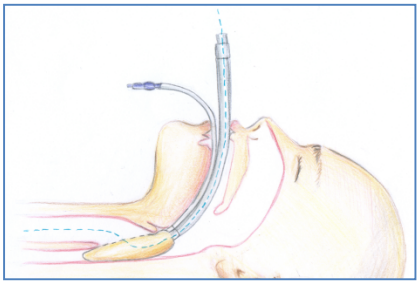
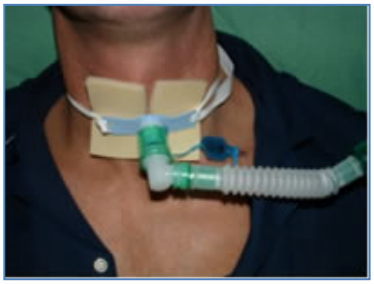
- **Anaphylaxis =** Adrenaline
- **Asthma =** Bronchodilators
- **Pneumonia =** Antibiotics
- **Tension Pneumothorax =** ICC (Intercostal Catheter)

Supportive Treatments:

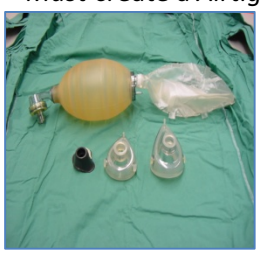
- **Secure an Airway:**
  - **Positioning:**
    - Jaw thrust
    - Chin Lift
    - Head Tilt
    - Lying on Side
  - **Basic Interventions:**
    - Oropharyngeal Airway (OPA) – "Guedel Airway"
    - Nasopharyngeal Airway (NPA)

  - **Advanced Interventions:**
    - Endotracheal tube (ETT)
    - Laryngeal Mask (LMA)
    - Surgical Airway (Trachostomy)

- **Breathing:**
  - **Increase FiO_2:**
    - (FiO_2 = *Fraction* of Inspired Oxygen in *Gas Mixture*)
    - Expressed as a %age.
    - No more than 24hrs on 60% → Toxicity - (Higher settings can lead to oxygen toxicity)
      - (Air is ≈20%)
  - **Assist Ventilation:**
    - Why? – If Pt is unconscious and has No Respiratory Drive.
    - What about Airway? – *Must be Clear*.
    - Different Sized Masks – *Must* create a *Airtight Seal*.



- **Supplemental Oxygen Delivery:**
  - **Principles for Supplemental O_2 Delivery:**
    - Precise control of FiO2 (Fraction of Inspired O_2; No more than 24hrs on 60% → Toxicity)
    - FiO2 is independent of Patient's Ventilatory Pattern
    - Avoid Rebreathing of Expired Gas (Ie. Avoid CO2 Retention)
    - Don't Increase of Work of Breathing
    - Provide Humidified Gas at 37°C
    - Comfort
  - **Various Delivery Systems:**
    - **Nasal Prongs/Cannulae:**
      - **Flow Rate**: 2-6 l/min (FiO2: 0.25 - 0.40)
      - **Problems:**
        - Variable and poorly predictable FiO2
        - High flow rates difficult to tolerate
        - Dries nasal mucosa
        - Uncomfortable at higher flow rates (> 4 l/min)
      - **Advantages:**
        - Comfortable at low flow rates
        - Allow patient to eat and drink
        - Cheap
        - No rebreathing occurs
    - **Hudson Mask:**
      - **Flow Rate:** 4-15 l/min (FiO2: 0.35 - 0.7)
      - **Problems:**
        - Maximum FiO2 less than 0.7 with 15 l/min
        - Variable and poorly predictable FiO2
        - Poor humidification with high FiO2
        - Rebreathing occurs with FiO2 < 0.35 (O2 flow < 4l/min)
      - **Advantages:**
        - Comfortable (usually)
        - Cheap
        - Allow a large variation in target FiO2
    - **Venturi Mask:**
      - **Flow Rate:** 3-12 l/min (FiO2: 0.24 - 0.60)
      - **Venturi Tunnel:**
        - Sucks in room air using an oxygen jet.
        - FiO2 varied by adjusting O2 flow rate and venturi aperture size.
      - **Advantages:**
        - More predictable control of FiO2
        - No rebreathing
        - Reasonable humidification at low FiO2
      - **Disadvantages:**
        - Unable to deliver FiO2 > 0.6
        - FiO2 fluctuates in severe dyspnoea with high inspiratory flow rates
        - Comfortable (usually)
        - Cheap
    - **Reservoir Mask (Non Rebreather Mask):**
      - **Flow Rate:** Max 15 l/min (FiO2: < 0.85)
      - **Reservoir:**
        - Decreases variability of FiO2 with changes in patient ventilation
      - **Advantages:**
        - Maximal FiO2 with a simple mask
      - **Disadvantages:**
        - Care with flow rates, faulty valves and consequent rebreathing
        - Minor expense increase

- **Bag Valve Mask:**
  - **Flow Rate:** 15 l/min (FiO2 nearly 100%)
  - **Bag Reservoir:**
    - 100% O2 is possible when the plastic bag reservoir is used.
  - **Uses:**
    - Positive Pressure Ventilation
    - Maximise FiO2 in Spontaneously Breathing Patient.

- **<u>Precautions With Use Of Oxygen:</u>**
  - **Oxygen Toxicity:**
    - Only if
      - - FiO2 > 0.6
      - - Duration > 24 hours
  - **Chronic Airway Limitation ("Hypoxic Drive"):**
    - ↑CO2 = The #1 Stimulus to Breathe
    - *However,* Patients with CAL have a ↓Sensitivity to CO2 due to chronic hypercapnia; and hypoxia assumes backup respiratory drive.
    - Supplemental Oxygen can remove the hypoxic stimulus to breath → Respiratory Depression.

SYNTHESIS SESSION CASES:

Respiratory Emergencies:

- **Case 1:**
  - Temperature
  - Otherwise well
  - 100% sats
  - Cough & runny nose
  - **Diagnosis =** Viral Infection
  - **Treament =** Reassurance, rest & fluids

- **Case 2:**
  - Febrile
  - Creps
  - 90% Sats (Hypoxic)
  - Tachypnoeic
  - Mild indrawing & creps
  - **Diagnosis =** Severe Pnemonia (Due to hypoxia)
  - **Treatment =** IV Antibiotics, IV Fluids, Oxygen

- **Case 3:**
  - Knife in man's chest on right hand side of sternum.
  - Breathless
  - Hypotensive
  - JVP not raised
  - **Diagnosis =** Massive Haemothorax (Because his JVP isn't distended → he prob has Hypovolaemia :. The knife has cut a blood vessel and is bleeding into his chest.)
    - Tension Pneumothorax is a possibility also
  - **How would you confirm this?**
    - Clinical assessment
  - **Treatment =**
    - If haemothorax → put a needle (drain) in his chest

- **Case 4:**
  - Attempted hanging
  - Unconscious
  - Stridor
  - **What is the 1st Priority?**
    - Airway Management – Probably use of Endotracheal Intubation (although may be very difficult)
      - Guedel airway is too short
      - If impossible to intubate → Emergency Tracheostomy

- **Case 5:**
  - Car accident
  - T-Boned at high speed
  - Pale
  - Sweaty
  - Hypotensive (40mmHg)
  - Whited out L Lung on Xray
  - **Diagnosis =**
    - Haemothorax
    - Probably also has a pneumothorax but not visible on xray
  - **Treatment =**
    - Emergency chest drain with Intercostal Catheter (ICC)

- **Case 6:**
    - Barking cough
    - Inspiratory stridor
    - Moderate indrawing
    - Very dyspnoeic
    - Agitated & crying
    - **Diagnosis =**
        - Croup (Barking cough & stridor)
    - **Assessment should include:**
        - Clinical Assessment should suffice (in ED situation)
        - Measure Sats.
    - **Treatment =**
        - No antibiotics are needed (croup is viral)
        - Nebulised adrenaline & steroids to decrease swelling.

- **Case 7:**
    - Ph 7.3 - Acidosis
    - Co2 66 – Raised → Respiratory
    - O3 150
    - HCO3 28
    - Base excess = -1 → No metabolic compensation
    - **Diagnosis =**
        - Respiratory acidosis with no metabolic compensation

Emergency Medicine Notes
Toxicology
& Endocrine Emergencies

Overview:

- **ABCs for Toxicology**
- **Red Flags:**
  - NB: 2 Tablets can kill a 10kg child
  - Be aware of the meds that are toxic
  - "Nice" or "Nasty"
- **Know where to get help:**
  - Poisons Information Centre

Causes of Toxic Syndromes:

- **Accidental:**
  - Household Drugs/Toxins (Typically little children)
  - Occupational Exposure – (Acute/Chronic)
- **Intentional Self-Poisoning:**
  - Recreational
  - Parasuicidal Gesture (Call for attention)
  - Suicide Attempt
- **Inappropriate use of Medication**
- **Envenomation**

Toxidromes:

- **Paracetamol Overdose:**
  - No *"Toxidrome"* & Mostly Asymptomatic
  - → Secondary Metabolite is Highly Hepatotoxic
  - **Antidote:** N-Acetyl Cystiene (*A Precursor to Glutathione – A Conjugator for Paracetamol)
    - Give if the Blood-Paracetamol Level is Above the *'Toxicity Line'* @ 4hrs.
    - (Small risk of Anaphylaxis)

- **Cholinergic (Eg. Organophosphates – ACh-Esterase Inhibitors) –** "SLUDGE"
  - Salivation, Lacrimation, Urination, Defecation, GI Upset, Emesis
  - **Antidotes:** Atropine (An Anti-Muscarinic) & Pralidoxime (An Ach-I-Inhibitor)
    - NB: Atropine can → VF/SVT/VT

- **Anticholinergic Syndrome (Eg. TCA's, Antihistamines, Atropine) –** "Anti-SLUDGE"**:**
  - Dry Mouth, Dry Eyes, Urinary Retention, Constipation, ….
  - **TCA Overdose →** Widening QRS + Right-Axis Deviation.
  - **Antidote:** Sodium Bicarbonate:
    - Works by preventing the combination of acid with the ionic form of TCA to form the TCA molecule (which is absorbed by cells).
    - Also prevents the positive feedback loop of acidosis (which increases the level of TCA Molecules)

- **Extrapyramidal (Eg. Antipsychotics) -** "TROD"
  - Tremor, Rigidity, Opisthotonos (Abnormal Posture), Dysphagia (Difficulty Swallowing)

- **Opioid:**
  - **Triad:** Coma, Respiratory Depression, Miosis (Pinpoint Pupils)
  - **Antidote:** Naloxone (An Opioid Receptor Antagonist)
    - Given ASAP after overdose
    - NB: Naloxone is often shorter-acting than the Opioids taken :. Require constant infusion.
      - Wears off after 45mins

- **Sedative/Hypnotic (Eg. Benzodiazepines):**
  - **Triad:** Coma, Respiratory Depression, Impaired Airway
  - **Antidote:** Flumazenil (Rarely Used)
    - NB: If Pt. is a Benzo Addict, Flumazenil can → Acute Withdrawal. :. Titrate Dose.

- **Sympathomimetic (Eg. Amphetamines, Cocaine):**
  - Paranoid Schizophrenia, Very Excited, Tachycardia, Hypertension

- **Serotonergic (Eg. SSRI Antidepressants):**
  - Agitation, Confusion, Diarrhea, Fever, Shivering, Tremor

- **Withdrawal Syndromes (Eg. Alcohol/Sedatives (Benzos)/Narcotics):**
  - Restlessness, Irritability, Chills, Hallucinations, Confusion, Sympathetic Overactivity (Sweating, Tachycardia, Hypertension), Seizures

Approach to the Poisoned Patient:

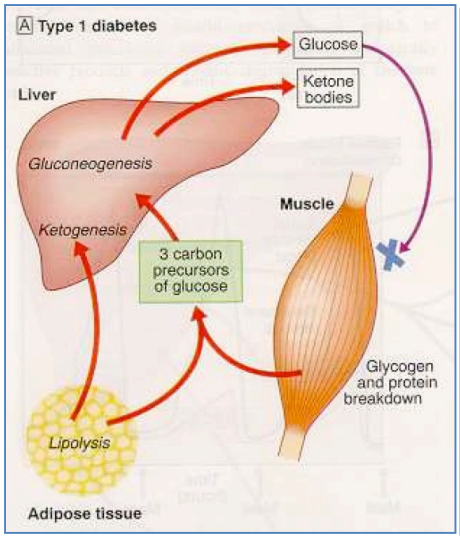
1) **Primary Survey:**
   a. **ABCD:**
      i. Airway (eg. Vomiting/Aspiration/Allergic Reaction → Swelling/Altered Consciousness → Loss of Ability to maintain Airway)
      ii. Breathing (Eg. Too Much/Too Little)
      iii. Circulation (Eg. Tachy/Brady/Hypertension/Hypotension) – (Monitor with ECG & BP)
      iv. Disability (Conscious State)/Danger (What did they take/When/How Much)
   b. **Drug Manipulation (Decontamination &/Or Decrease Absorption/Specific Antidotes):**
      i. **Ipecac** (Induce Vomiting) - *Minimal Use in ED*
      ii. **Gastric Lavarge** (Useful within the 1st hour) *– Now de-emphasized in ED*
      iii. **Activated Charcoal**
         1. (Binds free drug in lumen of gut → ↓drug concentration → ↓Absorption)
         2. Only effective for things that will bind. (won't bind small ionic compounds, heavy metals, or alcohols)
         3. Very Time Dependent
      iv. **Whole Bowel Irrigation (Eg. Polyethylene Glycol** – Golytley: An osmotic agent → Flushes out bowel)
      v. Skin Decontamination (eg. Organophosphate Poisoning)
      vi. Left Lateral Position → Delays Gastric Emptying.
      vii. Cathartics (flush things through)
      viii. Urine pH Alteration
      ix. Dialysis
      x. Chelation
   c. **Differential Diagnosis:**
      i. **Impaired Conscious State (SMASHED):**
         1. S Substrate/Sepsis
         2. M Meningitis/Mental Illness
         3. A Alcohol/Accident (CVA/SAH/Subdural/CHI)
         4. S Seizures/Stimulants
         5. H Hypo/Hyper- (Thyroidism/Thermia/Glycaemia/Tension/Carbia)
         6. E Electrolytes/Encephalopathy/Envenomation
         7. D Drugs
      ii. **Don't Forget Blood Sugar**
      iii. **ECG:**
         1. Screening for Lethal Toxicologies.

2) **Emergency Antidotes:**
    a. Paracetamol N-Acetyl Cysteine
    b. Opiates Naloxone
    c. Benzos Flumazenil
    d. Ca Antagonists Insulin & Glucose
3) **Secondary Survey:**
    a. **Examination & History:**
        i. Directed History:
            1. Who
            2. What
            3. Where
            4. When
            5. Why
            6. How
        ii. Systematic Examination:
            1. Identify Problems
            2. Recognition of Toxic Syndromes
        iii. Focused Investigations:
            1. BSL
            2. ECG
            3. Electrolytes
            4. LFTs (liver function tests)
            5. EtOH
            6. ABG
            7. FBE
            8. Paracetamol Level
            9. Toxic Screen
    b. **Education:**
        i. To Prevent Accidental Poisonings (eg. In kids)
        ii. Put drugs in childproof containers
    c. **Funny Behaviour:**
        i. Underlying Psychodynamics:
            1. Intent? – What did you think would happen?
            2. Suicidal? – Do a "SAD PERSONS" score.
            3. Predisposing Psychiatric Illness (Eg. Psychosis, Depression)
4) **Definitive Care:**
    a. Serious or Potentially Serious → ICU
    b. Mild → Observe in ED
    c. Self harm → Psychiatric Evaluation

Endocrine Emergencies:
Focus on Diabetic Emergencies

Diabetic Emergencies:

- **Diabetic Ketoacidosis:**
  - **Acute life threatening**
  - **Pathology – Combination of:**
    - **Insulin Deficiency**
      - Cell unable to absorb & metaoblis glucose
    - **Excess Counter-Regulatory Hormones (Eg. Glucagon/Adrenaline)**
      - Glycogen Breakdown
      - Lipolysis → Ketogenesis
      - Protein Catabolism
    - **Resultant Hyperglycaemia**
      - Osmotic dieresis → Dehydration
  - **Presentation:**
    - **– of Underlying Diabetes:**
      - Polyuria
      - Polydipsia
      - Weight loss
    - **– of Hyperglycaemia:**
      - Glycosuria/Osmotic dieresis
      - Salt & Water Depletion
    - **– of Hyperketonaemia → Metabolic Acidosis:**
      - Acetone Breath
      - Hyperventilation (Respiratory Compensation)
      - Peripheral Vasodilation → Hypotension
      - K^+ Depletion



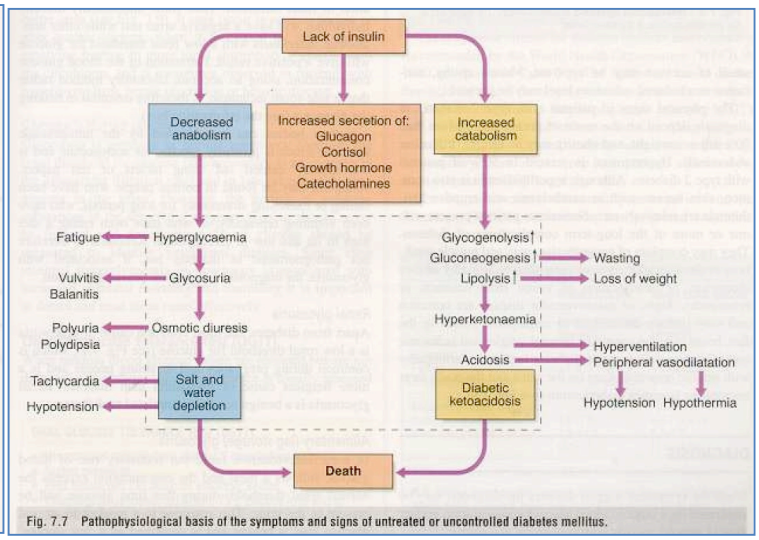


Fig. 7.7 Pathophysiological basis of the symptoms and signs of untreated or uncontrolled diabetes mellitus.

  - **Treatment:**
    - Supportive (ABCs)
    - Rehydration
      - Replace ½ fluid deficit in 1st 12 hrs
    - Insulin infusion
    - Close monitoring of Electrolytes

- **Hyperosmolar Non-Ketotic Coma:**
  - **Pathophysiology:**
    - Relative Insulin Deficiency
    - Enough to Prevent Lipolysis;
      - → NO Ketosis
    - But *Not* enough to Prevent Hyperglycaemia
      - → Hyperglycaemia
  - **Presentation:**
    - Confusion/Coma
    - Marked Dehydration
  - **Treatment:**
    - Supportive ABCs
    - Rehydration
    - Insulin Infusion

- **Hypoglycaemia:**
  - **Don't Ever Forget Glucose:**
    - Because severe or prolonged hypoglycaemia can cause Brain damage/death.
  - **Causes:**
    - ****Diabetic:**
      - Insulin Overdose (Accidental/Suicide Attempt)
      - Missed Meal
      - Exercise
      - Alcohol
    - **Alcohol Excess**
    - **Sepsis**
  - **Symptoms:**
    - **CNS Glucose Deficiency**
      - Confunsion/Coma/Seiure
      - Drowsiness
      - Incoordination
    - **Autonomic**
      - Anxiety
      - Sweating
      - Tremors
      - Palpitation
    - **Non Specifics**
      - Nausea
      - Headache
      - Fatigue
  - **Diagnosis:**
    - Hypoglycaemia
    - Clinical Symptoms
    - Response to Glucose Administration
  - **Treatment:**
    - Supportive:
      - ABCs
    - Suspect the Diagnosis:
      - Don't ever forget glucose
    - Correct serum Glucose:
      - Glucose Oral/IV
      - Glucagon (if IV Glucose isn't possible)
    - Disposition
      - Oral Hypoglycaemics (Admit to all patients)

Other Endocrine Emergencies:

- **Alcoholic Ketoacidosis:**
  - **Cause:**
    - Alcoholic with recent decreased food intake
  - **Presentation:**
    - Abdo pain, nausea & vomiting
    - Metabolic Acidosis & Possible Ketoneuria
  - **Treatment:**
    - Supportive ABCs
    - Rehydration

- **Thyroid:**
  - **Hyperthyroidism:**
    - **Cause:**
      - High free T4 & low TSH
        - Eg. Graves Disease: goitre, exopthalmos, pretibial myxoedema
    - **Presentation:**
      - →Nervousness, irritability, mental disturbance, tachycardia,
      - →palpitations, heat intolerance, weight loss, goiter
    - **Treatment:**
      - Supportive ABCs
      - Block Effects of T4 (Thyroid Blocking Drugs)
  - **Hypothyroidism:**
    - **Cause:**
      - Low free T4 & high TSH
    - **Presentation:**
      - →Fatigue, weakness, constipation, cold intolerance & depression
      - → Goitre, menstrual irregularities
    - **Treatment:**
      - Supportive ABCs
      - T4 Replacement (Thyroxine)

- **Adrenocortical Insufficiency:**
  - **Causes:**
    - **Primary:**
      - Adrenal Failure
    - **Secondary:**
      - Pituitary Failure
      - Adrenopituitary Suppression by Steroids
  - **Presentation:**
    - →Hypotension, abdominal pain, confusion, weakness, & pigmentation
    - →Hyponatraemia
    - →Hyperkalaemia
  - **Treatment:**
    - Corticosteroid Replacement (Hydrocortisone)