Summary File

Special thanks to 438's amazing team

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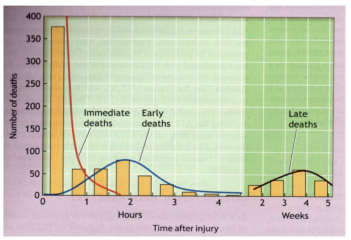
Feedback

Editing File

Trauma

Obj1: Mention the epidemiology of trauma

• Leading cause of death up to the age of 45
• 4th leading cause of death overall for all ages
•Leading causes of death & disability in Saudi Arabia



Trimodal Death Distribution [1]

First peak:
immediate deaths 50%
impacted by preventive measures

Second peak:
early deaths up to 4 hours (golden hour) 30%
impacted by early hospital care

Third peak:
late deaths by sepsis and MODS 20%
impacted by optimal early care and trauma center management

Obj2: Discuss the mechanism of trauma (Penetrating, Blunt)

Blunt injury:
• Motor Vehicle Collisions (MVC)
• Fall from height: adult = 6m, kids = 3m

Penetrating injury:
• High velocity (gun shot)
• Low velocity (stabbing)

Obj3: Discuss the triage and scoring for severity in trauma cases.

Anatomical:
AIS: The most commonly used system
ISS: summing the squares of the AIS

Minor = <9 **Moderate 9-15** **Serious 16-24** **Severe = >25**

Ex: injured face, neck and thorax.
AIS : face = 2, neck = 3, thorax = 4
ISS : (4)+(9)+(16) = 29 = severe

Physiological:
RTS: GCS + SBP + RR.

GCS: The best known physiological scoring system
Assess the ability to **open eyes + verbal response + motor responses**
The highest score is 15 (normal) and the lowest score is 3 (deep coma or death). GCS = 8 or less is considered to be in coma and must be intubated.

Table 7.1 Glasgow Coma Scale

Eyes open	
• Spontaneously	4
• To verbal command	3
• To pain	2
• No response	1
Best motor response	
To verbal command	
• Obeys verbal command	6
To painful stimulus	
• Localizes pain	5
• Flexion withdrawal	4
• Abnormal flexion (decorticate rigidity)	3
• Extension (decerebrate rigidity)	2
• No responses	1
Best verbal response	
• Orientated and converses	5
• Disorientated and converses	4
• Inappropriate words	3
• Incomprehensible sounds	2
• No response	1
Total number of points (minimum 3, maximum 15)	

Table 4. Pediatric Glasgow Coma Scale For Nonverbal Children.

Eye Opening	
Spontaneous	4
To speech	3
To pain	2
No response	1
Verbal Response	
Coos, babbles	5
Irritable cry	4
Cries to pain	3
Moans to pain	2
No response	1
Motor Response	
Follows commands	6
Localizes pain	5
Withdraws to pain	4
Decorticate flexion	3
Decerebrate extension	2
No response	1

Obj5: Describe the emergency department management per "ATLS" protocol

Injury > primary survey > resuscitation > reevaluation > secondary survey > reevaluation > optimize > transfer.

- What's the objective of **prehospital care**? prevent further injury, initiate resuscitation and transport the patient safely and rapidly to the most appropriate hospital.

Trauma

Obj6: Discuss the primary survey (diagnosis of the problems and immediate management)

1- Precautions (PPE): Cap, Gown, Gloves, Mask ...
2- Quick assessment: simple Q, the answer confirms ABCD > A: airway B: breath C: circulation D: clear sensorium
3- ABCDE:-

Airway: protect c-spine with a collar and establish patent air by:
Basic airway technique: 1-chin-lift maneuver (no head tilt) 2-jaw-thrust maneuver (better)
if not breathing then advanced airway technique: **Orotracheal intubation**, failure or extensive facial injuries? **Cricothyroidotomy**.
Extensive laryngeal injury? **Tracheostomy**.
Changed mental status > most common cause of airway obstruction > the most common indication for intubation

Breathing: adequate oxygenation and ventilation by RR, chest movement, air entry, O2 saturation (must be >90)

Immediate life threatening injuries:

1- Laryngotracheal injury / Airway obstruction
2- Open pneumothorax = initial: 3 sided occlusive dressing, definitive: chest tube
3- Massive hemothorax = Chest tube drainage
4- Tension pneumothorax = Needle thoracocentesis
5- Flail chest and pulmonary contusion
6- Cardiac tamponade = needle pericardiocentesis

Circulation: Level of consciousness, Skin color and temperature, Pulse rate and character. Management: Control hemorrhage; Apply direct local pressure when site is visible), Restore volume, Reassess patient, direct pressure, **(Lethal triad (Acidosis, Hypothermia & Coagulopathy)**

Disability: GCS and pupillary response

Exposure / Environment: undress patient and don't miss injuries, prevent hypothermia

4- Resuscitation: protect airway, O2, stop bleeding, Vigorous shock therapy and Protect from hypothermia
Special consideration: pregnant = rest on left side, pediatric and elderly = altered physiological signs and pre-existing cardiac disease or medications

Obj7: Recognize the adjuncts to primary survey

1- ECG
2-Vital signs
3-ABGs
4-Urinary Output
5-Urinary/gastric catheter
6-Pulse oximeter and CO2
7-Imaging studies: Chest X-Ray and Pelvic X-Ray.
8-Diagnostic tools: FAST and DPL
9-Consider early transfer (the time spent waiting for transportation should be spent stabilizing the patient)

Obj8: Discuss the secondary survey

Start after doing primary survey (ABCDEs) and Vital functions are returning to normal
Secondary survey: History > Physical exam Head to toe > Complete neurologic exam > diagnostic tests > Reevaluation

Shock:

Obj1: Define shock

- Inadequate oxygen delivery to meet metabolic demand
- Results in global tissue hypoperfusion and metabolic acidosis
- Can occur with a normal blood pressure

Obj2: List the types and clinical features of shock

• **Hypovolemic:** blood pump is working with no blood, most common and most readily corrected (surgical patient). Losing blood or plasma (burn) or water and electrolytes (dehydration). Clinical features: Hypotension, tachycardia, cold clammy skin then dry.

• **Cardiogenic:** caused by Impaired inflow, primary pump dysfunction and Impaired outflow. Clinical features: Hypotension, tachycardia, cold clammy skin.

• **Neurogenic:** Loss of sympathetic tone > hypotension and heart won't compensate (no sympathetic alert) typically occurs following injury to the spinal cord. Rare, **trauma** in head? Think of **hypovolemic** shock caused by hemorrhage **FIRST**. Clinical features: Hypotension, **bradycardia**, warm and dry skin (anhidrosis)

• **Vasogenic:** **Septic**: Bacteria > toxins > cell damage > release of cell mediators > uncontrolled vasodilation and leakage

• **Anaphylactic:** Allergic reaction resulting in the the release of histamine which causes vasodilation and shortness of breath. Clinical features: Hypotension, tachycardia, warm skin and **febrile** in septic

	Hypovolemic	Cardiogenic	Neurogenic	Septic	Anaphylactic
Clinical picture	-Hypotension -Tachycardia -Cold,clammy skin	-Hypotension -Tachycardia -Cold, clammy skin	-Hypotension -Bradycardia -Warm, dry skin	-Hypotension -Tachycardia -Warm, dry skin -Febrile	-Hypotension -Tachycardia -Warm, dry skin

Shock type	Hypovolemic	Cardiogenic	Obstructive[4]	Distributive[2]	Neurogenic	Dissociative[5]
Example	-Hemorrhage, -Dehydration	-Myocarditis -Dysrhythmia	-Tamponade -Tension pneumothorax	-Sepsis -Anaphylaxis	-Spinal cord injury -Traumatic brain injury	-Carbon monoxide -Cyanide
HR	↑	↑[1]	↑	↑	↓	↑
BP[6]	↓	↓	↓	↓	↓	Normal or ↑
CO (cardiac output)	↓	↓	↓	↓ Or ↑[3]	↓	↑
Capillary Refill	Delayed	Delayed	Delayed	Flash or delayed	Flash or normal	Normal
Extremity Temperature	Cool	Cool	Cool	Warm or cool	Warm	Normal
SVR (systemic vascular resistance)	High	High	High	Low or high	Low	Low to normal
Treatment	- Stop bleeding -Fluid restriction	-Inotropes -Caution with fluids -ECMO	-Pericardiocentesis -Chest tube	-Antibiotics -Fluids -Epinephrine	-Fluid resuscitation -Vasopressors	-Antidotes -Hyperbaric therapy

★Very important to memorize (classes of hypovolemic shock)

	I	II	III[7]	IV[7]
Blood loss (mL)	Up to 750	750–1500	1500–2000	> 2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	> 40
Pulse rate (per minute)	< 100	100–120	120–140	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14–20	20–30	30–40	> 35
Urine output (mL/hour)[8]	> 30	20–30	5–15	Negligible
Central nervous system/ mental status[9]	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

Obj3: Define the terminology distributive and obstructive shock

• **Distributive:** abnormal distribution of blood flow in the smallest blood vessels results in inadequate supply of blood to the body's tissues and organs.

• **Obstructive:** type of cardiogenic shock where the problem is outside the heart obstructing it. eg: tension pneumothorax, pulmonary embolism, and cardiac tamponade.

Obj4: Discuss the Pathophysiology of shock (Macrocirculation, Microcirculation, Cellular function)

• **Macrocirculation:**
In hypovolemic there is fall in intravascular volume > fall cardiac output > increase contractility and vasoconstriction.
In septic shock circulating proinflammatory cytokines leads to smooth muscle relaxation, vasodilatation and a fall in SVR.
In neurogenic loss of cardiac accelerator fibres and anhidrosis as a result of loss of sweat gland innervation.
In cardiogenic shock circulating volume is typically normal or increased (AT-II and aldosterone).

• **Microcirculation:** complications of shock override compensatory vasoconstriction > pooling of blood within the capillary bed and endothelial cell damage > loss of fluid into interstitial spaces > increase in blood viscosity formation of microthrombi.

• **Cellular function:** shifting of aerobic glycolysis to anaerobic glycolysis > metabolic acidosis. Sepsis is associated with significant mitochondrial dysfunction and marked inhibition of oxidative phosphorylation (cytopathic shock)

Shock:

Obj5: Discuss the Systemic effects of shock

CVS: ischaemia, Acid–base and electrolyte abnormalities. in sepsis inflammatory mediators depress myocardial contractility and DIC.

Nervous: anxiousness

Respiratory: tachypnoea driven by pain, pyrexia, reduced arterial PCO2 and a respiratory alkalosis (compensating for the metabolic acidosis). Septic and hypovolemic shock could cause RDS.

Renal: acute tubular necrosis, renal failure occurs in about 30–50% of patients with septic shock.

GIT: stress ulceration and haemorrhage, SIRS and multiple organ failure.

Hepatobiliary: ischaemic hepatic injury, Increases in prothrombin time and/or hypoglycaemia are markers of more severe injury.

Obj6: Discuss the general principles of management (airway, breathing and circulation)

discussed in details in trauma lecture

Obj7: Discuss the Specific treatment of each type of shock

• **Hypovolemic Shock:** ABCs > Control any bleeding > establish 2 large bore (14 or 16 gauge) better than central line > crystalloids > PRBCs preferred in hemorrhage (O negative or cross matched) > definitive treatment, Then evaluate:

- CBC & Electrolytes
- ABG (arterial blood gas/Lactate)
- Kidney function: BUN (blood urea nitrogen./Creatinine)
- Coagulopathy: Coagulation studies
- Types and cross match

As indicated:

- Trauma patient: CXR, Pelvic X-Ray
- Stable patient to detect bleeding source: CT scan
- Incase of hematemesis: GI endoscopy
- Incase of hemoptysis: Bronchoscopy
- Incase of history of AAA: Vascular radiology

• **Septic shock:** obtain blood culture > broad spectrum antibiotic > resuscitate > Hypotension persist? Vasopressors

• **Anaphylactic shock:** clinical signs of anaphylactic reaction (the hallmark airway compromise) > ABCs > oxygen > epinephrine > IV fluid > second line (corticosteroids and antihistamine)

• **Cardiogenic Shock:** based upon the identification and treatment of reversible causes then supportive: administration of high concentrations of inspired oxygenation > IV opiates (MI patient) > correction of hypovolaemia (not in HF patient) not responding? dobutamine

• **Neurogenic Shock:** IV fluid resuscitation > hypotension persist? Vasopressors

How to know resuscitation is enough?
1-Normal vital signs.
2-Normal serum lactate levels.
3-Evidence of adequate tissue perfusion (normal mental status, normal urine output (best marker) and normal liver function).

Burn:

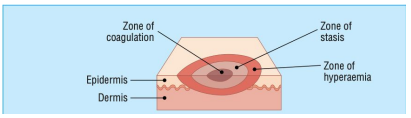
Obj: Define burn. Destruction of tissues caused by various etiologies including flames, and hot liquids, that ranges from trivial to life threatening which requires extensive treatment and rehabilitation with the chances of permanent dysfunction and distortion.

Obj: Discuss the incidence of burn.

- 2 million burns per year in the US.
- Mortality is highest in the age groups: 2-4 years & 17-25 years.
- 500K burns treated in the ER.
- 70K burn hospital admissions.

Obj: Discuss the pathophysiology of burn

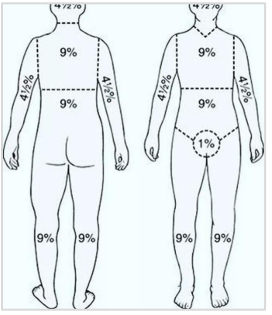
- ★ local effects → inflammatory response → capillary dilation (erythema) or capillary damage (edema) → if 15% of body surface burned = severe hypovolemia = hypovolemic shock and decreased preload
- Destruction of the Epidermis impair physical barriers and predispose to infections: most common organisms = staph, strept and pseudomonas (sepsis)
- ★ **Burns have 3 zones :** 1-Zone of coagulations 2-Zone of stasis (the area of potential reversible cell damage) 3-Zone of hyperemia.



Obj: Recognize the calculations

Parkland formula is very important

- ★ **Rule of nine:**
  - **Palm without fingers = 1% (Palmer method)**
  - **Head in kids = 18% (9% in adults)**
  - **Single leg in kids = 9% (18% in adults)**
- **Mortality:** (body surface area% + age)/100
- ★ **Parkland formula:** administration during the **first 24 hours = 4mL × weight (kg) × TBSA%**
  - ★ 50% given in the first 8 hours
  - ★ 50% given in the next 16 hours
  - ★ Start counting the hours from the start of injury not from the time patient reach the hospital
- ★ **4-2-1 rule (for maintaining fluid) :**It is the calculation of hourly fluid need according to weight.
  (4 ml/kg/ hour for the first 10 kg , 2 ml/kg/ hour for the next 10 kg and 1 ml/kg/ hour for every kilogram after that.)
  Example (70kg): 4x10 + 2x10 + 1x50 = 110
- **When to transfer to a burn center (transfer criteria)?**
  - >25% body surface area (BSA).
  - >20% BSA in children.
  - High voltage burns, Inhalation injuries and chemical burns.
  - Burns in the genital area, face, neck, feet and hands in addition to full thickness burns.



Obj: List the types of burn

Based on depth:-

- **First degree (superficial):** involves epidermis only (edema and erythema)
- **Second degree (superficial/deep partial thickness):** superficial: epidermis and part of dermis (papillary), deep: epidermis and dermis (reticular) (blisters).
- **Third degree (full thickness):** involves Entire dermal layer and subdermal fat (dry, inelastic and waxy scar)(Escher)
- **Fourth degree (full thickness) :** all skin layers + bones, muscles and tendons
- In clinical practice, most burns are a mix of types. Any burn is surrounded by lighter zones eg: 2nd degree burns are surrounded by 1st degree burns and 3rd degree burns are surrounded by an engorge erythematous area (2nd degree burns). First site to come in contact with the burning agent is the deepest site of the burn. Retainment of sensations at the site of the burn suggest more superficial injury.

Based on agent:-

- **Thermal**
- **Chemical :** can cause **Hypo**calcemia
- **Electrical**

Burn:

Obj: Explain the inhalation injury

- Usually fatal caused by inhalation of smoke from burning objects eg:plastic.
- Carbon monoxide displaces oxygen and binds to hemoglobin forming (Carboxyhemoglobin)
- Cyanide inhalation is also common and can be fatal
- Damage to lung parenchyma can happen, patient can be saved if he/she was put on 100% O2

Obj: Explain the chemical Burn

- chemical agents react with the body's tissue and the injury extends deeper it doesn't just end with removing the agent , it must be flushed.
- **Acid:** the most common and causes necrosis
- **Base:** the most dangerous
- **Factors worsen:** Area of contact, PH & concentration, form of agent, duration, amount.

Obj: Explain the electrical Burn

- burning from inside out from deeper organs, patients who experience electric shock may have no external injuries.
- **Severity depends upon:** Voltage, Amperage, Resistance, Type of current, Duration
- Low voltage is the worse and it follows the least resistance organs. High voltage direct flow
- ★ **Systemic injury:** Cardiac arrhythmias, sepsis, Renal failure (Due to muscle necrosis), PNS injury , initial management should be cardiac monitoring.
- Cause compartment syndrome (details in arterial lecture)

Obj: Discuss the burn management

- **Objective of treatment:** 1-prevent edema 2-prevent contractures 3- prevent infection 4-preserve viable tissue
- **First degree:** Mild analgesics/NSAIDs (nerve endings intact), daily cleansing and silver sulfadiazine (flamazine).
- **Second degree:** same as 1st degree and leave blisters intact, if debrided? Occlusive dressing.
- **Third degree:** surgically debride and remove the eschar then skin graft.
- **Fourth degree:** skin graft not adequate. Amputation or flap coverage.
- **Wound closure:** in deep second degree when burn didn't heal after 2 weeks
- ★ **Chemical:** as GP dilute it with water to minimize its effect "The solution of pollution is dilution"
- **Electrical:** Fasciotomy (compartment syndrome), Debridement of dead tissue, definitive: Amputation or flap coverage
- ★ **Prophylaxis against Tetanus**
- **Surgical:-**

★ **Escharotomy:** incision through the eschar, most important indication is **circumferential burn** (which causes hypoperfusion due to edema).

Grafting: removing thin sheet of skin from one area of the body and transplanting it to a different area of the body, doesn't have blood vessels, used more with 3rd degree burns .

Flap: using bulky tissue (e.g. muscle flap, subcutaneous tissue flap) that has its own blood supply , used with deep burns (4th degree burns).

Obj: Identify the complications of burn

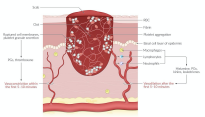
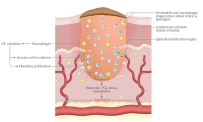
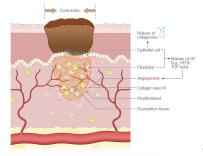
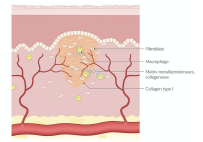
- **Early consequences:** Hypovolemic shock, Electrolytes imbalance, Sepsis, Hemolysis and Hypothermia.
- **Short term consequences:** Nutritional depletion, Respiratory failure, Renal failure, Venous thrombosis and Curling's ulcer
- **Long term consequences:** Permanent disfigurement, Prolonged hospitalization, Psychological disturbance

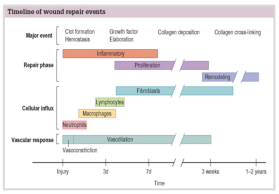
Wound Healing:

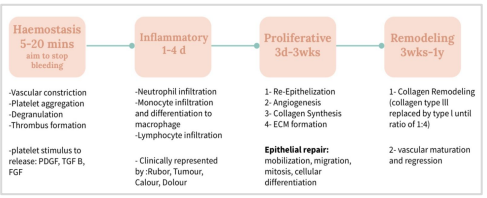
Obj1: Define the wounds

Wound: disruption of normal anatomical structure and function, can be chronic and acute.
Classified into Incised wounds (sharp instrument), Abrasions, Degloving, Crush, Burn and Gunshot.

Obj2: Discuss the phases of wound healing

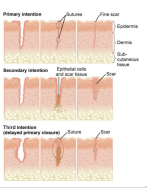
Heamostasis (5-10min)	**Main cells are: platelets**: form the platelet plug, degranulation of platelets and recruitment of neutrophils stimulated to release: • **Platelet derived growth factor (PDGF)** • **Transforming growth factor β (TGFβ)**: has a major role in wound healing (Excess causes abnormal scars) produced by **α granules** • **Fibroblast growth factor**	
Inflammatory / Migratory "lag" phase (1-4 Days)	Presented by rubor (redness) , tumour (swelling), calour (heat) and dolour (pain). **Main cells are:** First 24 hours → **PMNs (neutrophils)** After 24 hours (Key cell) → **Macrophages**: Phagocytosis, wound debridement, activation of fibroblast, angiogenesis and matrix synthesis regulation	
Proliferative / Fibroplasia "incremental" (3 days - 3 weeks)	**Main cells are: Fibroblasts** Dominant cell type peaking at 7-14 days Re-Epithelization, angiogenesis, collagen synthesis and ECM formation	
Remodeling / Maturation (3 weeks - one year)	• Type III collagen replaced by type I collagen and become organized and the scar became mature. • Re-establishing normal 4:1 ration (I:III): Duration depends on age, genetics, type of wound, location. Tensile strength increases to 80% of pre-injured skin.	





Obj3: List the types of wound healing

By types:	• **Primary:** horizontal repair generate fine scar • **Delayed Primary:** we wait for 2-4 days then primary healing • **Secondary:** horizontal and vertical repair (edges far from each others) • **Partial-thickness:** vertical repair (dermis not involved) - **Horizontal repair**: by contraction (edge to edge proximation) - **Vertical repair**: by epithelializatiion
By timing:	• **Acute:** 1st week • **Subacute:** 1-6 weeks • **Chronic:** > 6 weeks
By abnormal healing:	• **Overgrowth:** Hypertrophic and Keloid • **Undergrowth:** Chronic unstable wounds (pressure sore and diabetic wounds) • **Abnormal pigmentation** • **Contour abnormality**
Wound closure:	• **1° intention:** Primary closure by suturing the edges together. • **2° intention:** Wound left open to heal by processes of granulation, contraction and epithelialization • **3° intention:** delayed primary healing (a combination of primary healing and secondary healing). Desired for contaminated wounds



Wound Healing:

Mnemonic

DID NOT HEAL

Obj4: Recognize the factors affecting wound healing

General (patient):	Local (wound):
1. Nutrition 2. Drugs/Toxins 3. Age 4. DM 5. Smoking 6. Vascular disease 7. Obesity 8. Systemic diseases 9. Idiopathic 10. Inherited diseases 11. Surgical technique	1. Oxygen (Hypoxia) 2. Infection 3. Acidity 4. Radiation 5. Loss of growth factors 6. Denervation (in diabetics) 7. Iatrogenic 8. Edema 9. Cancer 10. Foreign body Chemotherapy treatment can be started after 14 days.

Obj5: Identify the abnormal wound healing

- **Ideal scar**: flat narrow, good color, parallel to RSTL, doesn't restrict function, asymptomatic and mature in 6-18 months

- **Abnormal scars:**
  Keloid scar: excision can be done as an alternative
  Hypertrophic scars: these scars should **not** be excised
  Wide scar: Caused by traumatic wounds that are not closed properly

- **Treatment** :
  - Prevention
  - Medical: steroids (first line), chemotherapy (5-FU) or radiation
  - Non surgical: pressure, silicone gels or sheets
  - Surgical: laser or surgery

  If failed in keloids the best treatment approach is a combination of intralesional excision followed immediately by low-dose radiotherapy

Very important

Features	Hypertrophic scar	Keloid scar
Genetic	Not familial	May be familial
Race	Not race related	Black > White
Sex	Female = Male	Female > Male
Age	Children	10-30 years
Borders	**Remains within wound (in the same area of scar)**	**Outgrows wound area**
Natural history	Subsides with time	Rarely subsides
Site	Flexor surfaces	Sternum, shoulder, face
Aetiology	Related to tension	Unknown

Not included in objectives but important!

Classification of contamination in Surgical wounds		
1-Clean (class I)	Nontraumatic, non infected wounds & no breach of Resp, GI, or GU tract. (No need for antibiotics)	Thyroid and breast surgeries.
2-Clean-contaminated (class II)	Small breach in protocol; Resp/GI/GU tract are entered with minimal contamination.	Cholecystectomy **Uncomplicated** appendicitis Intestinal resection ONLY if there was **no spillage.**
3-Contaminated (dirty) (class III)	Fresh traumatic wounds; major break in sterile technique, **nonpurulent** inflammation; in or near contaminated skin.	Hemicolectomy Resection of the intestine **with spillage.**
4-Infected (class IV)	**Purulent** infection.	Traumatic open bone fracture **Purulent** pyogenic perforated appendicitis.

Wound hematoma increases the risk of infection in surgical wounds.
To avoid contamination of the surgical wounds the sterile wound dressing pad should be applied after cleaning and drying the wound before removing the drape

Collagen:

- ★ Lysine and proline (purines) hydroxylation required for cross linkage (the main step in collagen synthesis) the role of Vit.C
- ★ **Normal skin ratio - Type I:Type III = 4:1,** Hypertrophic / immature scar 2:1 ratio
- ★ Formation of collagens is inhibited by: Colchicine, penicillamine, steroid, Vit.C deficiency and Fe deficiency. (They activate collagenase which degrades collagen synthesis and inhibits cross linkage hydroxylation of lysine and proline)

Metabolic response to injury

Obj1: Factors mediating the metabolic response.

Our aim is to blunt EBB phase as it will lead to further tissue damage and supplement patients in Flow phase to assure healing and prevent complications

Ebb phase (↓energy expenditure)	**Flow phase** (↑energy expenditure)	
Characterized by hypovolemic shock. Maintain homeostasis by: ↓Cardiac output ↓Oxygen consumption ↓Blood pressure ↓Tissue perfusion ↓Body temperature ↓Metabolic rate	Burn elicit the most severe metabolic response and we try to support the patient during the flow phase ↑Catecholamines ↑Glucocorticoids ↑Glucagon Release of cytokines , Lipid mediators , Acute phase protein production , ↑ metabolic rate	
	Initial Catabolic phase	**Anabolic phase**
	Lasts about a week Characterized by: 1-High metabolic rate 2-Breakdown of protein and Fat 3-A net loss of nitrogen (negative nitrogen balance) 4-Weight loss.	Lasts for 2-4 weeks 1- Protein and fat are stored 2- Positive nitrogen balance 3- Weight gain.

	Response
Acute Inflammatory Response	• Cellular activation. • Inflammatory mediators (TNF, IL1,IL2, IL3, IL4 and IL6). • Paracrine vs endocrine effects.
Endothelium	• Selectins, Integrins and ICAMs. • Nitric Oxide > vasodilation > edema. • Tissue Factor > coagulation > DIC.
Afferent nerve stimulation	• Sympathetic nervous system activation. • Release of hormones from adrenal medulla. • Stimulation of other pituitary hormones.
Endocrine system	★ More stress hormones, less anabolic hormones: ○ **Pituitary gland:** ↓GH, ACTH and ADH. ○ **Adrenal:** cortisol, aldosterone. ○ **Pancreas:** glucagon,↓Insulin. ○ **Other:** renin, angiotensin,↓sex hormone,↓T4.

Obj2: Consequences of the metabolic response.

- The metabolic response limits the injury and initiate the repair process by mobilizing substrates.
- Help in preventing the infections but could cause distance organ damage.
- **More consequences**:

Hypovolemia	- Characterises moderate to severe injury - Loss of blood, electrolyte, water and protein rich fluid
Energy	★ Overall energy expenditure rises up to 50% due to: 1. **Thermogenesis**: 1C°increases BMR by 10% 2. **Increased BMR**
Catabolism and starvation	★ It will affect the metabolism of carbohydrate, fat, protein **Acute phase:** glycogenolysis and gluconeogenesis in liver releasing glucose for brain, Lipolysis and FFAs release. **Chronic phase:** initially muscle catabolism to convert amino acids into glucose and FFAs into ketones (mainly) then when fat stores depleted muscles are used again as final energy source
RBC synthesis	Anemia and hypercoagulability 1-Anemia: when Hgb <8 transfusion indicated. 2-Hypercoagulability and may develop DIC.
★ Anabolism	After the inflammatory mediators are no longer produced + (adequate nutrition and early mobilization) body will regain weight and skeletal muscle mass . Hormones contributing: Insulin, GH, androgens and IGF-1

Metabolic response to injury

Obj3: The differences between metabolic responses to starvation and trauma.

Hormone	Source	Change in Secretion
Norepinephrine	Sympathetic Nervous System	↓↓↓
Norepinephrine	Adrenal Gland	↑
Epinephrine	Adrenal Gland	↑
Thyroid Hormone T4	Thyroid Gland (changes to T3 peripherally)	↓↓↓

	Starvation	Trauma or Disease
Metabolic Rate	↓	↑↑
Body Fuels	Conserved	Wasted
Body Proteins	Conserved	Wasted
Urinary Nitrogen	↓	↑↑
Weight Loss	Slow	Rapid

Obj4: The effect of trauma on metabolic rate and substrate utilization.

Endocrine Response

Fatty Deposits → Fatty Acids

Liver & Muscle (glycogen) → Glucose

Muscle (amino acids) → Amino Acids

Obj5: Modifying the metabolic response

- **Nutritional status**
- **Temperature**
- **Medication (Before or after injury)**
- **Severity of injury**
- **Anesthetic technique**

Cardiac science

Common cardiac operations:

- **Coronary Artery Bypass Grafting (CABG) most common**
- Valve Replacement/Repair
- Repair of congenital defects: VSD or ASD
- Heart Transplantation

Classes of surgical indications:

Class I	There is confirmed benefit from surgery We have to **do surgery**
Class IIa	The studies which show **benefit is more** than studies show no benefit
Class IIB	The studies which show **no benefit is more** than studies show benefit
Class III	No study shows benefit **Do not do it**

Basic principles of cardiac Surgery:

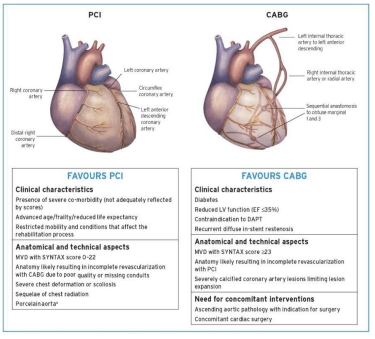
- **Access** (Sternotomy, Thoracotomy, Robotic or Endoscopic)
- **Bloodless Operative Field** (Suction and re-transfusion)
- **Static Operative Target** (Cardiac Arrest, Ventricular Fibrillation, Mechanical Stabilizers)
- **Preservation of Myocardium** (Off-pump Techniques, Hypothermia ,Cardiac Arrest with cardioplegia)
- **Preservation of body perfusion** (heart lung machine)

Coronary Artery Bypass Grafting (CABG)

- A vascular graft is sutured to the coronary artery beyond the stenosis to Improves blood flow to the heart.

★ Most important preoperative evaluation of CABG is **cardiac catheterization.**

- **Indications for CABG:**
  - Failure of medical therapy or percutaneous intervention.
  - Mechanical complications of myocardial infarction (rupture of wall of the heart, septum and chordae tendineae. Tamponade. Valve weakening)
  - Associated valve disease.
  - **Stenosis in these major arteries:**
    - **Left main stenosis > 50% (most important vessel)**
    - **Stenosis of proximal LAD (Most commonly affected artery in IHD) and proximal circumflex > 70%**
    - **3 vessel disease with left ventricular dysfunction/Diabetes**
    - **2 vessel disease**



- **Conduit for CABG:**
  - **Venous** : Long saphenous vein with antiplatelets, statins It gives a better outcome has patency rates of around 70% at 5 years
  - ★ **Arterial**: Internal thoracic artery (internal mammary artery) It has a very high graft patency exceeds 95% at 5 years , Radial artery

- **Types of surgery:**
  - With pump: Can affect the organs > severe systemic Inflammatory reaction, May cause end organ damage
  - Without pump : Do the surgery while the heart is working (Used only for CABG). Risk of bleeding, No end organ damage
    - Benefits of without pump in CABG:
      - Reduced incidence of stroke and cognitive problems
      - Lesser renal dysfunction
      - Reduced inflammatory response (elderly can't tolerate the inflammatory response associated with the pump)
      - Lesser coagulopathy requirements of blood transfusion and less bleeding
      - Reduced length of time in intensive care & hospital stays
      - Reduced morbidity and mortality rates

Cardiac science

Valvular Heart disease

- **Aortic stenosis**
  - Etiology : Rheumatic , Congenital, Degenerative, calcific aortic stenosis
  - Symptoms: chest pain, Dyspnea , Syncopal attacks
  - Signs : pulsus parvus and tardus, harsh ejection systolic murmur. aortic component of S2 is soft
  - Treatment :
    - Medical: treat the symptoms
    - Surgical **indication**: **(valve area <1cm, L.V dysfunction <50% or dilation , AFib, pulmonary HTN)** Transcutaneous aortic valve implantation (TAVI) , percutaneous balloon valvuloplasty

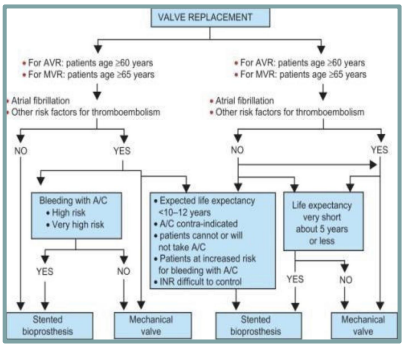
- **Aortic regurgitation**
  - Etiology :Rheumatic , Congenital, Connective tissue disorder, Aortic dissection or aneurysm
  - Symptoms: Asymptomatic, Palpitations , dyspnea. Orthopnea, P.N.D, Angina in severe cases only
  - Signs : wide pulse pressure , Peripheral signs of aortic regurge , Generalized vasodilation resulting in warm hands and feet, and increased sweating , Hyperdynamic displaced apex
  - Treatment : Surgical: Aortic valve replacement(A.V.R) should be considered if there is excessive dilatation of the L.V.

- **Mitral stenosis**
  - Etiology :Rheumatic , Congenital, L.A myxoma
  - Symptoms: asymptomatic, pulmonary congestion , Palpitations ,Dysphagia, compression of left main bronchus Symptoms, Symptoms of low cardiac output
  - Signs : Loud S1, Mid-diastolic rumbling murmur, signs of pulmonary hypertension,Irregular pulse
  - Treatment :
    - Medical: for mild cases
    - Surgical **indication**: **(valve area <1cm, L.V dysfunction <50% or dilation , AFib, pulmonary HTN)** percutaneous balloon valvuloplasty (with no evidence of a LA clot or mitral regurgitation) , Open mitral commissurotomy, Valve replacement/ repair

- **Mitral Regurgitation**
  - Etiology :Rheumatic, Congenital abnormalities, Degenerative, Endocarditis, Dilatation of the L.V. and mitral valve ring, Dysfunction of the papillary muscle, Calcification of the mitral valve annulus
  - Symptoms: Asymptomatic, fatigue and weakness, Dyspnea, Orthopnea, PND, Pulmonary hypertension
  - Signs : apical thrill , apical pansystolic murmur , signs of pulmonary hypertension
  - Treatment :
    - Medical : acute MR temporarily
    - Surgical: Valve repair/ replacement repair

Valvular prosthesis زراعة صمام القلب

- **TYPES**
  - ⭐Mechanical
    - Lasts > 20 years, Lifelong anticoagulation , Click
    - Unless there is contraindication to anticoagulation, mechanical valves are commonly used in a **younger** age group
  - Biologic
    - Lasts 8 – 10 years, No anticoagulation, No click
    - We use biological valve for old age and contraindication for anticoagulant like in female childbearing age

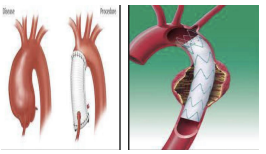


- **Complications:**
  - Thrombosis , BLEEDING, Paravalvular leak , Infective endocarditis , Degeneration

Cardiac science

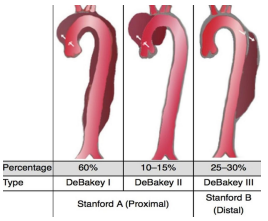
Aortic diseases

- **Aortic Aneurysm** (abnormal dilatation of the aorta, Symptoms are usually due to pressure on surrounding)
  - If the absolute size of the aneurysm is >5.5cm or Growing >0.5 cm /Year
  - Not all patients are the same, we treat each patient depend on his age, height, morbidity (bicuspid valve, connective tissue disease, Marfan's syndrome,) certain patient we call them high risk patients
  - We operate on them in 0.5 cm smaller ratio than other individuals (5 or 4.5cm), The operation is an aneurysm Repair



*(from Females slides)

- **Aortic dissection** (Tear in the intima creating 2 lumens separated by the dissecting membrane)
  Knife like pain in the chest radiating to the back and epigastric region with widened mediastinum on chest x-ray
  - TYPES according to stanford classification:
    - **A :** ascending aorta , Medical emergency and require immediate surgery
    - **B :** descending aorta , Carry a lower mortality rate and can be managed medically
  - TYPES according to DeBakey classification:
    - **I** and **II** : could be categorized as **A**
    - **III** : could be categorized as **B**



Heart failure

	Indications	Absolute Contraindication
Heart Transplant	• Cardiogenic shock requiring mechanical assistance = Medications didn't work • Refractory heart failure with continuous inotropic infusion. • NYHA functional class 3 and 4 with a poor 12 months' prognosis • Progressive symptoms with maximal therapy • Severe symptomatic hypertrophic or restrictive cardiomyopathy • Medically refractory angina with unsuitable anatomy for revascularization • Life-threatening ventricular arrhythmias despite aggressive medical and device interventions • Cardiac tumors with low likelihood of metastasis • Hypoplastic left heart and complex congenital heart disease • Sometimes we know the reason of HF and we transplant heart because we know the patient won't make it out with any other surgery or the ejection fraction very low	• Pulmonary hypertension (TPG "transpulmonary gradient" > 15 mmHg, SPAP "systolic pulmonary pressure" > 50 mmHg, PVR "pulmonary vascular resistance" > 4 WU, PVRI "pulmonary vascular resistance index" >6) • Diabetes mellitus with end organ damage • Elevated creatinine (>200 umol/L). • Psychosocial (substance abuse, smoking, medical noncompliance) • Active infection • Malignancy (within 5 years) • Marked cachexia (<60% ideal body weight) won't tolerate surgery • Morbid obesity (>140% ideal body weight) • Osteoporosis • Peripheral or cerebrovascular disease • Systemic disease (anticipated to limit long-term survival) won't live long, we may transplant heart for patient with chronic diseases if another organ gonna transplant for him, like patient with end stage kidney disease will transplant a kidney (for some reason transplanting 2 organs have higher success rate)
Ventricular Assist device	• Frequent hospitalizations for HF • Intolerance to neurohormonal antagonists • NYHA IIIb-IV functional limitations despite OMT • End-organ dysfunction owing to low CO • Increasing diuretic requirement • CRT non responder • Inotrope dependence • Low peak Vo2 (<14mL/Kg/min)	• Irreversible hepatic disease • Irreversible renal disease • Irreversible neurological disease • Medical nonadherence (don't take thrombolytic therapy) • Sever psychosocial limitations

Cardiac science

Heart failure (Cont..)

NYHA classification:

Class I	No limitations of activities. Symptoms only **occur with vigorous activities**
Class II	Slight or mild limitation of activity. Symptoms **occur with prolonged or moderate exertion**
Class III	Marker limitation if activity. Symptoms **occur with usual activities of daily living**
Class IV	Symptoms **occur at rest**. Incapacitating.

Endocarditis

- infection of cardiovascular structure including valves and intracardiac foreign bodies as pacemaker leads, prosthetic valves and surgical patches
- Diagnosis can be done according to **DUKE criteria,** if the patient have 2 major criteria, 1 major and 3 minor, or 5 minor criteria

Major criteria	Minor criteria
1- Microbiological evidence: ○ Typical microorganisms consistent with IE from 2 separate blood ○ cultures persistently positive blood cultures with other organisms: i. At least 2 +ve cultures drawn >12 hours apart ii. All 3 or majority of 4 separate cultures with the first and last at least 1 hour apart iii. Single +ve culture for Coxiella burnetti **2- Evidence of endocardial involvement:** ○ Oscillating intracardiac mass ○ Positive echocardiographic findings of vegetations, Abscess ○ New partial dehiscence of prosthetic valve ○ New valvular regurgitation	• Fever • Echo findings (Any finding not involved in the major criteria) • Vascular phenomena (arterial emboli) • Evidence from microbiology (Positive blood culture but does not meet a major criteria) • Risk factors and predisposition (VHD, prosthetic valve, previous IE) or IV drug users • Immunological phenomena (Glomerulonephritis)

- **Treatment :**
  - Mainly first line treatment is Medically
  - But there are certain conditions that require surgical intervention.
    - **urgent surgical intervention [immediate]:**
      - severe acute regurgitation or obstruction or fistula into a cardiac chamber or pericardium that's causing refractory pulmonary edema or cardiogenic shock
      - Locally uncontrolled infection (abscess , false aneurysm, fistula, enlarging vegetation, or Urgent dehiscence of prosthetic valve)
    - **Conditions that we wait for treatment and we assess [early intervention]:**
      - severe acute regurgitation or obstruction and persistent heart failure (not responding to therapy)
      - Infection caused by fungi or multi-drug resistant organisms , such as pseudomonas aeruginosa and other gram negative bacilli
      - large vegetation (>10mm=1cm in length) after one or more embolic episode, despite appropriate antibiotic therapy
      - Persistent Fever
    - **Conditions that we wait until finishing Abx course [Late intervention]:**
      - large vegetation (>10mm in length) and other predictors of complicated course (heart failure, persistence infection with staph aureus or fungal, or abscess)
      - HF that's easily controlled with medical treatment
      - Patient doesn't have any criteria but he has valve structural problem ex: AS,MR,MS

Cardiac science

Arrhythmia

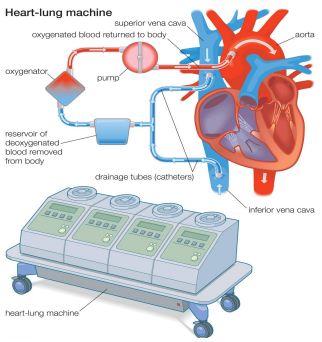
- **If we are suspecting that the pt have arrhythmia:**
  - Investigate
  - If positive findings start Medical therapy (first line)
  - If there's no response to medications then do Ablation by Cath
  - If still there's no response we go for surgery (ablation) , but not commonly done

- **Recommendations for Surgical ablation**
  - should be considered in patients with symptomatic AF undergoing cardiac surgery (for another reason)
  - may be performed in patients with asymptomatic AF undergoing cardiac surgery if feasible with minimal risk

Cardiac tumors

- **Clinical features:**
  - Common, Obstruction and Embolization
  - Nonspecific, Fever and fatigue
- **Investigation:**
  - Echocardiography
  - CT or MRI
- **Types:**
  - Benign
    - Myxoma , Rhabdomyoma , fibroma and others
  - Malignant
    - Rhabdomyosarcoma, Fibrosarcoma , Angiosarcoma
- **Management:**
  - Benign, excise it <u>with consideration of the size and site</u>
  - Malignant, Regardless the size we never excise it

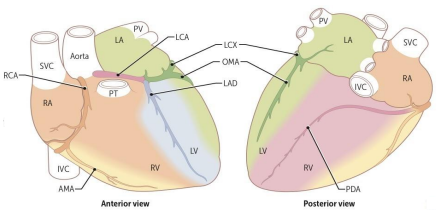
Heart/ lung Machine <u>aka cardiopulmonary bypass (CPB)</u>



- facilitate cardiac and thoracic aortic procedures by excluding the heart and lungs from the circulation whilst providing:

  1- adequate gas exchange
  2. systemic organ perfusion
  3. controlling body temp.

- **We have two main coronary arteries:**
  - Left coronary artery (LCA) supplies 75% of the heart
  - Right coronary artery (RCA) supplies 25% of the heart
- LCA gives:
  - ★ Left anterior descending (LAD) supplies 75%
    (most commonly involved in ischemic heart disease)
  - Left circumflex (LCX) supplies 25%



Transfusion of blood and plasma products

- Blood & Plasma component

- **PRBCs**
  - Administer with normal saline
  - 1 unit increase Hb 1g/dL and hematocrit 3%
  - Can be stored for 42 days in the refrigerator and 1 year in the freezer
- **Platelets**
  - Cannot be refrigerated
  - Should be Rh negative
  - Can be stored for 5 days
- **Plasma**
  - Used mainly by dermatologist
  - Contains (Albumin, Immunoglobulin, Factor VIII, IX, & Prothrombin)
  - Can be stored for 1 year in the freezer
- **Cryoprecipitate**
  - Source of fibrinogen & vWF
  - Can be stored for 1 year in the freezer

- RBC serology

	A+	A-	B+	B-	AB+	AB-	O+	O-
Abs in Plasma	Anti-B		Anti-A		None		Anti-B & Anti-A	
Antigens in RBC	A antigen		B antigen		A & B antigens		None	
Can donate to	A+ & AB+	A-, AB-, A+ & AB+	B+ & AB+	B-, AB-, B+, & AB+	AB+	AB-, & AB+	O+, A+, B+, & AB+	All blood types
Can receive	A+, A-, O+, & O-	A- & O-	B+, B-, O+, & O-	B-, & O-	All blood types	AB-, A-, B-, & O-	O+ & O-	O-

- Pre-transfusion testing

1. **ABO grouping (+RhD typing)**
2. **Antibody screen (**For non (ABO/D) antigens)
3. **Cross-Matching** Patient's Blood is sent to the blood bank and cross-matched **for specific donor units for possible blood transfusion**
4. Two qualified personnel check it at the bedside to prevent a potentially fatal clerical error.

Transfusion Indications

- Threshold of Hb <7
- Increase oxygen carrying capacity.
- Restoration of red cell mass.
- Correction of bleeding caused by platelet dysfunction .
- Correction of bleeding caused by factor deficiencies .
- Correction of anemia.

★ According to American society of anesthesiologists:
- **Rarely** >10g/dl, **always needed** <6 g/dl

Administration

- Double check
- Check by Id not by name
- Check Expiration date
- DO2= CO x CaO2
- Cardiac Output (CO) = HR x SV
- Oxygen Content (CaO2):
- (Hgb x 1.39)1 x O2 Saturation + (PaO2 x 0.003)2
- Hgb is the main determinant of oxygen content in the blood

- Transfusion Adverse effects. (All managed by stopping the transfusion)

Immunological	Non- immunological
Acute • Hemolytic • Febrile-Non Hemolytic • Transfusion-related Acute Lung Injury • Urticarial (allergic) • Anaphylactic Delayed • Hemolytic • VHD	Acute • Fluid overload • Hypothermia • Electrolyte toxicity Delayed • Iron Overload • Infections

★ **Massive blood transfusion:**
Transfusion of large amounts of RBC units

For example:
1) Complete replacement of a patient's blood volume (10 units of RBCs) within 24 hours
2) Replacement of 50% of patient's blood volume (5 units of RBCs) with in (3 or 4 h)
3) Transfusion of 3 units of pRBCs within 1 hour

Approach to common Thoracic & Lung Diseases:

1- Congenital Lung Disease:

Agenesis

No development / complete absence of the lungs.

Hypoplasia

Incomplete development of the lung.

Cystic adenomatoid malformation:

overgrowth of abnormal lung tissue that may form fluid-filled cyst

- **Presentation**:
  Pediatric patients with:
  - Pneumothorax/Hemothorax
  - Repetitive chest infections
  - Fever
  - CXR abnormalities
- **Treatment**: surgery → remove that mass

Lobar emphysema

Entire lobe replaced with a big cyst or emphysematous bullae (usually the upper right lobe)

- **Presentation:** newborn unable to breath → +ve pressure mechanical ventilation (long-term) → affects nearby organs as bullae enlarges
- **Treatment**: lobectomy → removal of the entire lobe

Pulmonary sequestration

Non-functional mass of normal lung tissue that lacks normal communication with the airways, can be intra or extra lobular (most common left lower lobe), Receives its own supply from the systemic circulation (most common thoracic aorta)

- **Presentation**:
  - Repetitive chest infection
  - Mass on CXR or CT-scan (usually left lower lobe)
- **Treatment**: CT-scan with contrast and tie the arterial supply (usually from thoracic aorta) then remove the sequestration.

Bronchogenic cyst:

Benign cyst with malignant position (Right para-tracheal most common & subcarinal)

- **Presentation**: dysphagia, stridor
- **Complications**: Infections, hemorrhage , dyspnea , transformation to adenocarcinoma
- **Treatment**: CT then a surgical removal by thoracoscopy, If SVC is involved → thoracotomy

2- Infectious Lung Disease:

Lung abscess: ★

- **Etiology**:
  - Immunocompromised (Diabetic, HIV)
  - Pneumonia
  - Bronchial obstruction
  - Foreign bodies
  - Bronchiectasis
- **Presentation**:
  - Large amount of foul-smelling sputum
  - Productive cough
  - +/- hemoptysis
  - Septic or toxic high fever & chills
  - Severe chest pain and Dyspnea
  - Weight loss
- **Investigation:**
  - CXR (air-fluid level abscess)
  - CT-scan
- **Treatment:**
  - **Concerted:** Antibiotic + Drainage (Pigtail catheter)
  - **Surgery:** Lobectomy, Segmentectomy, Pneumonectomy.

★ **Indication for surgery**: Failure of Tx , Giant abscess >5 cm , Hemorrhage , can't r/o carcinoma , rupture with resulting empyema.

Approach to common Thoracic & Lung Diseases:

Bronchiectasis ★

- **Etiology:**
  - Cystic fibrosis
  - Immotile cilia syndrome
  - Infections: Untreated pneumonia , Measles disease
  - Obstruction by foreign body.
- **Types:**
  - Cystic (surgically corrected)
  - Cylindrical (not surgically treated, Cystic fibrosis, immotile cilia syndrome)
- **Presentation**:
  - Productive morning cough
  - Dyspnea
  - Hemoptysis
  - Clubbing
  - Psychological problem (children)
- **Investigations:**
  - CXR
  - CT (confirmation)
  - Bronchoscopy (to detect obstruction)
  - Bronchogram Confirmatory
- **Treatment:**
  - **Medical:** for bilateral conditions (Abx, Supportive, Postural drainage)
  - **Surgery indication:**
    - Failure of medical Tx
    - Localized disease
    - Cystic type
    - Non perfused (measured by ventilation-perfusion scanning)

Tuberculosis

- **Etiology:**
  - Pulmonary
  - TB empyema
  - TB lymphadenitis
- **Presentation**:
  - Fever
  - Night sweats
  - Weight loss
  - Malaise
  - Dry cough
  - Purulent sputum
- **Investigation:**
  - CXR
  - CT (confirmation)
  - Bronchoscopy
- **Treatment:**
  - **Medical:** 1st & 2nd lines of anti-TB.
  - **Surgery indications:**
    - Failure of medical Tx
    - Destroyed lobe or lung
    - Hemorrhage
    - Persistent open cavity with +ve sputum
    - Broncho-pulmonary fistula

Aspergillosis

According to amboss it doesn't usually cause infection in immunocompetent patients.

- **Etiology:**
  - Aspergillus fumigatus
  - Aspergillus niger.
- **Forms:** 8 forms
  - Allergic bronchopulmonary aspergillosis
  - Saprophytic
  - Invasive aspergilloma
  - Chronic pulmonary aspergillosis.
- **Presentation**:
  - Aspergilloma (cavity ball-like on CT)
  - Chronic productive cough
  - Hemoptysis
- **Investigations:**
  - Skin test
  - Sputum
  - biopsy (invasive)
  - CXR
  - CT
- **Treatment**
  - **Medical:** Anti-fungal medications
  - **Surgery:** Lobectomy, Segmentectomy, Pneumonectomy.

★ **Surgery indication:** Significant aspergilloma & hemoptysis

Approach to common Thoracic & Lung Diseases:

Hydatid cyst

- **Etiology:**
  - Echinococcus granulosus
  - Germinal layer gives eggs
- **Life cycle:**
  - Dogs (definitive host)→ sheeps (intermediate host)→ when human eat an undercooked meat the parasite will enter the bowel→ lymphatics→ portal system→ portal veins→ liver→ IVC→ lungs (so if someone has hydatid cyst in the liver we should screen the lung and vice versa)
- **Investigations:**
  - CXR
  - Skin test (Casoni's reaction)
  - CT (Cyst will appear calcified)
- **Treatment:**
  - Surgery + inject hyperosmotic saline + albendazole (needle aspiration is contraindicated)

3- Lung Tumors:

Primary lung carcinoma

- **Etiology:**
  - ★ Smoking
  - Carcinogenic radiation
  - Asbestos and nickel
- **Pathology:**
  - **NSCLC:**
    - Adenocarcinoma (most common type in non-smoker & women)
    - Squamous cell carcinoma (associated with smoking)
    - Large cell carcinoma (associated with smoking)
  - **SCLC:**
    - Systemic dissemination
    - ★ CT-scan is used for staging here.
- ★ **Presentation**:
  - **Local:** Hoarseness, Dysphagia, severe pain in the brachial plexus, Horner syndrome (ptosis, miosis, anhidrosis), pleuritic chest pain, SVC obstruction syndrome
  - **Hormonal:** Hypersecretion (PTH, ADH, ACTH), Hypertrophic pulmonary osteoarthropathy (severe joint and bone pain due to hormonal secretion)
- **Investigation:**
  - CXR
  - ★ CT scan with IV contrast → **GOLDEN STANDARD**
  - MRI (poor for staging)
  - Bronchoscopy
  - Trans-thoracic needle aspiration FNA
- **Staging:** TNM staging (tumor size, lymph nodes, metastasis)
- **Treatment:**

  Depend on stage, cell type, patient physical fitness
  - **NSCLC:**
    - Early stage: surgical (it's possible)
    - Advanced stage: Radio, Chemo
  - **SCLC:** Chemo, Radio (surgery isn't possible)

Secondary lung carcinoma (metastatic)

- **Solitary lung nodule**
- **Types:** Primary lung carcinoma / tuberculosis granuloma / mixed tumor / secondary lung carcinoma / disk pneumonia.

4- Mediastinal Mass Lesions:

★Middle mediastinum

- Bronchogenic cyst
- Pericardial cyst

★Posterior mediastinum

- Neuroendocrine tumors
- Enterogenous cyst

★Anterior mediastinum (5T's):

- **Thymus (thymoma):** (commonest tumor of the anterior mediastinum) Benign or Malignant
  - **Presentation:**
    - Asymptomatic
    - Mass effect
    - Myasthenia gravis (commonest)

- **Classification:**
  - Epithelial
  - Lymphocytic
  - Lymphoepithelial
  - Spindle cell.
- **Stages**: I , II , III , IV
- **Investigations:**
  - All cases (CXR, CT, Biopsy)
  - Selected cases (Bronchoscopy, Esophagoscopy, Angiogram)
- **Treatment:**
  - Benign: complete excision
  - Malignant: complete excision if possible, if not Incomplete resection + Post-operative chemo +/- radio

- **Thyroid (Goiter)**
- **Teratoma**
- **T cell lymphoma**
- **TB lymphadenitis**

4- Trauma:

Road Traffic Accidents

Fractured ribs

- **Presentation:**
  - Hemothorax
  - Lung contusion
  - Surgical emphysema
- **Treatment:**
  - Pain meds for the ribs and <u>don't wrap</u> them.

Flail chest

Fracture in two or more consecutive ribs, causing paradoxical movement of that region of the chest.

- **Presentation:** paradoxical movement of the chest
- **Treatment:** <u>Use wraps</u> or weights to keep the in position to heal. (Needs to be on ventilators)

Hemothorax

- **Treatment:** Chest tube.

Lung contusion & Acute Respiratory Distress Syndrome (ARDS)

- **Treatment:**
  - Supportive (intubation + ventilation + antibiotics)
  - if there is a hemorrhagic shock: surgery and stop the bleeding.
  - I.V fluids : make sure there's no other internal bleeding , conservative amount of fluid/crystalloid (avoid its accumulation outside the lung intravascular structures)

Pneumothorax

- **Traumatic pneumothorax: Open pneumothorax:**
  - **Tension:** Decompression then dressing
  - **No tension:** Occlusive dressing taped from 3 sides.
- **Tension pneumothorax:**
  - Large-bore needle or chest tube followed by thoracostomy
  - If didn't work the patient may die from hemodynamic compromise
- **Spontaneous pneumothorax:**
  - observe, if symptomatic O2 + Chest tube insertion

5- Chest Wall Deformities:

Pectus excavatum (Funnel chest)

- May affect the heart and respiratory system
- Check for other congenital anomalies.

Pectus carinatum (Pigeon chest)

6- Pleural Cavity:

Mesothelioma

- **Presentation:** Shortness of breath
- **Treatment:** Therapy is directed towards controlling the symptoms. Radiotherapy and chemotherapy have no curative value.

Empyema is a collection of pus within the pleural cavity. (Patient present with: chest pain, hemoptysis with cough)

Pleural Effusion is an abnormal collection of fluid in the pleural space.

Esophageal diseases

GERD:

- **Etiology:** Hiatal hernia, Inappropriate relaxation of the LES, Decreased motility.
- **Types of hiatal hernia:**
  - Type 1 (sliding hernia): the most common, occur when the LES rises up to the level of the diaphragm
  - Type 2 (Rolling hiatal hernia) (Paraesophageal hernia): LES position is normal, but the stomach is herniated through the diaphragm.
  - Type 3 (mixed): combination of type 1 & 2
  - Type 4: another organ is herniated into the chest.
- **Clinical presentation:**
  - Classic GERD: Substernal heartburn and/or regurgitation, Postprandial, aggravated by lying down, relieved by antacid.
  - Extra-esophageal (Atypical GERD):
    - Pulmonary (Asthma, Aspiration pneumonia, Chronic bronchitis, Pulmonary fibrosis)
    - ENT (Hoarseness, Dysphonia, Chronic cough, Laryngitis & Pharyngitis, Sinusitis, Globus Sensation)
    - Others (Chest pain, Dental erosion)
  - Complicated GERD: Dysphagia, Odynophagia, Bleeding.
- **Diagnosis:**
  - Barium swallowing, **pH monitoring** (bravo capsule), Endoscopy & Biopsy (to detect malignancy), Esophageal manometry (to measure motility).
- **Treatment:**
  - Non-Pharmacological **(lifestyle modification)**: elevate the head, lose weight, stop smoking, modify the diet.
  - Pharmacological: H2-receptor antagonists, **PPI** (More effective)
  - Surgery: Stretta procedure, Endoscopic plication TIF, Enteryx, Nissen fundoplication (treat GERD and Hiatal hernia)
    - Indication for surgery: Failed Tx, Patient desire, Complication of GERD, atypical symptoms.

Barrett's esophagus:

- Stratified squamous epithelium is replaced by intestinal columnar epithelium with goblet cells in the distal esophagus(metaplasia), in endoscopy (pale-pink is the normal, Red spots Barrett)
- **S&S:** Symptoms of GERD, asthma & recurrent respiratory infection, infection in the head and neck are common complaints.
- **Diagnosis:** Endoscopy & Pathology culture (histology)/ The presence of any endoscopically visible segment of columnar mucosa within the esophagus that on pathology identifies intestinal metaplasia defines BE.
- **Treatment:**
  - Yearly surveillance is recommended in all patients with BE + suppression therapy and monitor the symptoms
  - Photodynamic therapy (most common used)
  - Endoscopic mucosal resection (low-grade dysplasia), Esophageal resection (high-grade dysplasia)

Esophageal perforation:

- Surgical emergency, detection and repair in the 1st 24 hrs results in 80-90% improvement and after 24 hrs survival decrease to 50%. (Tear in the EGJ)
- **Etiology:**
  - After **endoscopic instrumentation**, forceful vomiting (Boerhaave's syndrome), foreign body ingestion, Trauma.
- **Symptoms:**
  - Neck, Substernal & Epigastric pain, Vomiting, hematemesis, Dysphagia, odynophagia and fever
  - Cervical perforation (neck pain and stiffness, SOB, Pain lateralizes to the site of perforation)
  - Thoracic perforation (SOB, retrosternal chest pain lateralizes to the site of perforation)
  - Abdominal perforation (epigastric pain radiates to the back if the perforation is posterior)
- **Signs:** Hemodynamic instability, decreased breath sounds, subcutaneous air in the neck or chest, tender abdomen, patient may present only with tachycardia and tachypnea and low-grade fever with no other signs.
- **Diagnosis:** Barium swallow + CT (you have to place the patient in the lateral decubitus position / A surgical endoscopy if intervention is planned), elevated white blood cell count and an elevated salivary amylase in the blood or pleural fluid.

- **Treatment:** remember always start with IV fluids and broad spectrum Abx, food through central line (TPN)
  - Stable patient ⟶ + Antibiotic for 14 days then do barium swallow if the perforation is there surgery.
  - Unstable patient ⟶ Surgery
    - Surgery's determined by the degree of inflammation surrounding the perforat

Esophageal diseases

Carcinoma of the Esophagus:

- **Types:**
  - Squamous cell carcinoma (70% upper and middle third of the esophagus)
    - Etiology: smoking, alcohol both increase the risk 5-fold Combined, nitrosamines and hot liquid (2° complication of Achalasia) Plummer Vinson syndrome (iron deficiency "koilonychia", esophageal web and dysphagia)
  - Esophageal Adenocarcinoma: High acidity, Barrett's, Dysplasia
- **Clinical features:** Most patients with esophageal cancer present with dysphagia and weight loss + loss of appetite indicates malignancy
- **Diagnosis:**
  - ★ Barium Esophagram (First test in patients presenting with dysphagia although not specific for cancer, shows apple-core appearance)
  - Endoscopy (the best from endoscopic biopsy)
  - CT for chest and abdomen and pelvis (assess the length of tumor) / Positron Emission Tomography (better than CT)
  - Endoscopic Ultrasound (best for staging and treatment)
- **Treatment:**
  - Localized to the mucosa → Surgery
  - Metastasized to lymph nodes → Neoadjuvant chemotherapy then surgery
  - Distant metastasis → Radiotherapy + Chemotherapy

Achalasia:

- The most common type esophagus motility disorders due to the degeneration of the myenteric plexus that innervate LES and esophageal body.
- **Etiology:** Primary: Autoimmune, Viral, Familial. Secondary: cancer, Chagas disease, Post-fundoplication
- **Clinical features:** Dysphagia for solid and liquid (most common), Regurgitation, Heartburn, Chest pain
- **Diagnosis**:
  - CXR, Barium swallow (Diagnostic test, Bird's beak)
  - Endoscopy (Dilated esophagus, Retained food particles)
  - Esophageal manometry (the GOLD standard and Confirmatory test) Aperistalsis/ Hypertensive LES/ Closed LES (fail of relaxation)
- **Treatment:**
  - 1st line - Surgical myotomy (Heller's myotomy)
  - 2nd line - Pneumatic (endoscopic) dilation
  - Can't do invasive treatment → Nitrates, CCB (less side effect) and botulinum toxin injection

Esophageal diverticula:

- most diverticula are a result of a primary motor disturbance or an abnormality of the UES or LES
- **Sites:**
  - 1st **Pharyngoesophageal (Zenker)**. Herniation into Killian's triangle between: 1-Oblique fibers of the thyropharyngeus muscle 2-Horizontal fibers of the cricopharyngeus muscle.
  - 2nd Parabronchial
  - 3rd Epiphrenic
- **Types:**
  - True (all layers of esophagus) results from: External inflammatory mediastinal lymph nodes adhering to the esophagus.
  - False (mucosa & submucosa) results from: elevated intraluminal pressures generated from abnormal motility disorders e.g. Zenker's & epiphrenic diverticulum.
- **Symptoms:** Halitosis (Bad breath), Sticking in the throat, Cough, salivation
- **Investigation:** First exclude mouth and dental disorders, Barium esophagram.
- **Complications:** untreated Zenker's diverticulum → aspiration pneumonia or lung abscess.
- **Treatment:** surgery: Myotomy + Diverticulectomy / endoscopy: endoscopic Dohlman procedure

UTI (disorders, stones, infection):

- **Uncomplicated:** young (<65) women not pregnant healthy pt / no anatomic or neurological GU abnormality
- **Complicated UTI:** Ureteric obstruction (stones, strictures) / Urinary retention / decrease immune system (renal failure, transplant) / Foreign body (catheter or stent) / patient with spinal cord injury / Male or patient > 65 years / pregnant women.
- **Most common UTI causative organisms: KEEPS**
  - **K**lebsiella, Gram –ve bacteria: **E**.coli (most common cause), **E**nterococcus faecalis, **P**seudomonas aeruginosa, Gram +ve bacteria: **S**.saprophyticus (2nd leading cause)
- **Gram –ve Bacteria (Escherichia coli) & Enterococcus faecalis.**
- ★ **Sterile pyuria: TB, stones and cancer**
- **Bacteriuria only: Bacterial colonization without infection**
- **Bacteriuria and pyuria: Infection/inflammation**
- **Routes of UTI's : Ascending Route (most common) ,** Hematogenous , Lymphatic.

- **Urethritis:** Inflammation of the **urethra**.
  - **S&S**: urethral discharge / burning on urination / or asymptomatic (in women)
  - ★ **-Cause: Gonococcal (3-10 days) with purulent white (yellowish) discharge / Nongonococcal (Chlamydia) (1-5 weeks) with scant (greenish) discharge.**
  - **-Investigation**: Urethral swab (confirmatory) / Chlamydia specific ribosomal RNA (Confirmatory)
  - **-Treatment**: 1g Ceftriaxone IM & 1 dose Azithromycin PO

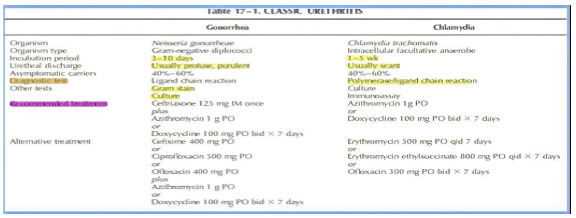


Table 17–1. CLASSIC URETHRITIS

	Gonorrhea	Chlamydia
Organism	*Neisseria gonorrhoeae*	*Chlamydia trachomatis*
Organism type	Gram-negative diplococci	Intracellular facultative anaerobe
Incubation period	3–10 days	1–3 wk
Urethral discharge	Usually profuse, purulent	Usually scant
Asymptomatic carriers	40%–60%	40%–60%
Diagnostic test	Ligand chain reaction	Polymerase/ligand chain reaction
Other tests	Gram stain	Culture
	Culture	Immunoassay
Recommended treatment	Ceftriaxone 125 mg IM once plus Azithromycin 1 g PO or Doxycycline 100 mg PO bid × 7 days	Azithromycin 1g PO or Doxycycline 100 mg PO bid × 7 days
Alternative treatment	Cefixime 400 mg PO or Ciprofloxacin 500 mg PO or Ofloxacin 400 mg PO plus Azithromycin 1 g PO or Doxycycline 100 mg PO bid × 7 days	Erythromycin 500 mg PO qid 7 days or Erythromycin ethylsuccinate 800 mg PO qid × 7 days or Ofloxacin 300 mg PO bid × 7 days

- **Testicular torsion:** Twist of the **spermatic cord**.
  - **S&S** : Acute scrotal pain after minor trauma / Elevated testicle / absence of cremasteric reflex / negative Prehn sign (positive in epididymitis) (in torsion when you elevate the testicle there will be more pain)
  - **Diagnosis** : Surgical exploration / color doppler US (no blood flow)
  - **Treatment** : Surgical detorsion and **bilateral** orchiopexy to the scrotum (has to be done in <6 H to save the testicle)

- **Epididymitis:** Inflammation/Infection of the **epididymis**.

Causative organism: E. coli (Elderly) / STD bacteria: N. gonorrhoeae & C. trachomatis (Young man)

1- Acute: pain and swelling of the epididymis < 6 Weeks

2- Chronic: Long-standing pain, usually no swelling

- **Diagnosis:** U/A (Confirmatory) / Urine culture / Swab if STD is suspected +- Doppler or nuclear study to r/o torsion
- **Treatment:**

2ndry to Bacteriuria wide spectrum Abx for 2 W

2ndry to Sexually transmitted urethritis Tetracycline or Doxycycline for 10 D

★ **Epididymitis → Older, Gradual pain, Fever, signs of inflammation (redness, hotness), US (Increase blood flow)**

★ **Torsion → Younger, Trauma, Sudden pain, High-riding testicle, pain when elevated, no cremasteric reflex, US (no blood flow),** if not treated the testicle dies within 6 H (Medical emergency)

- **Prostatitis:** Inflammation +- infection of the **prostate**.
  - **-S&S**: Dysuria, Frequency, Dysfunctional void , Perineal pain , Painful ejaculation.

Acute bacterial prostatitis

- **S&S**: Fever, chills, N/V, Hot prostate , malaise (urosepsis)
- **Treatment**: Antibiotic and Urinary drainage.
- Urological emergency, could lead to death or abscess.

- **Cystitis:** Inflammation of the lining of the **bladder** / More common in females.
  - ★ **- S&S**: Dysuria, Frequency, Urgency, **pain on percussion above symphysis pubis/Suprapubic /lower abdominal pain** & there is no filled bladder, sometimes microscopic hematuria.
  - **-Diagnosis**: Dipstick / Urinalysis / Urine culture
  - ★ **-Treatment**: Single dose for 3 or 5 days depending on the antibiotic chosen.
    Nitrofurantoin (for women) / Quinolone or Bactrim for 7 Days (for men)

UTI (disorders, stones, infection):

- **Pyelonephritis :** Inflammation of the **kidney** and **renal pelvis**

  -**S&S**: **Chills** / **Fever** / **Flank pain** (Murphy's punch sign) / Dysuria / frequency / N/V

  -**Diagnosis**: Urine culture (+ve 80% , **E.coli**) / Urinalysis (WBC , RBC , Bacteria) / ±↑ serum creatinine / CBC (Leukocytosis, not in cystitis)

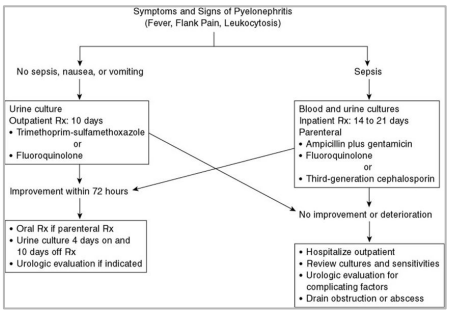
  -**Imaging**: IVP (not used now) / US / CT (to check abscess)

  -**Treatment**:

  Stable pt ⟶ in outpatient clinic / give the patient antibiotics even before the results of the culture.

  e.g. Ciprofloxacin (cover common bacteria)

  Elderly pt / unstable ⟶ admission before developing septic shock , hydration and antibiotics.



- **Urolithiasis (the big boss)**

  - **Risk factors**: Intrinsic (Genetics- cystine stones and RTA-, 20s-40s, Male) Extrinsic (water intake, Diet, Sedentary life, Climate, bariatric surgery/Crohn's disease ⟶ calcium oxalate stones)

  ★-**Types**: 75% Calcium stones (Radiopaque) / Uric acid (meat,Radiolucent) / Cystine (autosomal-recessive defect , Radiolucent) / Struvite (recurrent infection with **Urea-splitting organisms** which are: Proteus, Xanthomonas, Pseudomonas, Klebsiella, Staphylococcus, and Mycoplasma , Radiopaque)

  ★- **S&S** Dysuria / frequency / Hematuria / NV / Tachycardia (anxiety) / Fever

  - **DDx**: Gastroenteritis / Acute appendicitis / colitis / salpingitis.

  There is no guarding and rebound tenderness unlike appendicitis.

  - **Investigation**: Urinalysis microscopic hematuria (90%), **Non-contrast CT-scan** (if not contraindicated, or u know it's stones) then we use US and X-ray (KUB)

  - **Treatment**:

  If the stone is less than 5mm

  A- 1st step is conservative (Hydration, analgesia, Antiemetic, if stones <5mm will pass spontaneously)

  ★B- **Indication for admission:** Renal impairment / only have 1 kidney / Refractory pain / Pyelonephritis / Intractable N/V —> can't take medication

  ★* If patient came to the ER the first thing you do is **CT Without Contrast**

  If > 5mm

  Extracorporeal Shock Wave lithotripsy (SWL)

  Ureteroscopy

  Percutaneous Nephrolithotripsy (PNL)

  Laparoscopic/ Robotic

  Anatrophic nephrolithotomy (open surgery)

- **Lower urinary tract symptoms**

  A- **Storage: Dysuria, Frequency, Nocturia, Urgency, Incontinence**

  B- **Voiding: Hesitancy, Weak stream, Straining, Intermittency, Drippling, Retention**

- **Voiding dysfunction causes**

  A- **Failure to store**:

  Bladder problems (overactivity, hypersensitivity)

  Outlet problems (stress incontinence, sphincter deficiency) /

  combination of both.

  B- **Failure to void:**

  Bladder problems (neurologic, myogenic, idiopathic)

  Outlet problems (BPH, urethral stricture, sphincter dyssynergia) /

  combination of both.

UTI (disorders, stones, infection):

- ## Overactive bladder

⭐**- Symptoms:** Present with frequency, urgency, incontinence and nocturia , Hypersensitive bladder

-Investigation: History (if started days ago suspect infection, if diabetic ask if controlled), Physical Exam, U/A, US (to check the whole tract)

-**Treatment**: Behavioral (don't drink before bed, smoking cessation, reduce coffee and tea), pelvic floor exercise, Anti-cholinergic, Beta-3 agonists.

- ## Benign Prostatic Hyperplasia (BPH)

-Symptoms: LUTS (voiding Symp and storage if the bladder become hyperactive), Poor bladder emptying, Urinary retention, UTI, Hematuria, Renal insufficiency

-Investigation:

1-Physical exam: Digital rectal exam (Tenderness Prostatitis, Hard nodules cancer), Focused, Neurologic exam (anal tone will reflect the bladder tone), Abdomen (distended bladder w/o tenderness comparing to cystitis)

2-Urinalysis & Culture (UTI, Hematuria), **Serum Creatinine** (check urine reflux hydronephrosis), **Serum PSA** (PSA<4 ng/ml), **Flow rate**, **Ultrasound** (Kidney -no hydronephrosis, normal size is 20-25- / Bladder / Prostate), **Post void residual** (PVR)

-Complication: Bladder stones, UTI, bladder decompensation, Incontinence, upper tract deterioration, Hematuria, Acute urinary retention.

-Treatment: For symptomatic only

1- **Medical: alpha-adrenergic blockers** (Tamsulocin, Alfuzocin) / **Androgen suppression** (Finasteride, Dutasteride)

2- **Surgical: Endoscopic, Transurethral resection of the prostate** (TURP, if there's infection or hematuria or stones)**, Laser ablation, Prostatic stents, Open prostatectomy** (If size >100 cc)

- ## Hydrocele

-Scrotal disorder , More common in babies and can happen in adults.

-Types: Communicating & Non-Communicating.

⭐-The distinction between a cyst of the epididymis and a hydrocoele is easy. Epididymal cysts transilluminate brightly and almost always multiple, therefore nodular on palpation and the testes palpated separately from the cyst unlike in hydrocoele the testes palpated within a fluid filled sac and demonstrates transillumination.

-Management: Hydrocelectomy (large), Plication (small)

- ## Varicocele

-More common on the left side (by Left renal vein), Impairs fertility by increase intratesticular temperature.

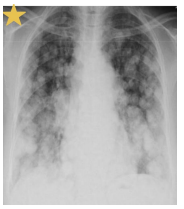
-**Treatment:** Ligation (open, microscopic, lap), Angioembolization

· **Treatment indication**: Infertility w/ abnormal semen, Testicular pain, Impaired testicular volume.

Almost There

Common genitourinary tract malignancy

RCC (Most common type CCRCC)

★ **Definition**	Renal tumor that arises from the PCT.
Risk factors/ Associations	○ **Sporadic**: Risk increased with being a male, PCKD, Smoking ○ **Hereditary**: 1. **VHL syndrome:** Autosomal dominant, mutation in short arm of Chromosome 3 (3p). Screen all family members. 2. **Tuberous sclerosis**: Tuberous sclerosis is caused by mutations in either the TSC1 or TSC2 gene. These genes are involved in regulating cell growth, and the mutations lead to uncontrolled growth and multiple tumours throughout the body.
Signs & Symptoms	○ **Triad:** Hematuria, flank pain and palpable flank mass. ○ In a middle-aged smoker with a left-sided varicocele, think renal cell carcinoma! ○ PNS (Paraneoplastic Syndrome): 1. **Stauffer's syndrome:** non-metastatic hepatic dysfunction characterized by elevated liver enzymes (esp. alkaline phosphatase) and clotting abnormalities. 2. **Budd-Chiari syndrome (Involvement of the IVC):** lower limb edema, ascites, hepatic dysfunction. 3. Hypertension, polycythemia, hypercalcemia. **NOTE:** - Hypercalcemia is the only PNS symptom that can be managed medically, others are managed by nephrectomy - RCC is usually in one side, if bilateral think of VHL and tuberous sclerosis.
Diagnosis	○ **Best initial**: **US** followed by CT-scan with contrast (used for staging as well). ○ **Confirmatory:** Histology on nephrectomy specimen - Polygonal clear cells filled with accumulated lipids and carbohydrate. (Grossly it's golden-yellow due to lipids) ○ To evaluate metastasis: CXR, IVP, CT, LFT, calcium or bone scan (if pt has bone pain) ★ Cannonball metastasis to lung on CXR, CT and MRI multiple white patches (deposits of renal cancer metastasis) that resemble miliary TB ○ ★ Kidney performance status is the best prognostic factor in pt with metastatic RCC. 
Treatment	○ Surgical resection as RCC is chemo-radio resistant ○ **Unilateral:** Radical nephrectomy. ○ **If bilateral:** Partial nephrectomy → if localized and less that 4cm ether in the upper or lower part of the kidney ○ Radiotherapy & chemotherapy (Radio & chemo resistant) Multitarget tyrosine kinase inhibitors TKIs (antiangiogenic) median 12 months progression-free survival benefit. ★ **For metastatic:** Immunotherapy: Infliximab & IL-2 are very effective but bad side effect & IFG second line. (Performance factor is an important prognostic factor)
Stages	○ **Stage I:** tumor < 2.5cm , no nodes, no metastases ○ **Stage II:** tumor > 2.5 cm limited to kidney, no node, no metastases ○ **Stage III:** tumor extend to IVC or main renal vein +ve regional lymph node but < 2cm, no metastases ○ **Stage IV:** distant metastasis or +ve lymph nodes > 2 cm, or tumor extends past Gerota's fascia

Bladder Tumors

Signs & Symptoms	○ Gross PAINLESS hematuria ○ Terminal hematuria (end of voiding) suggests bleeding from bladder		
Types & Risk factors/ Associations	**Transitional Cell Carcinoma (TCC)** **(Urothelial cell carcinomas)** (Most common, 90%)	**Squamous cell carcinoma**	**Adenocarcinoma**
	Pee SAC - **P**henacetin - **S**moking - **A**niline dye and chlorinated hydrocarbons - **C**yclophosphamide	Schistosoma haematobium	Urachal remnant
Diagnosis	○ Urothelial tumors can be multifocal, the entire urinary tract must be evaluated! ○ **GOLD standard:** Cystoscopy with biopsy ○ UA: Micro/Macro hematuria ○ Urine cytology ○ US (If there's obstructive Sx) ○ **CT urography (IVU-CT):** Filling defects ○ KUB If CT is contraindicated ○ What features indicate POOR prognosis? Hydronephrosis, Filling defects, Muscle invasion		
Treatment	○ **CIS:** TURBT, Immunotherapy (BCG), or intravesical chemo, if didn't work do radical cystectomy. ○ **Superficial TCC:** TURBT and need regular cystoscopy follow-up ■ **Low risk** of recurrence: TURBT then (after 24hrs) instill Chemotherapy e.g. mitomycin or gemcitabine. ■ **High risk** of recurrence: TURBT with intravesical BCG or chemotherapy. ■ NOTE: Chemotherapy reduces recurrence only while BCG reduces recurrence and progression. ○ **Invasive TCC:** Radical cystectomy with incontinent urinary diversion (Ileal conduit/Neobladder) for old pt or continent (cutaneous reservoir) in young pts, followed by chemotherapy. ○ **Invasive with metastasis:** Cystectomy and systemic chemotherapy. ○ **For recurrence**: Repeat TURBT and intravesical chemo or BCG.		

Common genitourinary tract malignancy

Prostate tumors

Difference between	**Adenocarcinoma** (Most common)	**BPH**
Location	70% Peripheral zone (Posterior lobe)	Transitional zone (Periurethral)
DRE	Hard with irregular lesions (nodules) and loss of central sulcus	Rubbery , symmetrical , smooth
S&S	Mostly asymptomatic, but may present with back pain due to vertebral metastasis	Obstructive and irritative symptoms
Associated with	BRCA2, Lynch syndrome	

Types

The following is for **adenocarcinoma**: Most of the cases.

Diagnosis

- **Best initial:** PSA (Serum prostate specific antigen). Routine screening after the age of 50, however with family history from the age of 40.
- If more than 4 ng/ml, do Ultrasound-guided transrectal biopsy (**GOLD standard**) to **confirm**.
- PR Diagnosis can be confirmed with locally advanced tumors. **Features: hard nodule or loss of central sulcus.**
- PSA is sensitive but NOT SPECIFIC, it could be high in prostatitis, BPH, prostatic trauma. PSA is also useful for **monitoring treatment.**
- **For staging:** MRI
- **For evaluating metastasis:** CT of abdomen/pelvis and bone scan

Treatment

- If local, >75y/o and low grade watchful waiting.
- If local, <74y/o and low grade active surveillance to catch cancer.
- Active treatment options:
  - >70y/o Radical Radiotherapy (Brachytherapy better than EBRT).
  - <70y/o Radical Prostatectomy (will lead to infertility bc u remove vas deferens as well)
- If metastatic hormonal therapy.
- Bilateral orchiectomy
- Or combination of LHRH agonists (e.g. Goseraline) and Anti-androgens (e.g. Flutamide, Bicalutamide, cyproterone acetate). (Anti-androgens are given b4 LHRH agonists).
- If both didn't work then it's castration resistant prostatic cancer (CRPC) Chemotherapy.

Testicular tumors

Types

- **Seminoma** (Radiosensitive)
- **Non-seminoma** (Chemosensitive)

Risk factors

Cryptorchidism, Klinefelter syndrome and testicular torsion

S&S

- PAINLESS enlargement of testis
- Negative transillumination test

Diagnosis

- Testicular ultrasonography
- CT for staging
- **Tumor markers** are useful for diagnosis and monitoring treatment

Type	Tumor Marker
Germ Cell Tumors (95% of All Testicular Tumors)	
Seminoma (most common testicular tumor)	Usually ⊖, ↑ β-hCG in some cases
Yolk sac (endodermal sinus tumor)	↑ AFP
Choriocarcinoma	↑ β-hCG
Teratoma	AFP and/or β-hCG
Non–Germ Cell Tumors (5% of All Testicular Tumors)	
Leydig cell	↑ Testosterone and estrogen (causing ↓ LH and FSH)
Sertoli cell	None
Testicular lymphoma	None. Arises from metastasis to testes

Treatment

- Radical inguinal orchiectomy (THROUGH INGUINAL (GROIN) INCISION **NOT** THROUGH SCROTUM BC OF THE RISK OF TUMOR SEEDING), then classify if:
  - **Seminoma** Radiotherapy of abdominal and pelvic nodes (dog leg)
  - **Non-seminoma** RPLND ± Chemotherapy (e.g. Bleomycin, Etoposide, cisplatin) (RPLND is associated with ED)
- **Note:**
  - If you have seminoma and non-seminoma, treat as non-seminoma bc it's more aggressive.
  - For stage IIc and above we treat with platinum-based chemotherapy regardless of the type of tumor.

Adjuvant treatment for testicular cancer

Staging according to the classification	Seminoma	Nonseminoma
Stage I	• Standard: active surveillance • Alternatively: chemotherapy (**carboplatin**) or radiation therapy of the regional lymph nodes	• **Active surveillance** or • **RPLND[3] ± cisplatin-based chemotherapy** • Chemotherapy: **BEP[1]**
Stage II	• **Radiation therapy** or • Chemotherapy: **BEP[1] or EP[2]**	• In patients with normal post-orchiectomy tumor markers: **RPLND[3] ± cisplatin-based chemotherapy** • In patients with elevated post-orchiectomy tumor markers: chemotherapy with **BEP[1] or EP[2]**
Stage III	• Chemotherapy (depending on prognosis): **BEP[1] or EP[2]** followed by evaluation of any residual disease that may require resection	

[1]BEP = chemotherapy with bleomycin, etoposide, and cisplatin, [2]EP = chemotherapy with etoposide, cisplatin, [3]RPLND= retroperitoneal lymph node dissection

Common genitourinary tract malignancy

Pheochromocytoma	
Definition	A tumor of chromaffin tissue that secretes catecholamines and is found either in the adrenal medulla or in extra-adrenal sites. Most commonly associated with MEN 2A, 2B, VHL, Neurofibromatosis.
Etiological factors	Cigarette smoking, Schistosoma haematobium.
S&S	○ 80% present with painless hematuria. either gross or microscopic. ○ **5 P's: P**ressure (HTN), **P**ain (headache), **P**erspiration, **P**alpitations, **P**allor.
Diagnosis	○ **Best initial: 24-hour urine metanephrines** and catecholamines or plasma-fractionated metanephrines. ○ CT, MRI or nuclear metaiodobenzylguanidine (MIBG) scan can localize extra-adrenal lesions and metastasis.
Treatment	**Surgical resection** ○ Preoperatively, use α-adrenergic blockade first (phenoxybenzamine) to control hypertension, followed by β-blockade to control tachycardia. ○ Never give β-blockade first, as unopposed α-adrenergic stimulation can lead to severe hypertension.

Emergency in urology

non-TRAUMATIC urological emergencies

1-Hematuria

- The most important symptom that needs immediate medical help.
- **Cause vary according to** :
  - Age
  - Painful or painless.
  - Risk factors for malignancy like smoking
  - Types gross or microscopic.
  - Timing
  - Presence of clot
- **Work up** :
  - History more important than PE
  - Physical examination (rectal examination).
- **Investigation**:
  - CT urography which is the gold standard.
  - You need also to to urine culture, urinalysis, electrolytes, urine cytology and CBC

2-Renal colics

- The most common urological emergency.
- Sudden, severe, colicy pain that radiates from flank down to the groin, scrotum & labia

★ **Investigation**:
  - Helical CT without contrast
  - **KUB** (x-ray) for follow up
  - Don't forget to exclude pregnancy
- **Treatment:**
  - Hydration
  - Pain relievers
  - Alpha blocker "for ureter stones only"
  - Schedule for follow-up 2 weeks later
  - Do surgery if one of the indications is present.

Urinary Retention

3a-Acute

- Painful ***inability*** to void
- Caused mainly by BPH. In children main cause is constipation

★ **Initial management**:
  - give the patient analgesic
  - urethral catheterization.
- **Late management** :
  - Treating the underlying cause.

3b-Chronic

- pain is not a feature and many patient come to ER with renal failure or DM.
- **Symptoms**:
  - Urinary dribbling

★ Overflow incontinence
  - Palpable bladder with no pain.
- **Management: treatment** :
  - Renal support
  - Bladder drainage.
  - Late treatment of the underlying cause.

TRAUMATIC urological emergencies

Testicular torsion

Presentation: (Young male with scrotal pain. Severe, sudden and short duration, referred to the ipsilateral lower quadrant of the abdomen.)

Physical examination:

- testis **is high riding.**
- Acute hydrocele or massive scrotal edema.
- Tender larger than other side.
- **Transverse -not vertical-** orientation
- **Cremasteric reflex is absent in torsion.**
- **Elevation of the scrotum causes more pain**

★ **Dx:** No need to do investigation in such patient because you can take him directly to OR, you can use ultrasound or nuclear medicine, although we don't use nuclear medicine anymore because its time consuming.

Tx: immediate bilateral Orchidopexy (+\-Orchiectomy).

Emergency in urology

TRAUMATIC urological emergencies: (Cont.)

Urethral injury

Presentation:
- Urinary retention or palpable bladder .
- blood at urethral meatus (Not haematuria, only blood without urine).
- Inability to pass urethral catheter (The most common sign).
- high riding prostate (When you per rectal examination you'll find the prostate high in position which will indicate urethral injury).
- hematoma (If you have perineal hematoma you have to investigate for urethral injury).

Dx: Urethrogram.

Tx:
- **Blunt injury:** Suprapubic catheter then definitive treatment (Realignment, urethroplasty).
- **Penetrating injury:** surgical debridement and repair.

Bladder Injury

Presentation: classic **triad** suggestive of a bladder rupture :
- Suprapubic pain and tenderness.
- Difficulty or inability in passing urine.
- With or w/o Haematuria.

Dx:

Hematuria:
- Upper tract imaging > later
- Bladder imaging > cystogram

Tx:
- **Intraperitoneal bladder rupture:** open them and repair.
- **Extraperitoneal bladder rupture:** keeping the catheter and repeat the study in 1-2 weeks and it'll heal by itself and give him antibiotics to prevent infection.
  -Open repair + (If not healed after 2 or 3 weeks).

Renal Injury

Presentation: trauma to the chest, abdomen and pelvis in a patient with tenderness, Pain, palpable mass or ecchymosis in their flank, abdomen or back. Also hematuria but is not always present.

Dx:

★ **Triphasic CT scan (urography)** **(CT with contrast and delayed images)** **, IVU (Intravenous urogram), Renal Ultrasound**

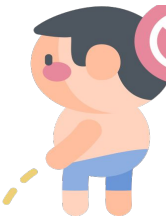
- **Indication for renal imaging:**
  - Macroscopic haematuria.
  - Penetrating chest, flank, and abdominal wounds.
  - Microscopic [>5 red blood cells (RBCs) per high powered field] or dipstick.
  - **Hypotensive patient** (SBP <90mmHg).
  - A history of a rapid acceleration or deceleration.
  - Any child with microscopic or dipstick haematuria who has sustained trauma for children no need to have hypotension to do imaging.

Tx:
- The grades don't correlate with the management, in patients with renal injury management depend on Clinical scenario.
- 438: Grade I, II, III, and even IV treated conservatively (As long as the patient BP is stable), While Grade V treated surgically right away.

- **Conservative management:**
  - Wide Bore IV line.
  - Bed rest.
  - Vital signs monitoring.
  - serial CBC (HCT)
  - F/up US &/or CT.

- **Surgical exploration**
  - Persistent bleeding (persistent tachycardia and/or hypotension failing to respond to appropriate fluid and blood replacement.
  - If the patient is already open because of unrelated injury like liver injury, we do the kidneys exploration.
  - Expanding peri-renal hematoma (again the patient will show signs of continued bleeding).
  - Pulsatile peri-renal hematoma.

Genitourinary anomalies



U Must Void Properly
Unless you Have an anomaly

1-UPJO:

- **Definition:**
  - Obstruction of UPJ
- **Causes:**
  - Narrowing of the ureter either : intrinsic (due to partial obstruction) or extrinsic (due to the crossing of vessels)
- **S&S:**
  - Usually detected on antenatal US or incidentally later on:
  - UTI, flank pain, mass, hematuria and stones in the renal pelvis
- **Diagnosis:**
  - **Best initial: US** shows only the hydronephrosis (Isolated hydronephrosis (**Mickey mouse sign**) No hydroureter.
  - **Next step: MCUG** (to exclude VUR since it's a more common cause of hydronephrosis)
  - **GOLD standard: Dynamic renogram** for conformation and to rule out obstruction
- **Treatment:**
  - Mainly observation as 80% resolve by themselves
  - **If any of the following is present, do dismembered pyeloplasty**:
    - Worsening hydronephrosis (Increase in grade)
    - Renal function:
      - Less than 40% in one kidney
      - >10% deterioration
    - Stones formation
    - Pyelonephritis
- **Imp note:** UPJO is the main ddx of antenatal hydronephrosis

2-Ureterovesical junction obstruction (UVJO), Megaureters:

- **Definition:**
  - Obstruction of UVJ
- **Everything is the same as UPJO except** that we will find **Hydroureteronephrosis "mega ureters"** (Both renal pelvis and ureter); **unlike UPJO** in which there's **isolated hydronephrosis** (Only renal pelvis)
- **Treatment:**
  - The same as UPJO, we start with observation and wait for spontaneous resolution unless surgery is indicated (same as UPJO indications) then we do **Ureteral reimplantation**

3-Multiple cystic dysplastic kidney (MCDK):

- **Definition:**
  - Multiple fluid filled cysts replacing normal renal parenchyma with non-functioning kidneys
- **S&S:**
  - Usually detected on antenatal US or incidentally later on:
    - **Unorganized** urine accumulation in kidney (black fluid) unlike UPJO in which it's organized. (**NO** MICKEY MOUSE SIGN)
- **Diagnosis:**
  - **Best initial: US**
  - ***GOLD standard* DMSA**
    - Used if you can't differentiate between UPJO and MCDK on US
  - **MCUG/VCUG:** Used if pt is symptomatic to detect contralateral VUR
- **Treatment:**
  - Observation until cysts disappear
  - **Go for nephrectomy (Open laparoscopic or robotic) if any of the following is present:**
    - Hypertension
    - Pain
    - Pyelonephritis

Genitourinary anomalies

4-VUR:

- **Definition:**
  - Retrograde projection of urine from the bladder to the ureters and kidneys
- **Causes:**
  - Posterior urethral valves, neurogenic bladder, urethral/meatal stenosis.
- **Grades:**
  - **Mild (1-2):** No ureteral or renal pelvic dilation. Often resolves spontaneously
  - **Moderate to severe (3-5):** Ureteral dilation with associated caliceal blunting in severe cases.
- **S&S:**
  - Patients present with recurrent febrile UTIs, typically in childhood. Prenatal ultrasonography may identify hydronephrosis and/or oligohydramnios.
- **Diagnosis: If u suspect VUR, treat UTI with abx first THEN do investigations**
  - **Best initial: US**
  - ★ **GOLD standard: and for classification: MCUG/VCUG**
- **Treatment:**
  - Initial: Give prophylactic abx and wait for spontaneous resolution
  - **Go for surgery (Ureteral implantation (higher success rate) or endoscopic treatment) if:**
    - Recurrent pyelonephritis despite abx prophylaxis
    - Non-compliant with medical treatment
    - Persistence of reflux (High grade 3-5)

5-Posterior urethral valve (PUV):

- **Definition:**
  - Presence of a valve in the posterior of urethra leading to urinary retention, VUR (40%), Bilateral renal dilation and eventually renal impairment (50%) or ESRD (30%).
- **S&S:**
  - Occurs in MALES ONLY. Most common cause of urine retention in males.
  - Most cases are diagnosed antenatally by US
  - Later on, may present with Urinary retention (distended, palpable bladder and low urine output), UTI, urinary incontinence.
- **Diagnosis:**
  - **Best initial: US**
    - Bilateral hydroureteronephrosis and Oligohydramnios
    - Keyhole sign and thick-walled bladder
  - ★ **GOLD standard: MCUG**
    - Trabeculation and sacculation of the bladder (Christmas tree sign)
    - Dilation of posterior urethra with normal anterior urethra
- **Management:**
  - **Stabilize the pt first and confirm:**
    - **Insert feeding tube then give abx prophylaxis**
    - Do US then MCUG then RFT
  - **After this you have go for surgery; you have 2 options:**
    - **Endoscopic valve ablation or incision:** Classical treatment
    - **Cutaneous vesicostomy:** This is done if pt has low birth weight, premature delivery or renal failure as a temporary solution THEN once the pt reaches the age of 1 we do Endoscopic valve ablation
- **Complications:**
  - Can lead to pulmonary hypoplasia and respiratory distress caused by oligohydramnios.

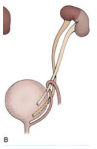
6-Ureterocele:

- **Definition:**
  - Cystic dilation of the distal aspect of ureter.
  - Either within the bladder
  - or spanning the bladder neck and urethra
- **S&S:**
  - Usually diagnosed by antenatal US
  - Later on, may present with UTI, stones and urinary retention (Hydroureteronephrosis)
- **Diagnosis:**
  - **Best initial: US**
  - **GOLD standard: MCUG/VCUG**
    - Shows filling defects
- **Treatment:**
  - - No obstruction ... no treatment
  - - Ipsilateral obstruction ... postnatal emergency treated by puncture
  - By cystoscopy (Endoscopic incision of ureterocele)

Genitourinary anomalies

7-Ureteral duplication:

- **Definition:**
  - 2 ureters unilaterally (85%), could be incomplete (One opening into bladder) or complete (2 separate openings into bladder in one side)
- **S&S:**
  - More common in females. Associated with reflux (in lower ureter) 43% or renal dilation 29% or Ectopic insertion 3% or ureterocele (in upper ureter).
- **Weigert-Meyer law:(Complete duplications)**
  - **The upper moiety** (Obstructed ureter): Will drain inferiorly and medially to the orifice of the **lower moiety** (Refluxing ureter)
- **Treatment:**
  - Treatment is indicated only in case of associated complications

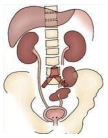
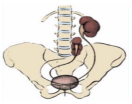
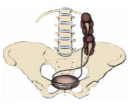


8-Horseshoe kidney:

- **Definition:**
  - Two kidneys connected to each other (90% lower pole or 10% upper pole) each kidney drained by its own ureter with calyces being normal in number, atypical in orientation and pelvis remains in the vertical or obliquely lateral plane.
  - The isthmus is bulky and consists of parenchymatous tissue Trapped under inferior mesenteric artery and remain low in the abdomen.
- **S&S:**
  - 60% are asymptomatic. But may present with symptoms secondary to UPJO (1/3 of cases) due to acute angle of the ureter eventually leads to Hydronephrosis
  - Higher incidence in chromosomal aneuploidy (eg, Turner syndrome*(also associated with malrotation), trisomies 13, 18, **21**).
- **Diagnosis:**
  - US or CT
- **Treatment:**
  - Nothing, we only treat the associated conditions
  -

9-Renal ectopia:

- **Definition:**
  - Kidney (L>R) doesn't fully ascend, could be arrested in the pelvis or lumbar area.
- **Types:**

Simple renal ectopia	Crossed renal ectopia
• Kidney location is abnormal in vertical axis but normal in horizontal axis • Left is more common than right • Usually asymptotic 	• Kidney location is abnormal in both vertical and horizontal axis (Located in the opposite side) • Could be fused (90%) or without fusion.  Without fusion  With fusion

  - In both types the ureters will drain in the normal side (Orthotopic).
  - Even though in crossed ectopia, the kidney will be in the other side the ureter will drain in normal side
  -
- **S&S:**
  - Mostly asymptomatic unless associated with other anomalies (15%):
    - Hydronephrosis (50%): due to UPJO, UVJO, VUR (Grade 3 or greater) or malrotation
- **Treatment:**
  - Nothing, we only treat the associated conditions

Genitourinary anomalies

10-Unilateral renal agenesis:

- **Definition:**
  - Absent kidney (L>R) and in 50% of cases ipsilateral ureter will be absent as well. Adrenal gland will be normal.
- **S&S:**
  - More common in males. Mostly asymptomatic.
  - Associated with other systems anomalies (CVS, GIT and MSC) and Müllerian ducts abnormalities (F>M)
  - found by Prenatal US or incidentally on US/CT of abdomen
  - Radiological findings: Absent kidney with the contralateral kidney being hypertrophied (Compensatory hypertrophy bc of high workload). The left side is absent more frequently.
- **Diagnosis:**
  - **Best initial:** US and CT
  - **Confirmed(GOLD standard):** with DMSA
    - You can't say "kidney is absent" using US, u need DMSA.
- **Treatment:**
  - Nothing, only treat associated conditions
  - Require follow-up annually: UA, BP measurements and US

11-Bilateral renal agenesis:

- **Definition:**
  o Both kidneys and ureters are absent. Bladder is either absent or hypoplastic. Mullerian duct anomalies are seen. BUT normal adrenal gland.
- **S&S:**
  o Potter's syndrome
  o Oligohydramnios; After 20wks, amniotic fluid is formed of urine. So, if there's no kidney —>No amniotic fluid
  o If there is no amniotic fluid, the uterus will compress on the fetus and there will be no space for development of kidney, the most imp. organ that will be affected are the lungs causing **severe pulmonary hypoplasia**
- **Prognosis:**
  o 40% stillborn (dead); They do not survive beyond 48 hours due to respiratory distress associated with pulmonary hypoplasia.

12-Ectopic ureter:

- **Definition:**
  o Ureter single or duplex, that drains anywhere other than the trigonal area of bladder.
  o In **females** it usually drains anywhere from the bladder neck to the perineum and into the vagina, uterus and even rectum in which there is no sphincter distal to it
    - **Continuous wetting (Especially when it opens into vagina)**
  o In **males** the ectopic ureter always enters the urogenital system above the external sphincter or pelvic floor, and usually into the wolffian structures including vas deferens, seminal vesicles, or ejaculatory duct. In which the ureter opens in an area with a sphincter near it
    - **NO wetting or dribbling**
- **S&S:**
  o Females: Continuous wetting
  o Males: Recurrent epididymitis, orchitis, infection of the seminal vesicles
- **Treatment:**
  o If kidney Is functioning normally: Reimplantation
  o If kidney isn't functioning: Nephrectomy

13-Supernumerary kidney:

- **Definition:**
  o Definitive **accessory** organ with its own collecting system, blood supply, and distinct encapsulated parenchyma.
- **Diagnosis:**
  o **Best initial: US**
  o **GOLD standard: CT**

14-Hypospadias:

- **Definition**
  o Abnormal position of EUM on the **ventral** surface. If associated with undescended testes > investigate for gender through chromosomal analysis , might be female with hormonal abnormalities , this is called disorder of sexual differentiation
- **Types:**
  o Distal (Anterior): Less curvature of the penis
  o Proximal (Penoscrotal): More curvature of the penis
- **Treatment:**
  o Surgical repair after 6-9 months (Don't do it before 6 months)
  o **DO NOT DO CIRCUMCISION**; we need the extra skin for reconstruction of meatus later

Genitourinary anomalies

15-Epispadias:

- **Definition:**
  - Abnormal position of EUM (above), on the dorsal side.
- **Treatment:**
  - Reconstruction surgery at the age of 1yr

16-Prune-Belly /Triad/Eagle-Barret/Abdominal musculation syndrome:

- Look for **MUG** triad:
  - **Musculoskeletal system:** Absent/hypoplastic Ant. Abdominal muscles
  - **Urinary tract:** Bilateral hydroureteronephrosis and enlarged bladder
  - **Genital tract:** Bilateral intra-abdominal testes (Empty scrotum)
- Note: Pt only have skin and fascia, you can easily palpate the kidneys, see bowel movement, see the edge of the liver and palpate the spleen.

17-Exstrophy (EMERGENCY):

- **Definition:**
  - Deficient skin, subcutaneous tissue and abdominal wall.
- **Types:**

Bladder exstrophy	Cloacal exstrophy
• Bladder is out, you can see it. • Males usually present with "Bladder exstrophy-epispadias complex" • Has to be fixed by surgery after birth. Usually done during the first week of life.	• More severe form of bladder exstrophy, here you can see the bowel AND the bladder • They have poor quality of life, usually terminate it during pregnancy. • Associated with Omphalocele, GI anomalies (malrotation, duplication, duodenal atresia, Meckel diverticulum) and GU anomalies (Separate bladder halves, bifid genetalia)

18-Urachal abnormalities:

- **Definition:**
  - The urachus (the intrauterine connection between bladder and umbilical cord), remains after birth.
- **S&S:**
  - Usually detected postnatally due to continuous umbilical discharge (Wetness near the belly button)
- **Diagnosis:**
  - US, CT and MCUG/VCUG
    - Continuation between the bladder and umbilicus
- **Treatment:**
  - **Asymptomatic**: Observation, wait for resolution
  - **Didn't resolve:** Surgical excision(partial cystectomy) Why? Bc it increases the risk of adenocarcinoma
  - **Infected urachal remnant:** Drain + Abx then surgical excision

19-Bladder Diverticulum:

- **Definition:**
  - Outpouching in bladder.(Expansion of the mucosa outside of the bladder)
- **Types:**
  - **Primary (Congenital)**: Localized herniation of bladder mucosa at the ureteral hiatus. Due to deficient bladder wall.
  - **Secondary para-ureteral (Acquired)**: Multiple sacculation and diverticulum due to existing infravesical obstruction
- **S&S:**
  - Asymptomatic but they can present with recurrent UTIs and stones
  - Post void urinary residual volume (Urinary retention)
- **Diagnosis:**
  - Usually detected by prenatal US
  - **GOLD standard: VCUG/MCUG** which will reveal possible accompanying VUR
- **Treatment:**
  - **Symptomatic (Especially with VUR):** Surgery (Bladder diverticulectomy)
  - **Asymptomatic**: observation

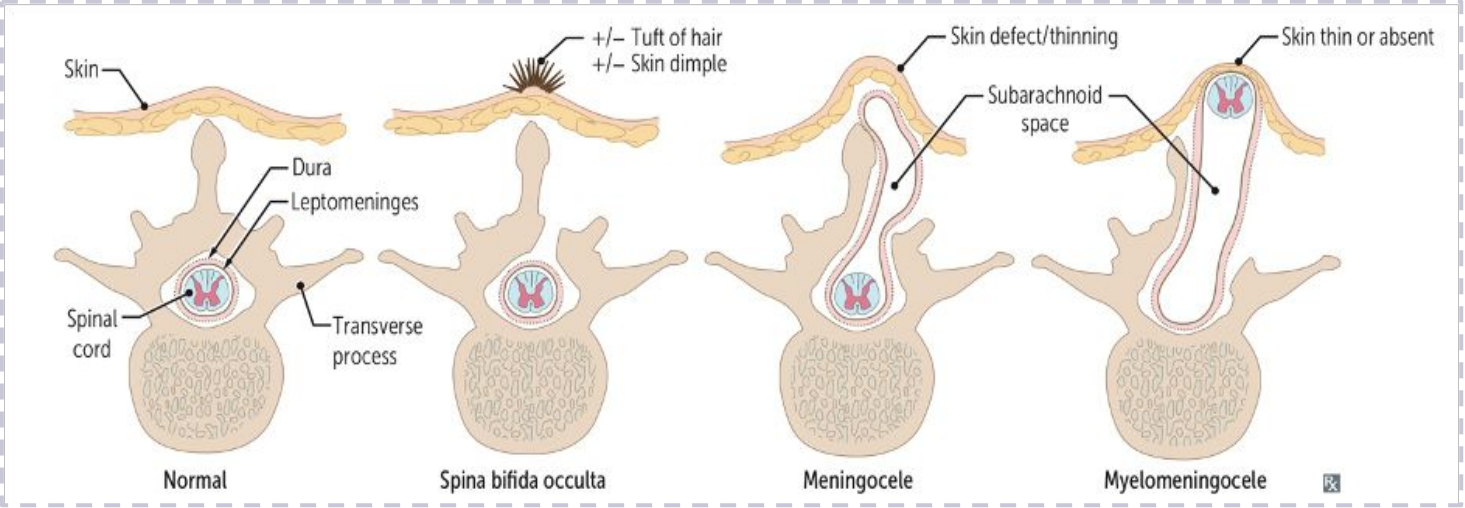
Genitourinary anomalies

20-Bladder duplication:

- **Definition:**
  - Two bladder, two urethra, two external urethral meatus. Bladder duplication is often associated with duplication anomalies of the external genitalia & lower GIT
- **Diagnosis:**
  - **GOLD standard:** MCUG
- **Treatment:**
  - Initial: Renal preservation and prevention of infx then surgery
  - Requires major surgery where we connect/reconstruct the the internal and external genitalia (Type of surgery depends on individual)

21-Neurospinal dysraphism:

- **Definition:**
  - Neuropores fail to fuse (4th week)—>persistent connection between amniotic cavity and spinal canal. Associated with maternal diabetes and folate deficiency.
  - High alpha-fetoprotein (AFP) in amniotic fluid and maternal serum (except spina bifida occulta = normal AFP).
  - Acetylcholinesterase (AChE) in amniotic fluid is a helpful confirmatory test
- **Types:**
  - **Spina bifida occulta:** Failure of caudal neuropore to close, but no herniation. Usually seen at lower vertebral levels. Dura is intact. Associated with tuft of hair or skin dimple at level of bony defect. Commonest cause of paraplegia (50%).
  - **Meningocele:** Meninges (but no neural tissue) herniate through bony defect.
  - **Myelomeningocele**: Meninges and neural tissue (eg, cauda equina) herniate through bony defect.
  - **Myeloschisis**: Also called rachischisis. Exposed, unfused neural tissue without skin/meningeal covering.
  - **Anencephaly**: Failure of rostral neuropore to close no forebrain, open calvarium. Clinical findings: polyhydramnios (no swallowing center in brain).
- **S&S:**
  - Most common cause of neurogenic bladder dysfunction in children.
  - Cutaneous lesions occur in 90% of children with various occult dysraphic states:
    - Small lipomeningocele
    - Hair patch
    - Dermal vascular malformation
    - Sacral dimples
    - Abnormal gluteal cleft
  - Commonest site is L5-S1
- **Treatment:**
  - Diagnosed immediately after birth and require surgery



Arterial diseases

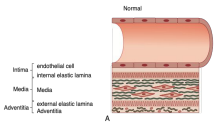
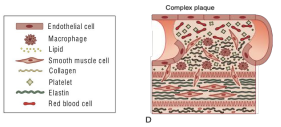
Types of arteries

Elastic Arteries	Muscular Arteries	Small Arteries
Aorta & beginning of its large branches have predominance of elastic fibers in Media.	Medium sized arteries, distributing arteries exhibit smooth muscles in their walls	Major site of autonomic regulation of blood flow.

Atherosclerotic risk factors

Non-modifiable	Modifiable	Other risk factors
• Age • Male gender • Menopause • Familial predisposition • Genetic	★ **Smoking** • HTN • DM • Dyslipidemia	• Sedentary lifestyle • Obesity • High carbohydrate intake • Stressful & competitive lifestyle • Type A personality ★ **Elevated homocysteine** (specific risk factor)

Pathophysiology of atherosclerosis

Endothelial Injury	- Chemical injury (Smoking, Hypercholesterolemia) - Physical injury - Atheroma (a reversible accumulation of degenerative tissue in the intima of the arterial wall) ➢ Hypertension increases this stress "lead to propagation of plaque to larger area"	
Fatty streak	- Increased permeability to lipids and inflammatory cells to leak into sub-endothelial area. - Leukocytes adhere into the subendothelial space and digest lipids to become **foam cells.** - Protease and free radicals liberated. - Cytokines attract more leukocytes and smooth muscle cells.	
Simple Plaque	- Smooth muscle cells exit the media. - Proliferate, take on the **characteristics of fibroblasts** and **produce collagen**, raising the atheroma.	
Complex Plaque	- Proliferation forms an endothelial cap, which may rupture, ensuing further endothelial Injury. - This results in thrombosis and distal embolization.	

Peripheral arterial disease (PAD)

Acute limb ischemia

- Emergency PADs
  - **Sudden** occlusion in the absence of adequate collaterals

Causes	- **Thrombosis or Atheroembolism** (plaque rupture, hypovolemia, ^ blood coagulability, Pump failure) - **Embolism or Thromboembolism** (70% caused by AFib) - **Injury**
Mechanism of injury	Pain on rest, emergency Thrombosis caused by ruptured plaque Embolism mainly caused by AFib / sites (aortoiliac, femoral, popliteal bifurcation)(most common site in the common femoral artery bifurcation)
S&S	6P (**P**aralysis, **P**aresthesia, **P**ain, **P**allor, **P**oikilothermia, **P**ulselessness)
Stages	A. **ALI early stage (non-fixed mottling):** you can fix it with surgery B. **ALI late stage (fixed mottling):** amputation
Management	Blood work, ECG, crossmatch IV heparin 5000 - 8000 IU if there's no contraindication **If ischemia is complete** (Embolectomy) / **If ischemia is incomplete** (do the rest of the work-up) A. Acute embolism: Sudden onset, severe ischemia, **normal pulses in the contralateral leg**, paraplegia sometimes Embolectomy under LA or GA IV heparin post-operatively B. Thrombosis in Situ: Pump failure, Hypovolemia, Hypercoagulable state previous claudication, no source of emboli, **lack of pulses in the contralateral leg**- Medically, limb remains threatened? Thrombectomy or Endoluminal techniques or Thrombolysis or Bypass.

Arterial diseases

Peripheral arterial disease (PAD)

Chronic PAD

Pathophysiology		Slow gradual luminal stenosis secondary to plaque collateral development compensate, Exertional symptoms appear first lead to IC & CLI & DF - 1 Area of stenosis Asymptomatic - Multiple Claudication - More Peripheral ischemia
Mechanism of injury in atherosclerotic disease		In chronic pain occur after walking 300m-1km A. Critical stenosis compensated by collaterals vessels / Symptoms on exercise. B. Acute thrombosis of critical stenosis / Little change in symptoms. C. Acute thrombosis of non-critical stenosis / Severe symptoms D. Atheroembolism form ruptured plaque/ cholesterol emboli E. Thromboembolism/ Afib most common cause. Severe ischemia because the lack of collaterals
★ **Intermittent Claudication (IC)**	**Clinical features**	Muscular pain, present with walking (Pain occur at the same distance every time), Relieved by resting, Impotence (leriche syndrome) , Skin change, toenail change, muscle wasting.
	Claudication site	Pain occur distal to the occlusion 1. Bilateral thigh and/or Buttock: Aortoiliac arteries 2. Unilateral thigh and/or Buttock: Iliac arteries (Buttock → Common iliac / Thigh → External iliac) 3. Calf: Femoropopliteal arteries 4. Foot: Tibial arteries
	Investigation	- **CBC** - **Electrolytes** - **Creatinine** - **Coagulation profile** (aPTT, INR) - **Type & screen** - **Lipid profile** & **Hbg A1C** (modifiable RF) - **Chest x-ray** - **ECG**, **Echo** (assess the coronary artery) → **ABI** (0.8-0.4 / Normal > 0.9) → **Toe pressure** (<50 mmHg) (Normal toe pressure = 70-110 mmHg) - **Segmental pressure=** 20 mmHg reduction - **Volume Plethysmography=** Measures the arterial volume changes → **Duplex US** (single occlusion or stenosis) - **CTA** - **MRA** - **Invasive vascular investigation**
Critical Limb Ischemia (CLI)	**Clinical features**	- **Rest pain**, **exacerbated by lying down** (because the pt relies on gravity to deliver blood) - **Relieved by hanging the feet during sleep** - **Tissue loss (Ulceration & gangrene)**
	Signs	- Positive Buerger's test - Skin is thin and dry - Veins are superficial and upon minimal elevation (venous guttering) - Examine all the pulses - **Arterial ulcers** (Pale and necrotic floor, on toes or foot, irregular margin, painful, pinkish surrounding)
	Investigation	- **CBC** - **Electrolytes** - **Creatinine** - **Coagulation profile** (aPTT, INR) - **Type & screen** - **Lipid profile** & **Hbg A1C** (modifiable RF) - **Chest x-ray** - **ECG**, **Echo** (assess the coronary artery) → **ABI** (<0.5/ Normal > 0.9) (first line) → **Toe pressure** (<30 mmHg) - **Segmental pressure** - **Volume Plethysmography** → **Duplex US** (Multiple occlusion or stenosis) - **CTA** - **MRA** - **Invasive vascular investigation**
Management of IC & CLI	**Primary intervention**	Modifiable risk factors-lifestyle change (weight & being active)
	Secondary intervention (BMT)	★ **Smoking cessation** - **Control HTN** - **Control diabetes** (HbA1c < 7%) - **Statin** - **Antiplatelet agent** - **Exercise** (Improve collaterals / the muscle use less O2) - **Reduce weight**
	Endovascular & Surgical intervention	- **Balloon-Angioplasty** (conventional or drug coated balloons with Paclitaxel) For short concentric stenosis. - **Endarterectomy** (direct removal, done in the carotid and femoral bifurcation, will be closed by **patch angioplasty**) - **Bypass grafting** 1. *Autogenous material* (commonly in veins, flipping or removing the valves) 2. *Prosthetic material* (ePTFE) 3. *Extra-anatomical bypass* (for those with limited life expectancy) **Femoro**-femoral crossover for **unilateral** occluded iliac, **Axillobi**femoral if **both** iliac arteries were occluded
	Indications	- **For Endovascular & Surgical intervention:** Disabling claudication pain / CLI always due to tissue loss - **For Bypass grafting:** High-flow and high-pressure **(Inflow)** / Suitable **conduit** / **Good Outflow or run-off**
Diabitic foot (DF)	Combination of **ischemia, neuropathy, immunocompromised patient.**	
	Clinical features	- **Sensory neuropathy** (pt doesn't feel pain, proprioception is affected, abnormal walking lead to Charcot's Foot), - **Autonomic neuropathy** (Dry foot, abnormal flow in the bones may lead to osteopenia and bony collapse), - **Motor neuropathy** (The flexor are affected more which lead to dorsiflexion of the foot leading to pressure on the metatarsal heads and causing ulceration)
	Prevention	Diabetic control (Hb A1c<7%), washing the foot with soup daily and dry it twice every time, don't walk barefoot, use clean cotton socks, daily inspection.
	Treatment	- **If the blood supply is adequate** excise dead tissue, control infection, protect the foot from pressure - **If there is ischemia** revascularize the foot if possible. - Many patients present late, with extensive tissue loss and **unreconstructable disease** accounting for the very high amputation rate

Arterial diseases

Post Ischemic Syndromes

★ **Compartment injury (Local)**	Pressure in the muscle is higher than the capillary (>25 mmHg) Rapid progression of symptoms (Pain, worse with passive stretching or extension of the muscle, swollen muscles) Prevention through expeditious revascularization low threshold for **fasciotomy** to relieve the pressure. (**Medial incision**= posterior compartment "superficial & deep") (**Lateral incision**= anterior and lateral compartment)
Reperfusion injury (Systemic)	**Caused by** activated neutrophils, free radicals, H, CO2, K, myoglobin released from reperfused tissue. **Leads to:** ARDS, Myocardial stunning, Endotoxemia, ATN, Multiple organ failure **Management:** Hydrate + Calcium (protect the heart) + Produce hypokalemia before the reperfusion + produce alkalosis / you may use Inotropes

Cerebrovascular Disease (CVD)

Transient ischaemic attack (TIA)	**Stroke**	**Amaurosis fugax**
• Symptoms last for **less than 24 hours** • E.g: "Patients had a slurred speech for half a day, then it got back to normal."	• An episode of **focal** neurological dysfunction lasting **> 24 hours**, of vascular etiology	• **Transient incomplete -sometimes complete- unilateral loss of vision,** never synchronously bilateral • A veil or curtain coming across the eye • E.g: "patient feel like there is a curtain closing in front of his eyes"

Clinical assessment

History (assess RF), Neurological exam (Exclude other causes), Carotid bruit are not reliable

Pathophysiology

A. 80% of strokes are ischemic / about half are thought to be Atheroembolism from carotid artery
B. Origin of the internal carotid artery is the most common site of atheroma comparing to the middle internal carotid & MCA
C. Stroke in the right side for a left-handed person will result in: dysphasia (inability to speak properly), ipsilateral loss of vision, contralateral weakness
D. Emboli in the **ophthalmic** artery may cause amaurosis fugax or permanent monocular blindness in ipsilateral side
E. MCA contralateral hemiparesis and hemisensory loss

Investigation

- **Doppler** (duplex) **ultrasound**
- **MRA**
- **CTA**
- **Angiography** (RF for TIA & stroke because its an invasive procedure)

Management

A. **BMT:** Asymptomatic patient

B. **Carotid Endarterectomy (CEA) + BMT**
Indication: ICA stenosis >50% / Life expectancy of at least 2 years / Undertaken with stroke and/or death rate <5% / The procedure can be done soon
Contraindication: pt with major stroke and little in the way of the recovery / occluded internal carotid.

C. **Carotid artery stenting (CAS)**
Benefits: Avoid neck and cranial nerve injury / reduces the risk of MI
Indication: CEA is not possible or desired because of anatomical or clinical factors.

Venous Diseases:

Valves:

- **Vertical valves**: prevent blood from going down **(flow direction:** down to up/distal to proximal**)**
- **Perforating veins valve:** directing blood from superficial to deep veins **(flow direction:** out to in**)**

Ambulatory Venous Pressure

1- Upon walking: it's around 25 mmHg and when a patient has **venous insufficiency** it will be higher than 25 mmHg. ★
2- High walking ambulatory venous pressure will develop chronic venous insufficiency

Discuss the pathophysiology and epidemiology of varicose veins.

Most venous disease arises as a result of incompetent valves due to:

- **Congenital**: Avalvular venous system (rare)
- ★ **Acquired (secondary causes)**: **obesity**, **pregnancy**, CHF, ascites, and prolonged frequent standing, tumor, surgery, radiation, **DVT.**
- ★ **Primary causes: family history,** female and age.

Discuss the classification and clinical features of varicose veins.

The majority are asymptomatic and seek treatment for cosmetic purposes, only portion develop complications of chronic venous insufficiency:

- **Trunk varices:** involve major tributaries of the GSV and LSV, usually ≥ 4mm
- **Reticular varices:** lie deep in the dermis, dark blue and ≤4mm
- **Telangiectasia:** superficially in the dermis, purple or bright red and 1mm or less

Discuss the diagnosis and managements of varicose veins

Diagnosis:

- Varicose diagnosed usually by **physical examination**
- **Duplex**: patient considered for intervention (best non-invasive diagnostic test)
- **MRI**: for severe varicose to assess the extent of the lesion and the arterial component

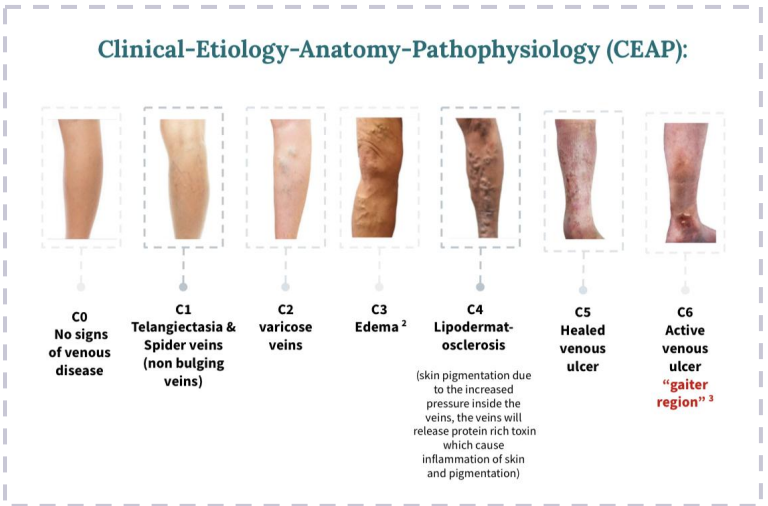
Management:

- **Uncomplicated**: conservative
- **Surgery**: removing the varicose
- **Endovenous**: replaces surgery. Radiofrequency ablation, Endovenous LASER ablation and sclerotherapy

Discuss the pathophysiology and clinical assessment of chronic venous insufficiency

- Manifestations of impaired venous return mainly due to failure of valves that leads to continued reflux of blood.
- 90% venous reflux & 10% venous obstruction
- ★ In someone with defect valves the ambulatory venous pressure won't fall during walking
- Venous leg ulcers are classically located in the "**gaiter**" region of the leg.
    - **Why?** Because the longest vein in the superficial venous system (GSV) starts at the medial malleolus. Once venous system failure occur, it will be the highest point of pressure in the venous system.
    - Lead to pain while standing and edema, protein rich fluid, lipodermatosclerosis and skin pigmentation and finally ulcers.

Clinical assessment: (History and physical examination):



Venous Diseases:

Investigation:

- **Doppler**: Tells us that there is fluid & it's moving. You can also asses valve closure
- ★ **Duplex**: evaluates the velocity and direction of blood flow in the vessel. the most commonly used and best non-invasive.
- **AVP**: ambulatory venous pressure (invasive):

Normal: >50% drop in pressure and ≥ 20 sec Venous Refill Time. **Abnormal**: <50% drop in pressure and <20 sec Venous Refill Time

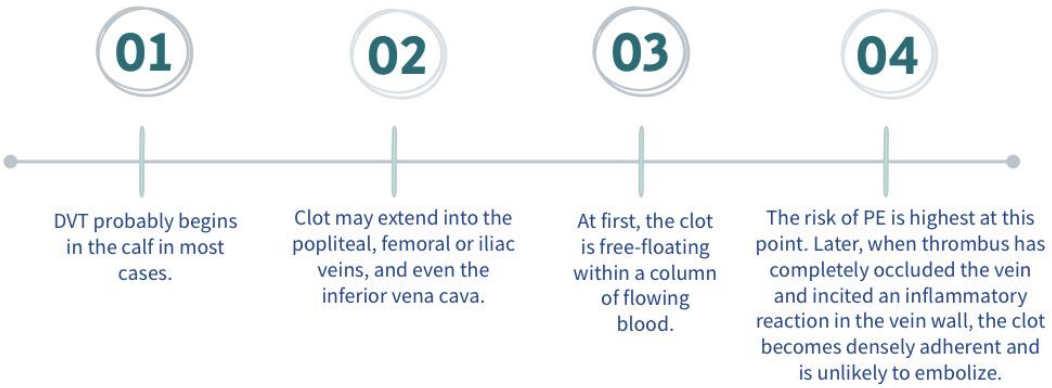
- **Venography**: injection dye and X-ray

Management:

We have to make sure to ask : Is the other veins functioning? What is causing this condition (tumor,DVT)? Is the rest of the veins working?

- **Mechanical**: Compression stocking (a temporary solution)
- **Chemical**: Sclerotherapy (small veins only)
- ★ **Thermal**: EndoVenous Ablation Techniques (laser)
- **Surgical**: Stripping saphenous vein. Big wound, very painful, contraindication: if the rest of the veins are diseased (e.g. DVT)

Discuss the pathophysiology and epidemiology of venous thromboembolism



Discuss the classification and clinical features of venous thromboembolism

- **Virchow's triad:** namely, venous **S**tasis, **E**ndothelial damage and **H**ypercoagulability of the blood (These include antithrombin, protein C and protein S deficiency, as well as factor V Leiden.)
- **Clinical features:** Leg swelling, Dilated superficial veins and Thrombophlebitis

Discuss the diagnosis and management of venous thromboembolism

Diagnosis:

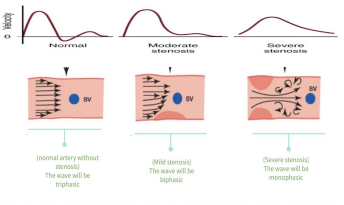
- **Duplex**: first choice
- **Venography**: MR or CT in doubt

Management:

- **Heparin:** if there's high suspicion of DVT
- **Uncomplicated:** elastic stocking and physical exercise and LMWH
- **Complicated:** use thrombolysis, insert a caval filter or consider thrombectomy

Vascular investigations

Obj1: Identify the types of vascular investigations including:



- **Handheld Doppler:** Used to hear the arterial signals in the peripheral arteries. if it is not palpable, The Doppler device compares the frequency of backscattered sound from moving red blood cells with the transmitting frequency.
  - If **stenosis** velocity will *increase*.
  - If **occlusion** velocity will be *zero*.

- **Ankle Brachial Index:** **(First step investigation in peripheral arterial disease)** Normally the pressure in upper limb and lower limb is the same so if you divide the systolic pressure of the ankle by the pressure of the brachial the result will be 1 (*we accept the range between 0.9-1.29*). ABI is limited in evaluating the calcified noncompressible vessels, it will give false positive.

★ Interpretation of ABI	
>1.30	Noncompressible (In diabetes with calcified vessels)
1.00-1.29	**Normal**
0.91-0.99	**Borderline** (equivocal) acceptable
0.41-0.90	Mild to moderate peripheral arterial disease Seen in intermittent claudication
0.00-0.40	Severe peripheral arterial disease Critical Limb Ischemia, Gangrene or ulcers

- **Segmental pressure:** Measures will be taken from multiple areas (upper thigh, middle thigh, upper leg, and middle leg).
- **Duplex:** help to see pulse, velocity (stenosis). Check for *DVT*, reflux (*Varicose veins, Venous insufficiency*). (Combination of US "anatomy" + Doppler "physiology")
- **CT Angiogram:** with injected contrast. Side effect/contraindications: 1- *Allergy*. 2- *Nephropathy* (renal failure is ok because it's not functioning anyway). 3- *Radiation*. (Check kidney function before)
- **MR Angiogram:** used in soft tissue diseases like *popliteal entrapment syndrome.*
- **Angiography:** Invasive procedure we enter a catheter in a specific artery and inject a dye. Might cause *bleeding*, *hematoma*, *thrombosis* or *pseudoaneurysm*. (The only therapeutic modality) Areas of entry:
  - **Femoral artery 90%:** Accessable, Clear anatomical landmark (femoral head) and Easily compressed against the head of the femur
  - **Brachial artery**
  - **Radial artery**

Obj2: Discuss the classification of vascular investigation based on sensitivity, operator dependency, toxicity and if therapeutic or diagnostic.

Test	Sensitive	Operator dependent	Toxic	Therapeutic
ABI	✓	✓✓✓	✗	✗
Segmental Pressure	✓	✓✓✓	✗	✗
Handheld doppler	✓	★ ✓✓✓	✗	✗
Duplex ultrasound	✓✓✓	✓✓✓	✗	✗
CT angiogram	✓✓✓✓	✗	✓✓✓	✗
MR angiogram	✓✓✓✓	✗	✓✓✓	✗
Angiography	✓✓✓✓✓	✗	✓✓✓	✓✓✓