Your Manual To

Surgery 351

Signs & Symptoms

Treatment

Investigations

Prognosis

Edited by: Ismail Raslan

INTRODUCTION

This booklet is designed to provide you with a better understanding of all the core surgical topics that you'll encounter throughout the "Surgery 351" course. The aim of this booklet was to create a single study source that is concise, yet covers the breadth of the course's topics, which will help you make the most of this course, and prepare you for the corresponding examinations. The content of this booklet is based on the "Surgery 351" course outline. It is a compilation of lecture notes provided by the faculty members to students throughout the year, handouts and notes provided by former students, and further explanations obtained from books.

Features include:

- *All chapters are color-coded according to theme, with a structured layout for all topics.*
- *Includes all topics covered in "Surgery 351" course for the year 2012/2013.*
- *Free space on the right margin of each page that allows you to add comments/notes of your own.*
- *Different symbols have been used throughout this booklet:*
    a. *Frequently tested high-yield facts noted by* 
    b. *Extra notes for further explanation noted by* 
    c. *OSCE hints noted by* 
    d. *Clinical case* 
- *MCQs section after each topic serves as a self-assessment tool for you to test your knowledge*
- *Margin boxes highlight important notes and provide further explanations*
- *Illustrations, tables, and clinical images to enhance understanding*
- *Available in softcopy and hardcopy*

We hope that you find this booklet useful, and we wish you good luck in all of your future medical endeavors. We also value your feedback and would like to hear from you if you have any general suggestions or any corrections for any errors that may have crept in.

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CONTENTS

BURNS AND WOUND HEALING

1 INTRODUCTION

1.1 SKIN FUNCTION

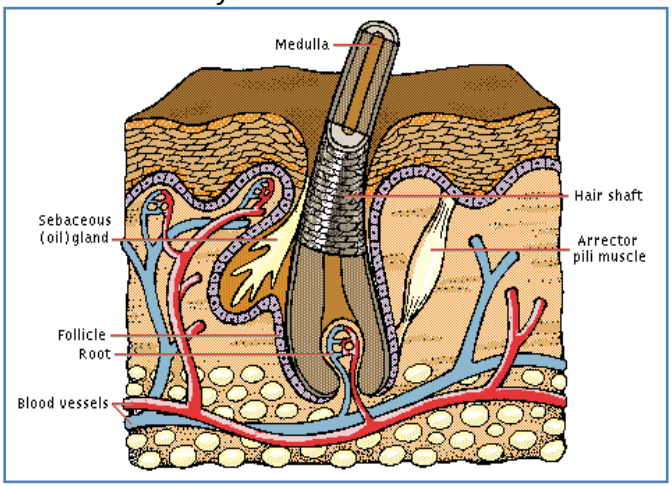
- Body Covering
- Permit movement of underlying muscles & joint
- Sensors for touch, pain, and temperature
- Vitamin D production
- Temperature regulation
  - Sweating, blood flow
- Sun protection
  - Detoxification/activation of drugs and chemicals
- Immuno-surveillance
  - Langerhans cells, t-lymphocytes

Skin appendages examples:

- Sebaceous Glands
- Sweat Glands
- Hair Follicles
- Nails

1.2 SKIN LAYERS

- **Epidermis**
  - Outer layer contains the stratum corneum
  - The rate limiting step in dermal or percutaneous absorption is diffusion through the epidermis
- **Dermis (Appendages)**
  - Much thicker than epidermis
  - True skin & is the main natural protection against trauma
  - Contains
  - Sweat glands
  - Sebaceous glands
  - Blood vessels
  - Hair
  - Nails
- **Subcutaneous Layer (Fat)**
  - Contains the fatty tissues which cushion & insulate



2 BURNS

2.1 CAUSES OF DEATH

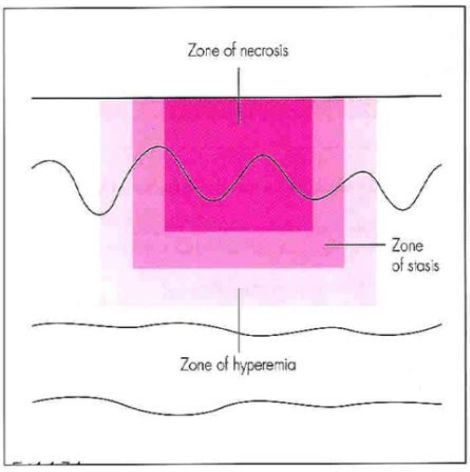
- Smoke inhalation, sepsis, pneumonia, shock
  - More common in elderly
  - (Age + BSA= %mortality)
    - most with>70% die

2.2 RISK FACTORS FOR DEATH:

- > 40% BSA (Body Surface Area),
- > 60 years
- Inhalation injury

2.3 PATHOPHYSIOLOGY OF BURNS

- Dynamic injuries
  - Cellular damage at >45° C
  - Dependent on temperature and duration
- **Three zones of injury**
  - Central necrosis
  - Zone of stasis (at risk of necrosis)
  - Zone of hyperemia
- Thermal injury triggers intense inflammatory response SIRS
  - Initial release of histamine, bradykinin
  - Release of prostanoids, free radicals, proteases
- Leading to:
  - Hypermetabolism.
  - Bacterial translocation.
  - MOF. (Multi-organ failure)



- **Central Zone Of Necrosis**
  Irreversible
- **Intermediate Zone Of Stasis**
  Reversible (The damage is not enough to be irreversible)
- **Outer Zone Of Hyperemia**
  Inflammatory response, vasodilatation

We can classify burns according to the cause:
Thermal, chemical, electrical and friction injuries

Zones of Injury

Our aim after a burn injury at the zone of stasis has occurred is to direct it towards the zone of hyperemia and away from the possibility of necrosis.

Sepsis is one of the major causes of death in burn patients

We can classify burns according to the cause: Thermal, chemical, electrical, friction injuries

Table 2. Burn Depth Classification.

Depth	Histology	Appearance	Sensation	Healing
First-degree:	Epidermis only	Erythema; blanches with pressure	Intact; mild to moderate pain	3-6 days without scarring
Second degree:				
· **Superficial**	Epidermis and superficial dermis; skin appendages intact	Erythema, blisters, moist, elastic; blanches with pressure	Intact; severe pain	1-3 weeks; scarring unusual
· **Deep**	Epidermis and most dermis; most skin appendages destroyed	White appearing with erythematous areas, dry, waxy, less elastic; reduced blanching to pressure	Decreased; may be less painful	>3 weeks; often with scarring and contractures
Third-degree:	Epidermis and all of dermis; destruction of all skin appendages	White, charred, tan, thrombosed vessels; dry and leathery; does not blanch	Anesthetic; not painful (although surrounding areas of second-degree burns are painful)	Does not heal; severe scarring and contractures

Examples:

- Sun burn→ 1st degree burn
- Spilled hot water→ mostly cause 2nd degree
- Flame burn→ mostly cause 3rd degree

Superficial Second Degree Burns

- Epidermis and the upper dermis
- Usually no scarring
- Severe Pain
- Blanches with pressure
- Pinkish in color
- Small **Blisters**
- Treatment --> Flamazine

Deep Second Degree Burns

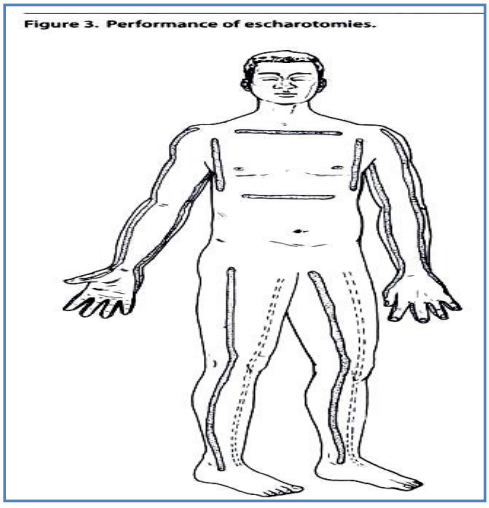
- Epidermis and most of the Dermis
- Lead to scarring
- Less Pain (Due to nerve damage)
- Doesn't blanch with pressure
- Whitish or Cherry Red
- Larger **Blisters** --> Sometimes with Hemorrhage
- Treatment --> Surgical

Third Degree Burns

- All Skin Layers
- Severe scarring
- Painless
- Whitish of Black in color
- Thrombosed vessels/No blood flow
- Skin feels like **leather**
- Eschar formation (Needs Escharotomy)
- Treatment --> Surgery and Skin grafing

Compartment Syndrome:

- Is the compression of nerves, blood vessels, and muscle inside a closed space (compartment) within the body.
- This leads to tissue death from lack of oxygenation due to the blood vessels being compressed by the raised pressure within the compartment.
- You must always look for **circumferential burns** around the chest, abdomen, limbs, etc… and perform an **Escharotomy** to release the pressure



2.4 DETERMINING EXTENT OF INJURY

- Burn extent determines therapy and prognosis
- Burn size estimate is often inaccurate
- Extent of injury described using percentage of total body surface area that is burned (TBSA)
- **For patients > 9 "rule of nines" may be used**
- **For small burns, the patient's palm covers 1%**
- **With young children proportions differ**

Blisters are always seen in **second degree** burns

2.5 EVALUATION OF BURNS

- Look for circumferential burns to chest, neck and limbs that may compromise ventilation or circulation
- Loss of distal pulses late
- Assess for warmth, sensation, motor, rigidity
- Doppler exam is helpful
- Identify potential abuse (Mostly suspected if the patient is a child or an elderly, check if the story matches the burns type and sit)
- Well circumscribed, feet, ankles, buttocks

Once the burn injury is more than 30% of the body surface area the inflammatory response will be systemic

- **(SIRS: S**ystemic **I**nflammatory **R**esponse **S**yndrome**)**
- There will be ***systemic vasodilatation***,
- Fluid will shift from the intravascular space to the extra vascular space
- Which leads to hypo-perfusion to vital organs such as the *kidneys*; causing renal failure.
- Hypo-perfusion to the *intestines* may happen causing Intestinal ischemia
- Bacteria will shift into the blood stream (Bacterial Translocation) → Leading to sepsis (un-managed will lead to death).

Figure 1. Estimating the percent total body surface area of burns in adults.

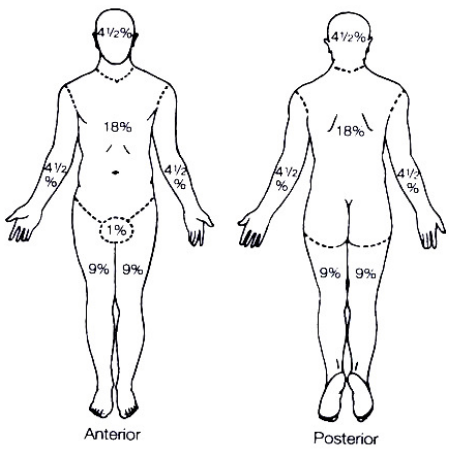
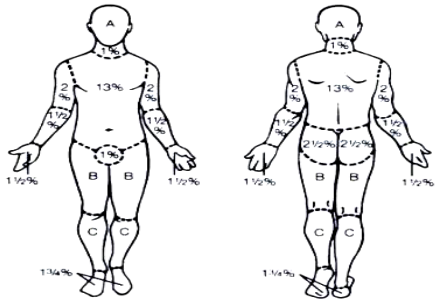


Figure 2. Estimating the percent total body surface area of burns in children.



Relative percentages of areas affected by growth

Age	Half of head (A)	Half of one thigh (B)	Half of one leg (C)
Infant	9½	2¾	2½
1 yr	8½	3¼	2½
5 yr	6½	4	2¾
10 yr	5½	4¼	3
15 yr	4½	4½	3¼
Adult	3½	4¾	3½

How do we calculate the surface area?

By using the rule of nine for normal sized adults (The body is divided into 9 areas) as the Following:

Adults

- All lower limbs 18% (9%front 9% back)
- All upper limbs 9%
- trunk: anterior 18% posterior 18%
- Head and neck 9%

Kids

- Head & neck: 18%
- Each Lower Limb: 4%
- Or by using the Lund-Browder chart

How can we calculate scattered burns?

- The palm of the patient is 1%, use it for measurement.

3 INHALATION INJURY

3.1 SMOKE INHALATION

- **Carbon Monoxide Poisoning**
  - **CO has stronger affinity for HGB than O2**
- **Signs of CO poisoning:**
  - **Confusion, dizziness, HA, NV, flushed skin**
  - **<u>Treatment 100% FiO2</u>**
- Upper Airway Obstruction
  - Common in head and neck burns and smoke inhalation
  - Edema continues at least 24 hours
  - **Protect airway with intubation**
  - Edema usually decreases by post burn day 3
- Pulmonary Injury from Chemical Inhalation
  - Develops ARDS within 24 hours post injury
  - Pneumonia may occur as late as post burn day 10
- Inflammation and systemic reactions
- **Poisoning:** When fire affects furniture (flame burn in closed space)→ Toxins get released into the air → inhaling these toxins affects the lungs directly causing" inflammation pneumonitis" and later pneumonia. There could also be systemic poisoning due to inhaled fumes like Cyanide

What to do in the ER:

1. ABC (A= Air way, B= breathing, C= Circulation)
2. Take detailed history
3. IV access
4. Blood test
5. Allergy (mainly to sulfa because *Flamazine* contains it)
6. Quick general exam
7. Estimate the percentage and depth of the burn.

Table 10. Burn Unit Referral Criteria.

1. Partial-thickness burns greater than 10% TBSA
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints
3. Third-degree burns in any age group
4. Electrical burns, including lightning injury
5. Chemical burns
6. Inhalation injury
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
8. Any patients with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality
9. Burned children in hospitals without qualified personnel or equipment for the care of children
10. Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention

4 FLUID RESUSCITATION

- **Hypovolemia was major cause of death**
- **Massive transudation of fluids from** vessels due to increased permeability
- Edema intensifies over 8-48 hours
- Goal: preservation of organ perfusion and urine output

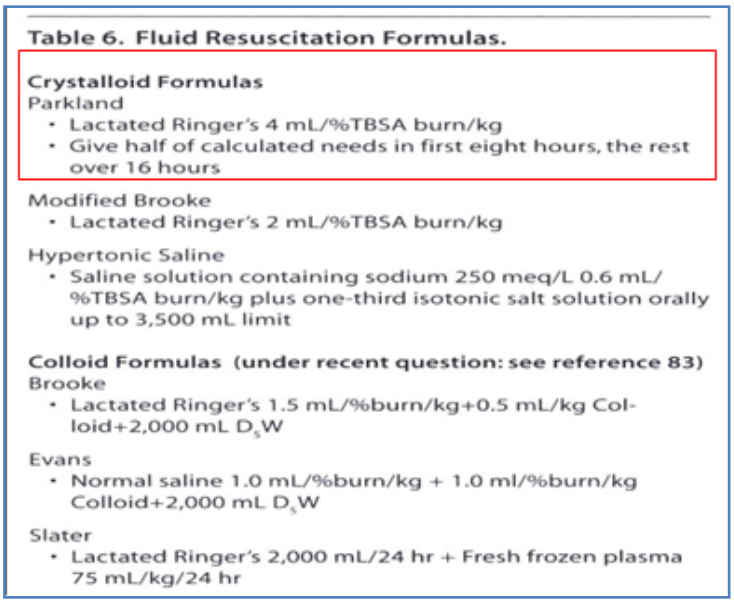


Table 6. Fluid Resuscitation Formulas.

Crystalloid Formulas

Parkland
- Lactated Ringer's 4 mL/%TBSA burn/kg
- Give half of calculated needs in first eight hours, the rest over 16 hours

Modified Brooke
- Lactated Ringer's 2 mL/%TBSA burn/kg

Hypertonic Saline
- Saline solution containing sodium 250 meq/L 0.6 mL/%TBSA burn/kg plus one-third isotonic salt solution orally up to 3,500 mL limit

Colloid Formulas (under recent question: see reference 83)

Brooke
- Lactated Ringer's 1.5 mL/%burn/kg+0.5 mL/kg Colloid+2,000 mL D_5W

Evans
- Normal saline 1.0 mL/%burn/kg + 1.0 ml/%burn/kg Colloid+2,000 mL D_5W

Slater
- Lactated Ringer's 2,000 mL/24 hr + Fresh frozen plasma 75 mL/kg/24 hr

How much IV fluids should we give a burn patient? (If needed)
- **PARKLAND formula (crystalloid)** → most common
- 4cc X (% of burn) X weight of patient = total amount of fluid needed in **24 hrs**
- Half of the amount calculated is given in first 8 hrs, the other half is to be given in the next 16 hrs (start counting from the time of burn NOT when you see the patient in the ER).

5 ELECTRICAL BURNS

- 4th degree if the current passes through the body
- Caused by passage of electric current
- Damage increased in small bony areas
  - Fingers, feet, lower legs, forearm
- Systemic effects
  - Low voltage (<1000 V) (May cause arrhythmias)
  - High voltage (>1000 V): Massive tissue damage, respiratory and cardiac arrest
- ECG, CPK, UA, monitor
- Local care often necessitates grafting and amputation

- Electrical burns are the only type of burns that have an entry and exit point.
- They may be minimal on the surface; we should check the muscles and bones for any injuries.
- Damage mostly affects the *small bones* (feet, hands, and forearms)
- Damage is due to resistance which generates heat, that's why it's common in small bones (*Because bones have the highest resistance in the body)*

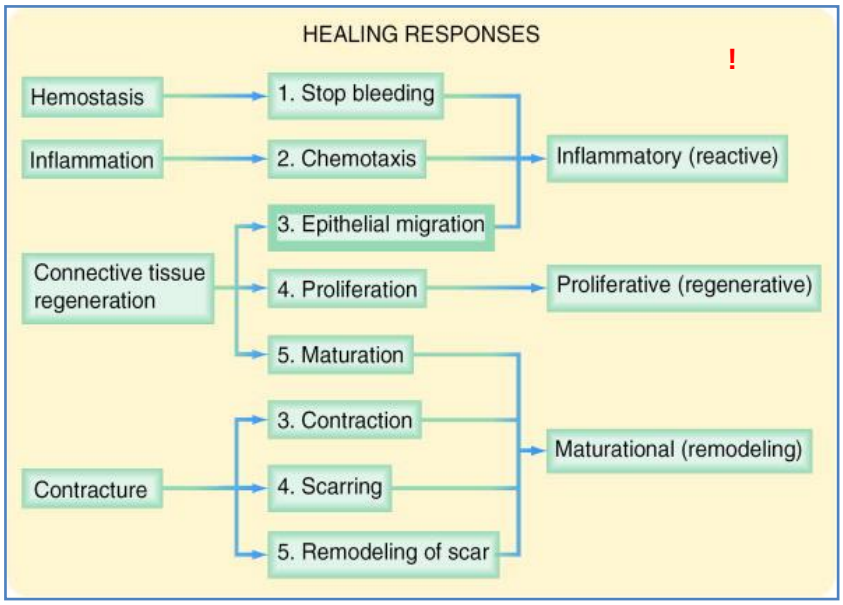
6 CHEMICAL BURNS

- 4th degree if it reaches the fat and muscle
- Delayed and progressive injury
- Deceptively superficial at first
- Acid more limited (coagulation necrosis)
- Alkalis more destructive **(liquefaction)**
- **HFl: significant necrosis, arrhythmias** (Worst chemical burn because it has both mechanisms of acid and alkaline. It burns like an acid because it is an acid and when the fluoride (alkaline) is released it reaches the bone and causes decalcification leading to hypocalcaemia and arrhythmias)
- **Hypo Calcemia**
- Removal of causative agent
- Brush off metals and powders
- Copious irrigation with water

Types of Chemical Burns:
1- Acids: cause **coagulation** and regular burn necrosis and will stop at that level (limited).
2- Alkaline: "worse" causes **liquefaction** that may continue for hours after the injury (deep)

7 WOUND HEALING (THREE PHASES)

- **Wound:** a disruption of normal anatomic relations as a result of injury intentional or unintentional. Regardless of causation or tissue type, wound healing presents with identical biochemical and physiologic processes, though wound healing may vary in timing and intensity.



7.1 INFLAMMATORY PHASE

Neutrophils are the first cells to arrive at the site of injury

- Substrate or reactive phase, immediate
  - Typically **days 1-10**
- Response to **limit and prevent further injury, inflammation, hemostasis, sealing surface, removing necrotic tissue and debris, migration of cells** into wound by chemotaxis, cytokines, and growth factors
- **Initial** intense local **vasoconstriction** of arterioles and capillaries **followed by vasodilation** and vascular permeability
- Tissue injury & blood vessel damage

- Exposure of **subendothelial collagen** to **platelets** and **vWF** activates the **coagulation** pathway
- **Plugging:** Platelet and fibrin
- **Provisional matrix**:
  - Platelets, fibrin, and fibronectin
- **Platelet aggregation**:
  - Thromboxane (vasoconstrict), thrombin, platelet factor 4

7.1.1 PLATELETS

- **Alpha granules** contain:
  - Platelet factor 4: aggregation
  - Beta-thrombomodulin: binds thrombin
  - PDGF: chemoattractant
  - TGF-beta: key component tissue repair
- **Dense granules** contain **vasoactive** substances: adenosine, serotonin, and calcium
- Other factors released: TXA, Platelet activate factor, Transform. growth factor alpha, Fibroblast growth factor, Beta lysin (antimicrobial), PGE2 and PGI2 (vasodilate) and PGF2 (vasoconstrict).

7.1.2 POLYMORPHONUCLEAR CELLS

- **Chemotoxins** attract after extravasation
- Migrate through the ECM by transient interaction with **integrins**
- PMNs **scavenge**, present **antigens**, provide cytotoxicity-**free radicals** (H2O2)
- Migration PMNs **stops** with wound **contamination control** usually a few days
- **Persistent contaminant**: continuous influx PMN's and tissue destruction, necrosis, abscess, & systemic infection
- **PMNs are <u>not essential</u> to wound healing**

7.1.3 MACROPHAGES

ⓘ **Macrophages** are the most important cells in the **inflammatory phase** of wound healing

- **Necessary**
- Monocytes migrate & activate: Macrophages
- Appear when PMN's disappear 24-48 hr
- Do the same activities as PMN's
- Plus orchestrate release of enzymes (collagenase, elastase), PGE's, **cytokines** (**IL-1**, **TNF alpha**, **IFN**), **growth factors** (**TGF** & **PDGF**), and **fibronectin** (scaffold/anchor for fibroblasts)
- **Activate** fibroblasts, endothelial and epithelial cells to form Granulation Tissue

7.2 PROLIFERATIVE PHASE

The Proliferative phase depends on **Fibroblasts.**

- Regenerative or Reparative
  - **day 5 to 3 weeks**
- **Angiogenesis:** endothelial cells activate & degrade basement membrane, migrate, and divide to form more tubules
- **Granulation Tissue:** capillary ingrowth, collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG)

7.2.1 FIBROBLASTS

- **Differentiate** from resting mesenchymal cells in connective tissue **3-5 days migrate** from **wound edge**
- **Fibroplasia**: Fibroblasts proliferate replace **fibronectin-fibrin** with **collagen** contribute ECM

7.2.2 COLLAGEN

- **Type III** predominant collagen synthesis **days 1 to 2**
- **Type I** days **3 to 4**
- **Type III replaced by Type I** in **3 weeks**

The most common collagen type in **normal woundless skin** is type1 followed by type 2

The most common type in **wounded (scarred) skin** is type 3

7.2.3 WOUND STRENGTH

- Week 6 = 60% original, 80% final strength
- Week 8 to 1 year ≈ 80% original (Max)
- Net Collagen = 6 weeks amount stays the same but cont. crosslink increase strength = maturation

I	80% of skin Most Common: skin, bone, tendon. **Primary type in wound healing.**
II	Cartilage
III	**20% of skin** **Increased Ratio in healing wound,** also blood vessels and skin
IV	Basement Membrane
V	Widespread, particularly in the cornea

Granulation tissue contains:

Capillary ingrowths, Collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG)

7.3 MATURATIONAL PHASE

- **Remodeling of wound 3 week-1+year**
- **Type I replaces Type III Collagen:** net amount doesn't change after 6 weeks, organization & cross-linking
- Decreased vascularity, less fibroblasts & hyaluronic acid
- Peripheral nerves regenerate at 1mm/day
- Accelerated Wound Healing: reopening results in quicker healing 2nd time around
- Contraction: centripetal movement of the whole thickness of surrounding skin reducing scar
- **Myofibroblasts**: special Fibroblasts express *smooth muscle* and bundles of actin connected through cellular fibronexus to ECM fibronectin, communicate via *gap junctions* to pull edges of the wound

8 ABNORMALITIES

- **Contracture (**A minimization for wound's size due to **Myofibroblasts)**
  - The physical constriction or limitation of function as the result of Contraction (scars across joints, mouth, eyelid)
- **Keloids: Beyond the Borders**
  - **Excess deposition of collagen causes scar growth <u>beyond</u> the border** of the Original wound

When burn-caused contractions affect the function of a joint it is called **a Contracture**

Most common sites: Perineum and Trunk, then Head and neck, then Extremities.

  - Tx: XRT, steroids, silicone sheeting, pressure, **excise**, **often Refractory to Tx & but not preventable.**
  - Occur in specific areas such as: earlobes and sternum
- **Hypertrophic Scar: confined within**
  - Excess collagen deposit causing raised scar remains within the original wound confined
  - **Darker pigmented skin & flexor surfaces of upper torso**
  - Often occurs in burns or wounds that take a long time to heal, sometimes preventable
  - Can regress spontaneously
  - Tx: steroids, silicone, pressure garments
  - Surgical excision makes it worse

9 IMPEDIMENTS TO WOUND HEALING

- **Bacteria**>$10^5/cm^2$: Decreased O_2 content, collagen lysis, prolonged inflammation
- **Devitalized Tissue & Foreign Body**: Retards Granulation Tissue formation and healing
- **Cytotoxic drugs**: **5FU, MTX, Cyclosporine**, **FK-506** can impair wound healing. **D-Penicillamine**- **inhibit collagen x-linking**
- **Chemotherapy:** no effect after 14 days
- **Radiation:** Collagen synthesis abnormal, fibrosis of vessel
- **Diabetes**: impedes the **early phase** response
- Malnourishment: **Albumin<3.0**, **Vit-C**
- **Smoking:** vasoconstriction, atherosclerosis, carboxyhemoglobin, decreased O_2 delivery
- **Steroids**: inhibit macrophages, PMNs, Fibroblast collagen synthesis, cytokines, and decreased wound tensile strength
- **Vit A** (25,000 IU QD) counteracts effect of steroids
- **DENERVATION** has **NO EFFECT** on Wound Healing

10 DISEASES ASSOC WITH ABNORMAL WOUND HEALING

- **Osteogenesis Imperfecta**: Type I Collagen defect
- **Ehler-Danlos syndrome**: Collagen disorder, 10 types
- **Marfan Syndrome**: fibrillin defect (collagen)
- **Epidermolysis Bullosa**: Excess fibroblasts Tx: phenytoin
- **Scurvy**: **Vit C** req. for proline hydroxylation

11 MCQS

1. **Which type of Collagen is the primary type in wound healing**
   A. Type 1
   B. Type 2
   C. Type 3
   D. Type 4

2. **Platelet secretes ____ in vasoconstriction phase:**
   A. Collagen
   B. ILKs
   C. Thromboxane
   D. Neutrophils

Answer Key → 1A, 2C

SHOCK

1 OBJECTIVES

- To understand Physiology of sustaining blood pressure.
- To learn about the classifications of shock.
- To understand the consequences of the natural history of shock.
- To be able to diagnose and plan appropriate treatments for different types of shock.

2 BLOOD PRESSURE REGULATION

Changes in many elements regulate **BP** and **perfusion**:

1. Intravascular volume
2. Heart
3. Arteriolar bed
4. Capillary exchange network
5. Venules
6. Venous capacitance circuit
7. Large vessel patency

2.1 PERIPHERAL RESISTANCE

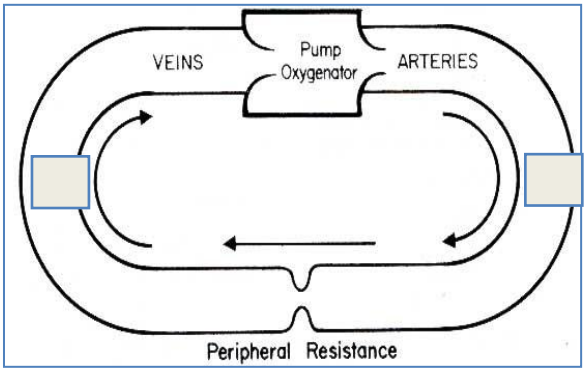


Figure 1

- Decreased peripheral resistance:
  - Decreased arterial blood pressure (**MAP = CO X PR**)
- Increased peripheral resistance
  - Decreased venous return
  - Decreased EDV
  - Decreased SV
  - Decreased CO (**CO = HR X SV**)
  - Decreased arterial blood pressure (**MAP=CO X PR**)

Heart Rate X Stroke Volume *(↓ intravascular volume, ↓ EDV)* ***= Cardiac Output***
Cardiac Output X Peripheral Resistance = Arterial Pressure

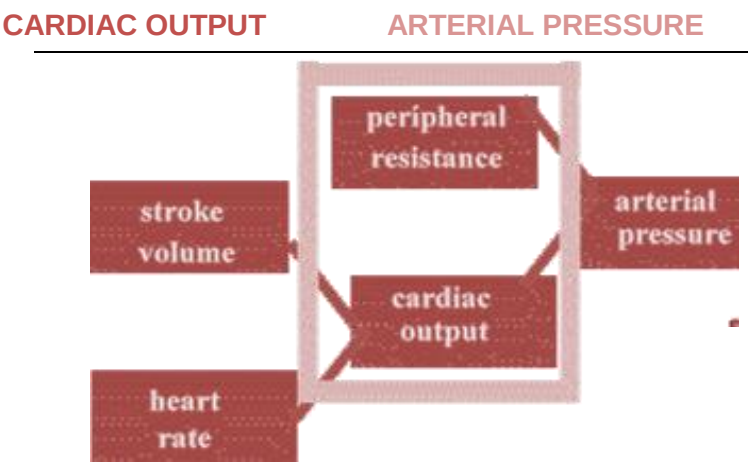


Figure 2

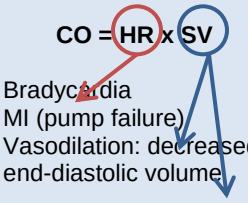
2.2 EFFECTS OF INTRAVASCULAR VOLUME ON BP & PERFUSION

- Alters mean blood pressure
  - Decrease in intravascular volume=decreased BP
- Alters venous return to the heart
  - Decrease in intravascular volume=
  - Decreased venous return=
  - Decreased end diastolic volume
  - CO = HR x SV
  - COXSVR=MAP
- How can intravascular volume be lost?
  - Examples:
    - Bleeding
    - Failure to rehydrate
    - Loss of third space fluids (sweating)

What maintains blood pressure in our system and arteries is the heart.

2.3 EFFECTS OF CARDIAC FUNCTION ON BP & PERFUSION

- Cardiac output is the result of:
  - Heart rate
  - Contractility
  - Loading conditions
- Examples of changes that can alter cardiac output:
  - Heart rate (bradycardia or tachycardia)
  - Contractility (MI or cardiomyopathy) i.e. pump failure
  - Load (histamine release: vasodilation



2.4 EFFECTS OF THE RESISTANCE CIRCUIT (ARTERIOLAR BED) ON BP & PERFUSION

- Decreases in arteriolar tone produce:
  - Hypotension

- Decreased perfusion to vital organs
- Increases in tone will prevent optimal cardiac performance (increased afterload=decreased contractility)

Further explanation:
The heart delivers blood to all organs by the same mean arterial pressure. Because of that, the width of the arterioals is what determines blood flow to each organ. Arterioles dilate and contract to alter their vascular radius depending on each organs requirement. Arteriolar tone can be modulated by are regulated by complex substances and mechanisms, but the most are:

- **Vasoconstrictors**:
  1. Sympathatic tone
  2. Vassopressin
  3. Endothelin
- **Vasodilators**:
  1. Decreased organ perfusion
  2. Nitric Oxide (NO)
  3. Any decrease in inherits vasoconstriction regularly provided by the myogenic activity and sympathetic stimulation.

What determine the BP in the arterioles?
- Increase in the permeability and the oncotic pressure. Oncotic pressure will increase due to the presence of proteins in the blood vessel which lead to fluid shift from extravascular to intravascular space, leading to volume expansion, this physiological process can be disturbed in case of sepsis, trauma or systemic inflammatory response (SIR)

What is oncotic pressure?
- It is the osmotic pressure, which result from the presence of proteins in the blood vessel.

2.5 EFFECTS OF THE CAPILLARY EXCHANGE NETWORK ON BP & PERFUSION

- Largest area of the vascular tree
- Site of exchange of nutrients, electrolytes and fluids
- Alterations in microvascular integrity (e.g., capillary leak syndrome) result in loss of intravascular volume
- Blockage of or shunting away from small vessels leads to decreased tissue perfusion

2.6 EFFECTS OF THE VENOUS CAPACITANCE CIRCUIT ON BP & PERFUSION

- Portion of the circulatory system contains 80% of the intravascular volume
- Decrease in effective circulating blood volume and MAP caused by:
  - Decreases in venous tone
  - Increases in venous vascular capacitance

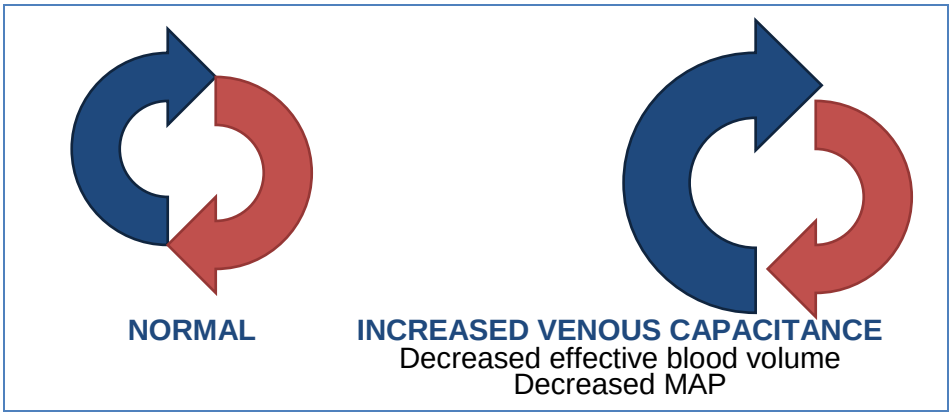


Figure 3

2.7 EFFECTS OF LARGE VESSEL PATENCY ON BP & PERFUSION

- Obstruction of the systemic or pulmonic circuit will decrease ventricular ejection and systemic perfusion
- Venous obstruction will decrease venous return
- Examples of obstructive shock:
  - Massive pulmonary embolism
  - Venous occlusion

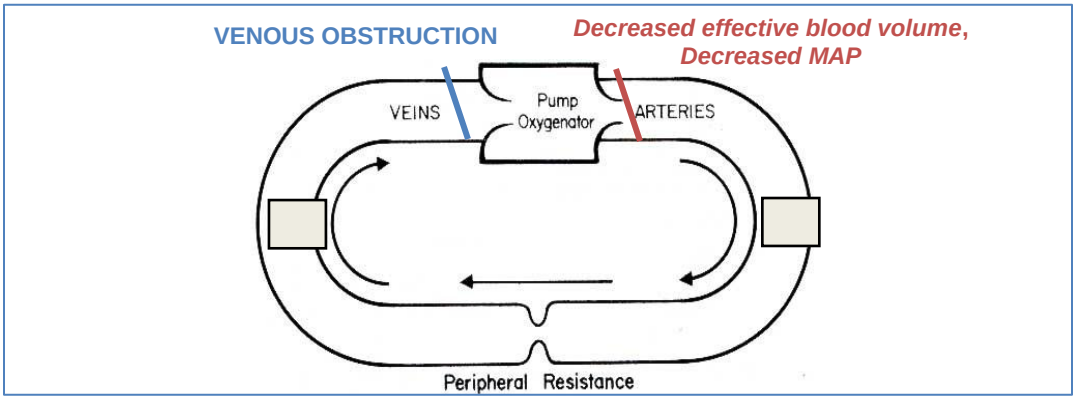


Figure 4

3 SHOCK:

- **Definition**: It is the state of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function. In short: low perfusion that causes tissue hypoxia.

3.1 TYPES OF SHOCK:

  - ⓘ More than one type may be present!

Type of Shock	Clinical Causes	Primary mechanism
1. Hypovolemic	Volume loss	Exogenous blood, plasma, fluid or electrolyte loss
2. Cardiogenic	Pump failure	Myocardial infarction, cardiac arrhythmias, heart failure
3. Distributive *"shock that will result in vasodilatation > vasodilatation or leak > lead to the movement of the blood outside the vessel > decrease the end diastolic volume."*	↑ venous capacitance or arteriovenous shunting	Septic shock, spinal shock, autonomic blockade, drug overdose ***"Neuorogenic, anaphylactic, septic"***
4. Obstructive	Extra-cardiac obstruction of blood flow	Vena caval obstruction, cardiac tamponade, pulmonary embolism, aortic compression or dissection

3.2 SIGNS AND SYMPTOMS:

Clinical signs and symptoms of shock relate to decreased organ perfusion:

- **Mental status changes**: decreased cerebral perfusion
- **Decreased urine output**: decreased renal perfusion
- **Cold clammy extremities:**
  - Decreased perfusion to the skin due to diverted blood flow
- **EKG changes:**
  a. May indicate myocardial ischemia
  b. May be primary event (cardiogenic shock) or due to decreased myocardial perfusion due to shock from other causes

3.2.1 HEMODYNAMIC PARAMETERS THAT MAY INDICATE SHOCK:

- **Heart rate**: Initial tachycardia (attempt to increase CO)
- **Rhythm**: Regular and tachycardic
- **Blood pressure**: Low
- **Cardiac output**: Usually low

3.3 EFFECTS OF SHOCK AT THE ORGAN LEVEL ⓘ

- **Kidney**: Oliguric renal failure
- **Lung**: Capillary leak associated with or caused by sepsis and infection
- **GI tract**: Failure of intestinal barrier (sepsis, bleeding)
- **Liver**: Liver failure, which is a rare cause.

ⓘ What happens to the lung in systemic inflammatory response (SIR)?
Answer: ARDS (adult respiratory distress syndrome)
ⓘ What is ARDS?
Answer: it is a systemic release of inflammatory mediators, causing inflammation, hypoxemia and frequently multiple organ failure. And it may accompany many conditions, but most importantly: sepsis, pancreatitis, and severe traumatic injury.

3.4 HEMODYNAMIC RESPONSE TO SHOCK:

- Mechanisms for restoring cardiovascular homeostasis
  1) Redistribution of blood flow: Attempt to preserve perfusion to vital organs
  2) Augmentation of cardiac output: Increased heart rate Increased peripheral resistance
  3) Restoration of intravascular volume

3.4.1 REDISTRIBUTION OF BLOOD FLOW

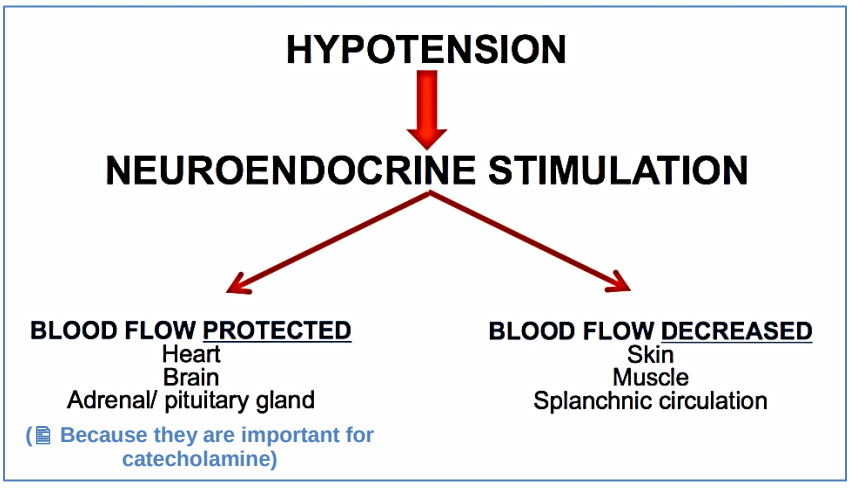


Figure 5

The organ that will contribute in responding to shock is the kidney, how?
- The kidneys are part of the solution not the problem when the body responds to shock; it will retain salt that will maintain intravascular volume.

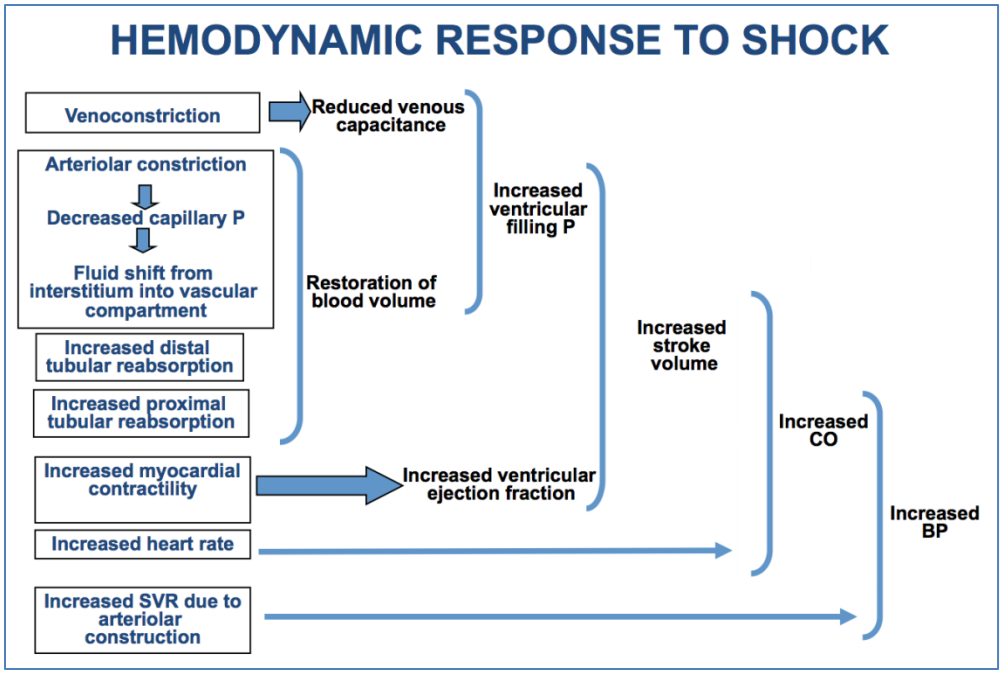


Figure 6

4 TYPES OF SHOCK

4.1 CARDIOGENIC SHOCK:

- Caused by the progressive loss of myocardium
- Usually due to an acute myocardial infarction
- When the total amount of myocardium affected reaches a critical point, myocardial function begins to deteriorate
- While stroke volume decreases, the heart rate increases in an effort to maintain cardiac output (CO = SV x HR)
- But increased HR is limited and CO falls to levels that are inadequate to support end-organ function
- Coronary perfusion decreases and this in turn causes progressive myocardial ischemia with progression of myocardial injury

How do you know if it is cardiogenic or not?
1. SOB
2. Rest JVP?
3. Lower limb edema

4.1.1 DECREASED CARDIAC FUNCTION

- Decreased ventricular function
  - Myocardial infarction
  - Pericaridal tamponade
  - Tension pneumothorax
- Ineffective cardiac contraction
  - Primary arrhythmias

4.1.2 CLINICAL FINDINGS:

1. Hypotension
2. Tachycardia
3. Tachypnea
4. Oliguria

-In cardiogenic shock the volume is not the problem.
-The only shock that you DON'T give fluid is cardiogenic because the pt. might develop pulmonary edema (b\c the ventricle is not functioning, all volume will go to the RT ventricle then to lung)!
-The treatment will depend on the cause.

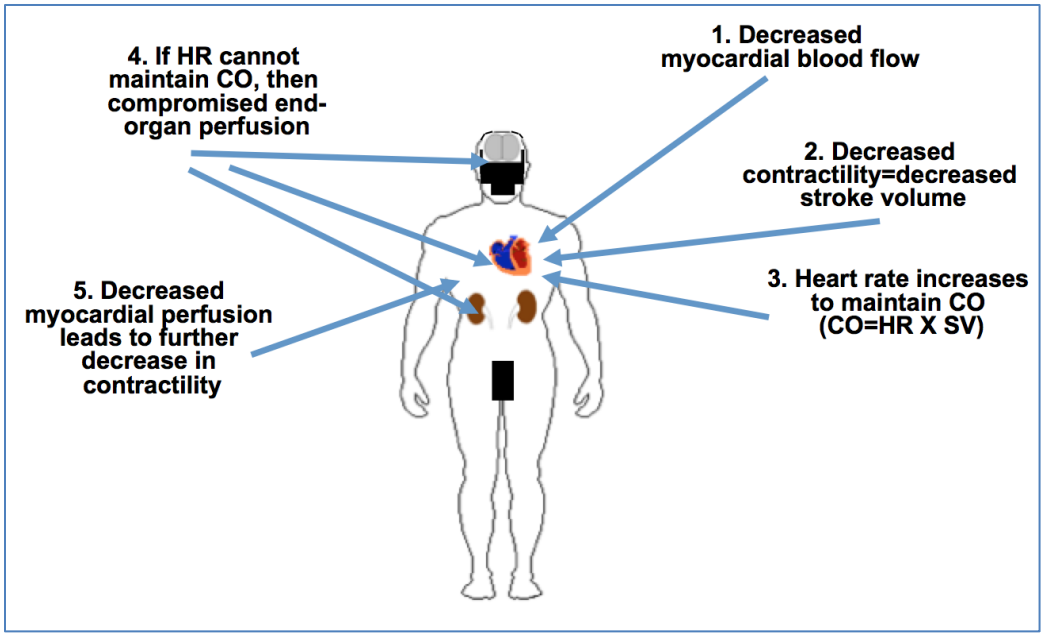


Figure 7

4.1.3 EVENTS IN CARDIOGENIC SHOCK

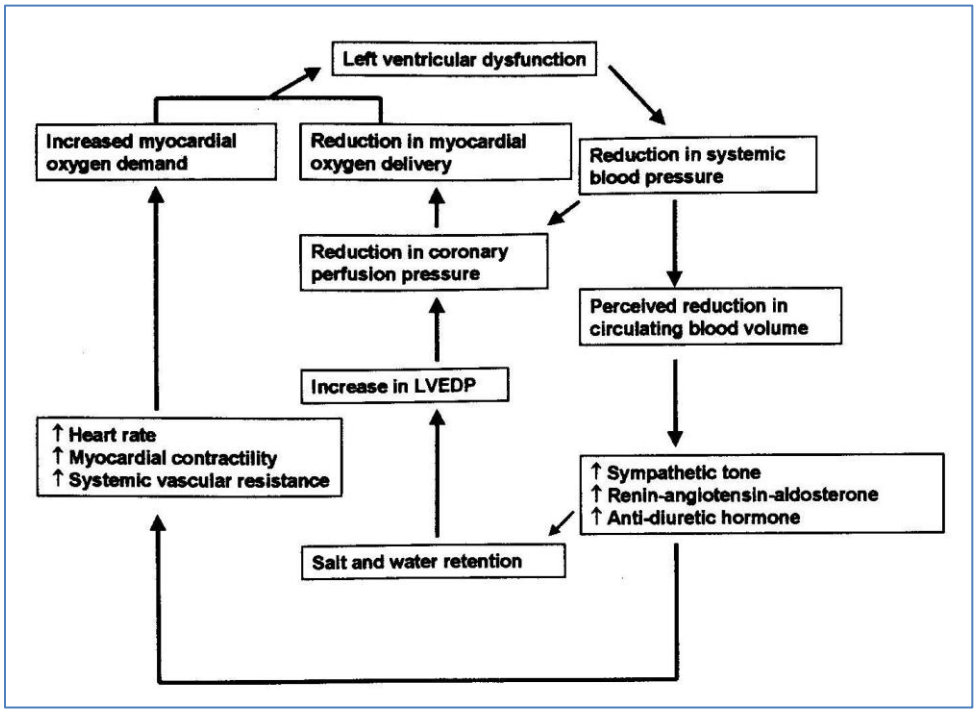


Figure 8

4.2 HYPOVOLEMIC SHOCK

- **Decrease in intravascular blood volume** → e.g. (hemorrhage. Vomiting, diarrhea, fluid sequestration "intraluminal – bowel obstruction, intraperitoneal – pancreatitis, interstitial – burns") → **decrease in cardiac output and tissue perfusion**

- **To treat it**: replace volume + treat the underlying cause.

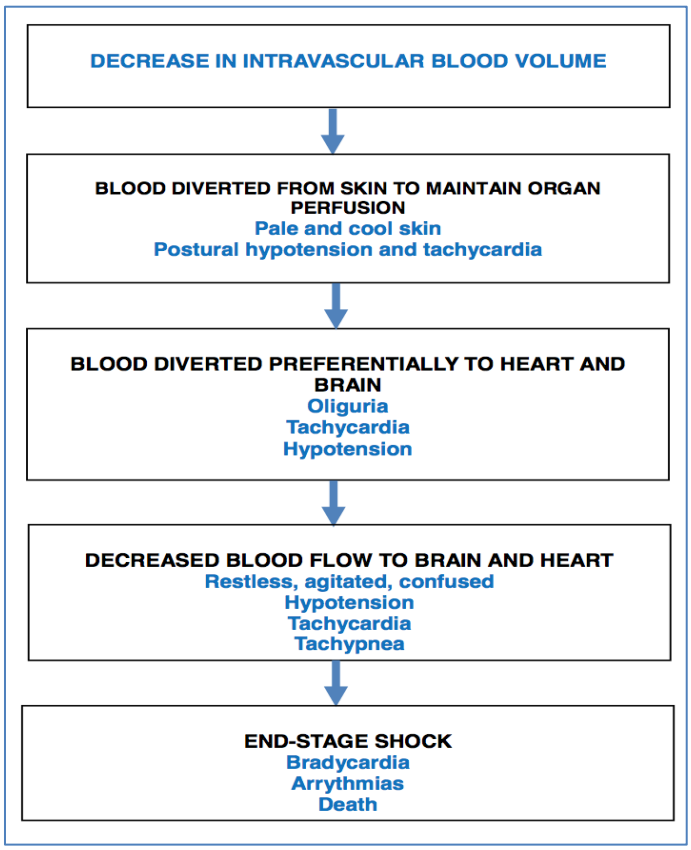


Figure 9

4.3 SEPTIC SHOCK

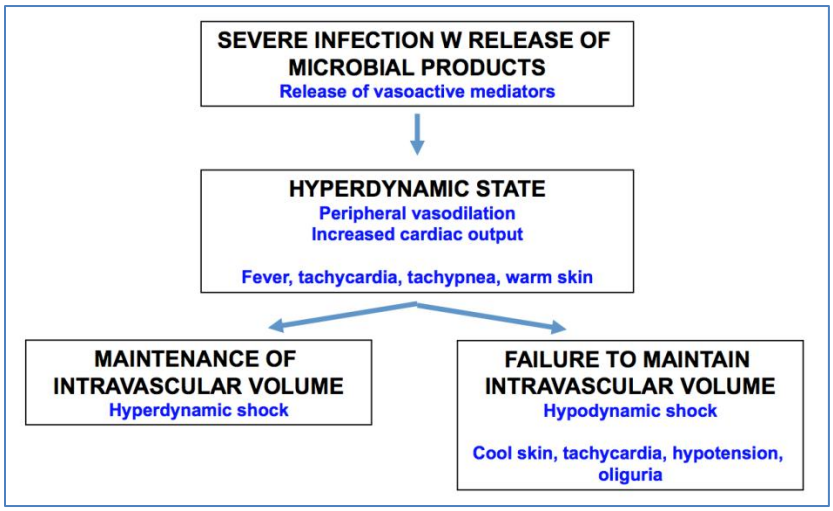


Figure 10

- **To treat it**: replace volume + give antibiotics.

4.4 SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS):

- The patients demonstrate a similar response as sepsis but **WITHOUT INFECTIVE AGENTS**. It's just an inflammatory process.
- The criteria are: (two or more to call it SIRS)
  1) Temperature >38 or < 36 (in sepsis it could be hypothermia OR hyperthermia!)
  2) Heart rate >90
  3) RR > 20 or a pco2 < 34 mmHg (4.3 kpa)
  4) WBC > 12,000 0r < 4,000 with more than 10% bands

5 NEUROGENIC SHOCK:

- It is a shock that result from a high spinal cord injury (e.g Cervical spine traumatic injury). The injury is at level T2 or above
- This will result in loss of sympathetic tone
- Loss of sympathetic tone will result in:
  - Arterial and venous dilatation causing **hypotension**.
  - **Bradycardia** as a result of unopposed vagal tone.
- The typical feature is hypotension with bradycardia (non- neurogenic patient usually have tachycardia as a result of shock).
- **Management** of neurogenic shock:
  1) Assessment of airway
  2) Stabilization of the entire spine
  3) Volume resuscitation
  4) R/O other causes of shock
  5) High dose corticosteroids

6 PRINCIPLES OF RESUSCITATION:

1. Maintain ventilation: ensure oxygen delivery
2. Enhance perfusion
3. Treat underlying cause

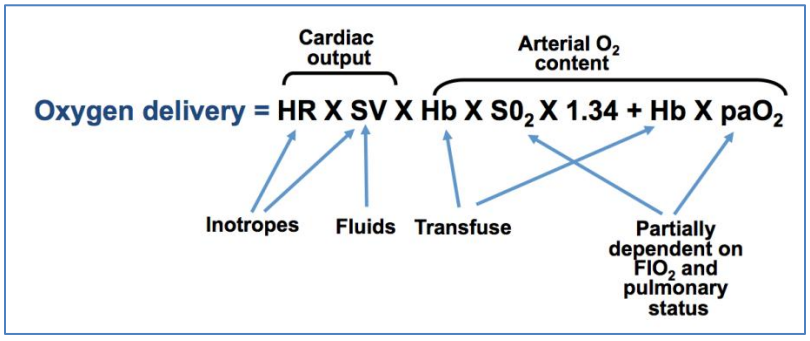


Figure 11: Treatment of shock enhancing perfusion/oxygen delivery:

7 SUMMARY:

1. Shock is an altered state of tissue perfusion severe enough to induce derangements in normal cellular function
2. Neuroendocrine*, hemodynamic and metabolic changes work together to restore perfusion

3. Shock has many causes and often may be diagnosed using simple clinical indicators
4. **Generic classification of shock:**
    a. Circulatory shock:
        i. Critical reduction in tissue perfusion results in organ dysfunction and, if not treated, death.
        ii. Usually accompanied by signs and symptoms:
            a) Oliguria
            b) Mental status changes
            c) Weak thready pulse
            d) Cool clammy limbs
    b. Septic shock:
        i. Hypotension
        ii. Vasodilatation with warm limbs.

TYPE	Central Venous Pressure	Cardiac Output	SVR
Hypovolemic	Decreased	Decreased	Increased
Cardiogenic	Increased	Decreased	Normal or Increased
Septic	Decreased or increased	Increased	Decreased
Traumatic	Decreased	Decreased or increased	Decreased or increased
Neurogenic	Decreased	Decreased	Decreased
Hypoadrenal	Decreased or increased	Decreased or increased	Decreased or increased

Figure 12: Diagnosing shock state based on hemodynamic parameters

5. **Treatment of shock is primarily focused on restoring tissue perfusion and oxygen delivery while eliminating the cause**

8 CASES

Case 1: Circulatory Shock

10 y/o female fell off bike riding down a hill. Initially well but 4 hrs later complained of abdominal pain and left shoulder pain.

- On examination:
  - Vital signs (VS): BP 90/60, P 120 (tachycardic), RR 30 (tachypneic), T 100.1, O2 sat 95% (low)
  - General (GEN): pale, anxious
  - Lung: clear to auscultation
  - Precordium (COR): tachycardic with murmur best heard at base
  - Abdomen (ABD): diffuse tenderness without peritonitis or mass
- Labs: Hb 7.5 (low)
- Hemodynamics:

Central venous pressure	Decreased
Cardiac output	Decreased
Systemic vascular resistance	Decreased

- Abdominal CT: splenic laceration with free peritoneal fluid
- Patient is in **respiratory failure**:

- **Treatment** of respiratory failure:
  1) Primary resuscitation
  2) Oxygen
  3) Mechanical ventilation if necessary

Case 2: Septic Shock

15 y/o male with a 4 day history of abdominal pain, N/V and anorexia

- On examination:
  - VS: BP 70/60 (low), P 130 (high), RR 28 (high), T102.4, O2 sat 99%
  - GEN: moderate distress from abdominal pain
  - COR: tachycardic
  - ABD: diffuse tenderness w peritonitis
- Labs:
  - WBC 19,600 (high), 90% segments
  - Hb 14.2
- Hemodynamics:

Cardiac output	Increased
Systemic vascular resistance	Decreased

- **Dx**: perforated appendicitis

Case 3: Neurogenic Shock

17 y/o male, diving into water

- On examination:
  - VS: BP 90/60 (low), P 110 (high), RR 24 (high)
  - Paralysis below C5
- Hemodynamics:

Central venous pressure	Decreased
Cardiac output	Decreased
Systemic vascular resistance	Decreased

- Cervical X-ray: C5 fracture

Case 4: Cardiogenic Shock

17 y/o male, training for track team

- On examination:
  - VS: BP 70/50 (low), P 140 (high), RR 35 (high), O_2 sat 88%
  - Absent breath sounds in left lung field, distended neck veins
- Dx: tension pneumothorax
- Hemodynamics:

Central venous pressure	Increased
Cardiac output	Decreased
Systemic vascular resistance	Normal

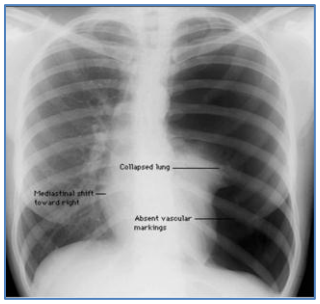


Figure 13: Case 4

Case 5: Capillary Leak Syndrome

3 y/o male, clothes ignited from roaster

- On examination:
  - VS: BP 60/60 (low), P 170 (high), RR 35 (high), T102.4, O2 sat 89%
  - GEN: moderate distress
  - LUNG: tachypneic, clear to auscultation
  - COR: tachycardic, regular
  - SKIN: 60% TBSA partial and full thickness burn
- Hemodynamics:

Cardiac output	Decreased
Systemic vascular resistance	Increased

- **Dx**: 60% of total body surface area (TBSA) burn; hypovolemic shock (loss of fluid into interstitium, called “third spacing”)
- **Rx**: MAINTAIN VENTILATION

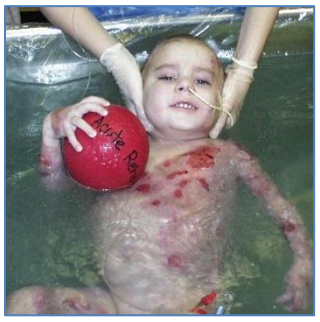


Figure 14: Case 5

What is capillary leak syndrome?

1. Vasodilatation
2. A-V shunting
3. Maldistribution of flow
4. Increased capillary permeability + interstitial edema
5. Decreased oxygen extraction
6. Primary defect of oxygen utilization at cellular level

9 MCQS

1. **Which one of these parameters will appear first and can be diagnostic for shock?**
   a. Hypotension
   b. Bradycardia
   c. Decreased tissue perfusion
   d. Tachycardia
2. **The most sensitive tissue to ischemia is:**
   a. Muscle
   b. Nerve
   c. Skin
   d. Adipose tissue
   e. Bone
3. **Which one of the following does not cause hypovolemic shock?**
   a. Hemorrhage
   b. Trauma
   c. Surgery
   d. Myocardial infarction
   e. Burns
4. **A 25 y/o driver sustained a car accident presented to the ER with flaccid paralysis, bradycardia, and hypotension. The most likely diagnosis is:**
   a. Neurogenic shock
   b. Cardiogenic shock
   c. Hypovolemic shock
   d. None of the Above
5. **The commonest cause of the previous case is:**
   a. Massive external bleeding
   b. Ischemic heart disease
   c. Injury to the high thoracic spine
   d. Internal bleeding

🗝 Answers: 1:d, 2:b, 3:d, 4:a, 5:c

INTRAVENOUS FLUIDS

1 INTRODUCTION

- IV fluid is the giving of fluid and substances (electrolytes) directly into a vein.
- **Substances that may be infused intravenously**: volume expanders (crystalloids and colloids), blood based products ⓘ, blood substitutes & medications

ⓘ Blood- based products include: Whole blood, fresh frozen plasma, cryoprecipitate which is a frozen blood product prepared from plasma.

1.1 PHYSIOLOGY

- Water makes up around two thirds of our total body mass. To be exact, men are 60% water, whilst women are slightly less at 50-55%.
- Total body fluid water is 60% of body weight (BW).

Example:
A 70 kg man will contain about 42 liters, and a 70 kg woman will contain nearly 38 liters. The reason of this difference between the sexes is that women contain an extra 5% adipose tissue; the difference is only occasionally of clinical significance.

Do not get confused:
1 mEq/L = 1 mmol/L
1 cc = 1 ml

1.1.1 FACTORS THAT AFFECT OUR TOTAL BODY FLUID

- Age: the older you get, the more body fluids you lose.
- Gender: females have less total body fluid water.
- Lean body mass: (muscles): Increases total body water. (↑TBW)
- Weight: the higher the levels of fat in the body the lower the total body water will be(↓ TBW)
- **How to calculate TBW?**
  - Male sex TBW= BW× 0.6 / Female sex TBW= BW x 0.5

1.1.2 BODY FLUID COMPARTMENTS

- **Intracellular volume**: (40%) rich in water, the majority of our total body water is in the intracellular compartment.
- **Extra cellular volume**: (20%) rich in water divided into:
  - Interstitial space: contains 15% of water
  - Intravascular space: contains 5% of water
  - The intravascular compartment holds the smallest amount of water at around 3 liters (further 2 liters of red blood cells makes up our total blood volume)
  - The intravascular space is the most important compartment for physicians because:
    - It is the compartment fluid is infused in
    - It absorbs and loses fluid to the interstitial space or to the intracellular compartment.
    - Through this compartment almost all significant losses and gains occur.
    - We'll see the electrolytes also if the patient is dehydrated by pricking the vessel.

Example: How to measure fluid in different compartments?

- 70 kg male: (70x 0.6) TBW= 42 L
- Intracellular volume = .66 x 42 = 28 L or .4 x 70 = 28
- Extracellular volume = .34 x 42 = 14 L or .2 x 70 = 14
- Interstitial volume = .66 x 14 = 9 L

1.1.3 FLUIDS SHIFT/INTAKE

- Water moves freely between the compartments
- We lose water through our renal and gastrointestinal tracts, and this can be seen and measured. The water from our skin and respiratory tract cannot be measured with ease, and makes up our insensible loss. It increases in sickness, particularly when febrile.

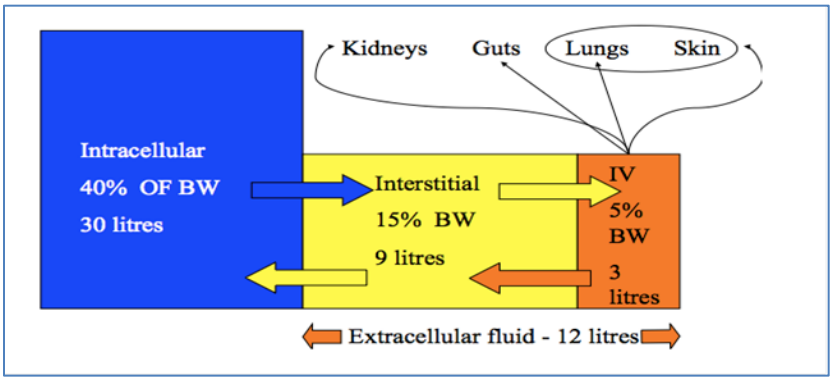


Figure 1

1.1.4 BODY ELECTROLYTES COMPARTMENTS

- Intracellular volume includes: K+, Mg+, phosphate (HPO3-)
- The main +ve **intracellular** electrolyte is **K+/** the main –ve intracellular electrolyte is HPO3-
- Extra cellular volume (**intravascular**) includes **Na+**, Cl^-, Ca^{++}, and albumin.
- The main +ve extracellular electrolyte is Na+ / The main –ve extracellular electrolyte is Cl^-

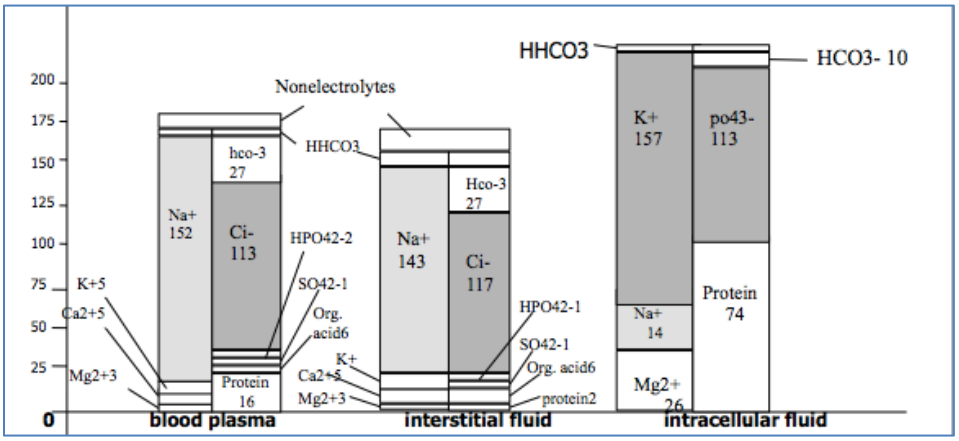
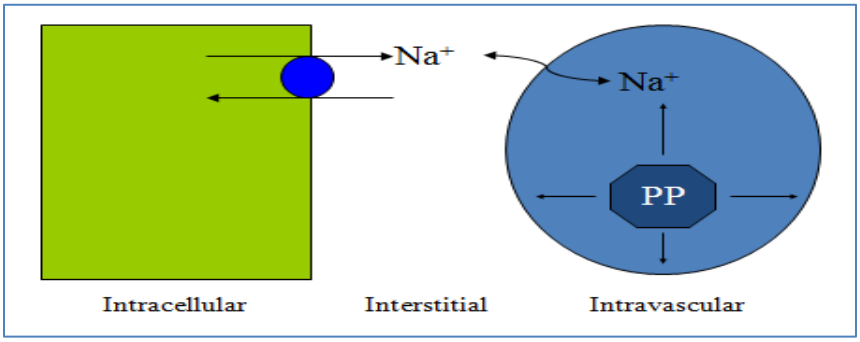


Figure 2: Normal values of electrolytes (know the main cations and anions)

ⓘ Normal electrolyte values:
Serum Na+ = 135 – 147 mmol/L
Serum K+ = 3.5 – 5 mmol/L
Serum Cl^- = 98 – 108 mmol/L
Serum HCO_3^- = 20 – 28 mmol/L
Serum Ca^{+2} = 8.5-10.2 mg/dL (~2.2-2.6 mmol/L)

1.1.5 OSMOTIC / ONCOTIC PRESSURE

- Gibbs – Donnan Equilibrium:



  - It refers to the movement of chargeable particles through a semi permeable membrane against its natural location <u>to achieve equal concentrations on either side</u> of the semi permeable membrane. For example, movement of Cl^- from extra cellular space (natural location) to intracellular space (unusual location) in case of hyperchloremic metabolic acidosis because negatively charged proteins (natural location in intravascular space) are large molecules that cannot cross the semi permeable membrane for this equilibrium.
  - The distribution of water throughout depends on:
    - **Size** of the compartment available (the bigger the size the more fluid it will get)
    - **Tonicity (mainly)**. Water balance is adjusted to maintain osmolality at a constant throughout all three compartments.
    - **Oncotic pressure** is generated by large molecules e.g. plasma proteins (PP) and adds to the forces that retain water within the vascular space.
  - Sodium moves freely between the vascular and interstitial spaces, but is actively extruded (forced out) from the intracellular space; therefore it is the principle extracellular cation ⓘ.
    - It is also the cation that we most frequently administer by giving normal saline (NaCl). When we do this, we **increase extracellular tonicity** and water must move from the intracellular space to the extracellular space to normalize osmolality
  - Electrolytes are exchanged between compartments, if an electrolyte increases in the blood, it will move to the interstitial or intracellular space.
  - Some electrolytes move freely → they **diffuse** via gradients. But some need active transportation e.g. Na^+ moves freely extracellulary (between the blood and interstitium), but needs active transport (Na/K pump) to move intracellulary (because this is not its normal position)

ⓘ Cations are positively charged ions (e.g. Na+) due to loss of an electron (e-) and anions are negatively charged ions (e.g. Cl-) due to gain of an electrone (e-)

- **How to measure the osmolality of the blood:**
  - Its either you <u>measure and add all</u> active molecules, or
  - <u>Multiply</u> the amount of **sodium** in the blood by **2** and you <u>add</u> amount of **urea** and **glucose** in blood → **[(sodium x 2) + urea + glucose]**
  - We measure it to know if the fluid is isotonic, hypotonic or hypertonic
  - In conditions such as *hypernatremia, renal failure (raised urea) or hyperglycemia*, osmolality is raised.

📄 Because sodium is the major extracellular **cation**, the majority of extracellular **anions** will be equal to its concentration.

2 TYPES OF IV FLUIDS

2.1.1 COLLOID SOLUTIONS

- They are fluids with high molecular weight, or contain protein. Used for volume expanding (e.g. in hypotension), or protein replacement (e.g. when albumin is low). They do not contain electrolytes. Examples: Dextran, hetastarch, albumin…
- They tend to stay within the vascular space and increase intravascular pressure.
- Colloids are used as a volume expander not for electrolyte imbalance or a physiological condition, just for volume depletion (hypotension) or low albumin.

2.1.2 CRYSTALLOID SOLUTIONS

- They're IV fluids that contain varying concentrations of electrolytes (water and minerals e.g., sodium, potassium, calcium, chloride).
- They are not given to a patient with hypoalbuminemia because they don't contain proteins.
- They come in different preparations and volumes.
- It's important to know the crystalloid's osmolality ⓘ related to the blood's
- Crystalloid solutions are classified according to their tonicity ⓘ into 3 categories:
  1) **Isotonic**: almost equal tonicity to the plasma, such as:
     - **Normal saline**: it is the commonly-used term for a solution of 0.9% weight/volume of NaCl, about 300 mOsm/L or **9.0 g per liter**.
       - 1 liter of normal saline contains 154 mmol/L of Na and 154 mmol/L of Cl only.
     - **Lactated Ringer's solution**: 1 liter contains: 130 mmol/L of Na, 109 mmol/L of Cl, 28 mmol/L of lactate, 4 mmol/L of K, 1.5 mmol/L of Ca
     - **Hartmann's solution:** 1 liter contains: 131 mmol/L of Na, 111 mmol/L of Cl, 29 mmol/L of lactate, 5 mmol/L of K, 2 mmol/L of Ca
  2) **Hypotonic**: have lesser tonicity than plasma, e.g. 2.5% dextrose
  3) **Hypertonic**: have greater tonicity than plasma, e.g. D5 NaCl

ⓘ Osmolality is the dissolution of a solute in whole blood measured in kilograms.
Normal blood osmolality = 280-303 miliosmoles/kg

ⓘ Tonicity of a solution Means effective osmolality in relation to plasma (=285 milliosmol/L).

Type of fluid*	Sodium (mmol/L)	Potassium	Chloride	Osmolarity
Plasma	136 -145	3.5 – 5.0	98 -105	**280 - 300**
5% Dextrose	0	0	0	**278**
Dextrose 0.18% saline	30	0	30	**283**
0.9% "normal" saline	***154***	***0***	***154***	***308***
0.45%"half normal" saline	***77***	***0***	***77***	***154***
Ringer's lactate	***130***	***4***	***109***	***273***
Hartmann's	***131***	***5***	***111***	**275**
Gelatin 4%	145	0	145	**290**
5% albumin	150	0	150	**300**
20% albumin	-	-	-	**-**
Hes 6% 130/0.4	154	0	154	**308**
Hes 10% 200/0.5	154	0	154	**308**
Hes 6% 450/0.6	154	0	154	**308**

ⓘ Ringer's lactate and Hartmann's are very similar but not identical.

The only difference is that Hartmann's contains 131 mmol/L of Na and Ringer's contains 130 mmol/L.

3 FLUID REQUIREMENTS

- Fluid losses in disease and in health are those that can be seen and measured, while insensible losses cannot be measured.
- Any fluid lost from the body is potentially in need of replacement, be it urine, stool, or fluid from drains, or other tubes. If possible, measuring these losses is a great help.
- The aim of fluid administration is the maintenance of organ perfusion by keeping total body water at 55 - 60% - this is the euvolemic state.
- **Hypovolemia**, when total body water is deficient, is not compatible with normal organ perfusion. Causes of hypovolemia include
  - GI: diarrhea, vomiting, etc.
  - Renal: diuresis
  - Vascular: hemorrhage
  - Skin burns
- **Hypervolemia**, when body water is in excess, is occasionally necessary for organ perfusion, but is usually harmful. Causes of hypervolemia include:
  - Heart /liver/kidney failure
  - Iatrogenic

In order to assess how much fluid should be given to someone, we need to know what their level of hydration is, what losses they may expect, and what gains they may receive (oral intake: fluids, nutritional supplements, bowel preparations – or IV intake: colloids & crystalloids, feeds, drugs); **you have to calculate the amount of fluid** the person needs before giving him IV fluids.

- **Normal adult requires approximately 35 cc/kg/d**

3.1 HOW TO CALCULATE FLUID REQUIREMENTS

- **Fluid requirements = normal requirement + amount of lost fluid per day + insensible loss**
  - Normal fluid needed = body weight x 35
  - You should know if the person has diarrhea or any disease to know how much fluid he has lost.
    - Fever increases insensible loss by 200 cc/day for each degree (C)
    - Monitor abnormal GI loss e.g. NGT suctioning (nasogastric tube).
  - Insensible water loss **makes up about 500 ml a day**. It is the amount of fluid lost on a daily basis from the lungs, skin, respiratory tract, and water excreted in the feces. *The exact amount cannot be measured.*

	Volume (ml)	Na+ (mmol)	K+ (mmol)
Urine	2000	80	60
Insensible losses (skin and respiratory tract)	700	--	--
Faeces	300	--	10
Minus endogenous Water	300	--	--
Total	2700	80	70

Figure 3: Fluid losses

3.1.1.1 HOW TO MEASURE HOW MUCH FLUID SHOULD BE GIVEN IN AN HOUR

- There are 2 rules:
  - Either you find out the normal fluid required and divide it by 24 hours.
  - Or Apply the "4, 2, 1" rule.
    - You give 4 cc/kg/hr for the first 10 kg
    - You give 2 cc/kg/hr for the second 10 kg
    - You give 1cc/kg/hr for each additional 10 kg

Example: How much fluid does a 100 kg male require?
- Daily requirement: 35 cc/kg → 35x100 = 3500 cc/day
- IV fluid rate (per hour), there are 3 methods:
  1) **Divide** 3500 by 24 = 140 cc/hr
  2) **"4, 2, 1" rule:**
     - 1st 10 kg x 4 = 40
     - 2nd 10 kg x 2 = 20
     - 3rd 10 kg x 1 = 10
     - 4th 10 kg x 1 = 10, 5th 10 kg x 1 = 10, 6th 10 kg x1 = 10,…. 10th kg x 1 = 10
     - Total = 40 + 20 + (10 x 8) = 40 + 20 + 80 = 140 cc/hr
  3) **Using the "4, 2, 1" rule**, we can conclude that: body weight + 40 = IVF rate
     - 100 + 40 = 140 cc/hr
     - How does this work? According to 4, 2, 1 rule:
       - You need 40 ml for first 10 kilograms, but you used 1 ml/kg = 1 x 10 = 10 ml → 40 – 10 = 30, so you need to **add 30 ml**
       - You need 20 ml for second 10 kilograms, but you used 1 ml/kg = 1 x 10 = 10 ml → 20 – 10 = 10, so you need to **add 10 ml**
       - Total you need to add is 30 (for 1st 10 kg) + 10 (for 2nd 10 kg) = 40
       - **So body weight + 40 = fluid requirement as calculated by 4, 2, 1 rule**

- This assumes no significant renal or cardiac disease and NPO [nil per os (nothing by mouth)]
- This is the maintenance IVF rate; it must be adjusted (**increased**) for any dehydration or ongoing fluid loss.
- Conversely, if the patient is taking fluids PO (by mouth), the IVF rate must be **decreased** accordingly.
- **Daily** electrolytes, BUN, creatinine, input/output, and if possible, weight should be monitored in patients receiving significant IVF. (*BUN: blood urea nitrogen*)

4 ELECTROLYTES REQUIREMENTS

4.1.1 SODIUM REQUIREMENT

Na^+ required: 1-3 mEq/kg/day

- 0.45% saline (half normal saline) contains 77 mEq NaCl per liter.
- 0.45% saline is usually used as maintenance of IV fluid assuming there are no other volume or electrolyte issues.

Half-normal saline (0.45%) **will** result in hyponatremia if given rapidly or in excess amounts. Maintenance therapy should be tailored to the patient's specific requirements.

Example 1:
- 70 kg male requires 70 - 210 mEq NaCl in 2600 cc fluid per day.
- In such case, you give the patient half normal saline. Why?
  - The patient needs 70 – 210 mEq NaCl in 2.6 L a day,
  - The half normal saline contains 77 mEq NaCl per liter
  - When you measure it: 77 x 2.6 = 200 mEq, It meets the daily requirement of the patient.
  - Unlike giving normal saline which contains 154 mEq NaCl per liter.

Example 2:
- Patient weighs 100 kg, requires100 to 300 mEq NaCl in 3500 cc /d.
- In such case, you give the patient half normal saline. Why?
  - The patient needs 100 to 300 mEq NaCl in 3.5 L a day,
  - The half normal saline contains 77 mEq NaCl per liter
  - When you measure it: 3.5 L x 77 = 269.5 mEq, It meets the daily requirement of the patient.
  - Unlike giving normal saline which contains 154 mEq NaCl per liter.3.5x154 it will be 539, it will exceed the amount needed.

4.1.2 POTASSIUM REQUIREMENT

K+ required: 1 mEq/kg/day

- K+ can be added to IV fluids. Remember this increases the osmolality load.
  - **20 mEq/L** is a common IVF additive.
- The potassium flow rate **shouldn't exceed 10-20** mmol/hour because it might cause hyperkalemia.
- If significantly hypokalemia occurs, order separate K+ supplementation.
- **Oral** potassium supplementation is always preferred when feasible.
- The most important surgical abnormality is hypokalemia because they always give fluids but not K+.

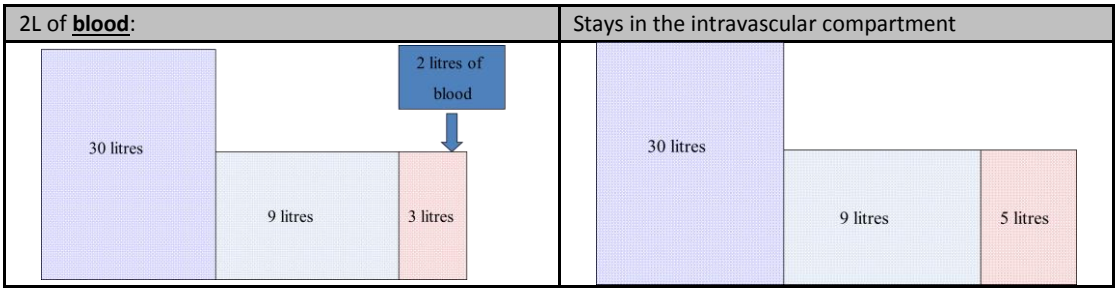
Example 1:
- 70 kg male
  - First you measure the amount of fluid the patient needs per day.
  - Then you measure the amount of potassium the patient needs, which is 70 mEq/kg/day of K+.
  - After that you'll add the amount of K+ the patient needs to the fluid you chose to give the patient.
  - You'll divide it into 20 mEq/L
  - This will supply basal needs in most patients who are NPO.

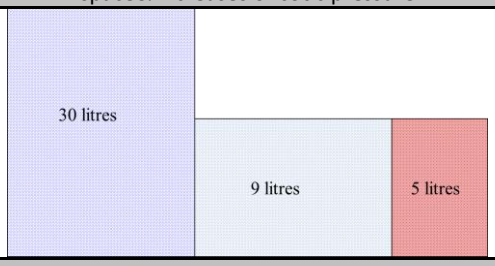
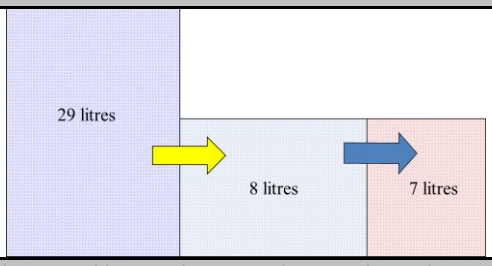
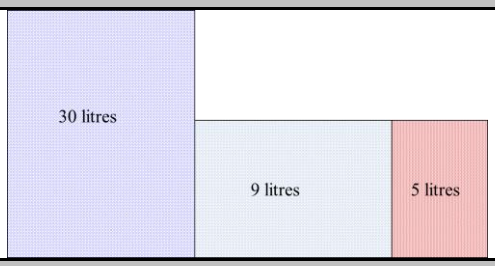
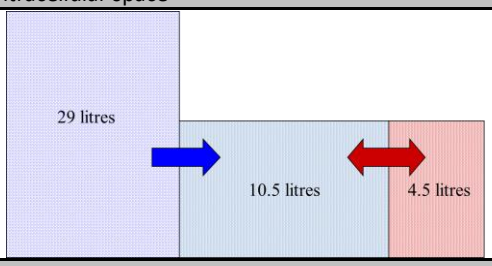
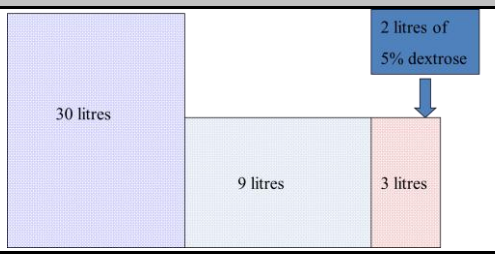
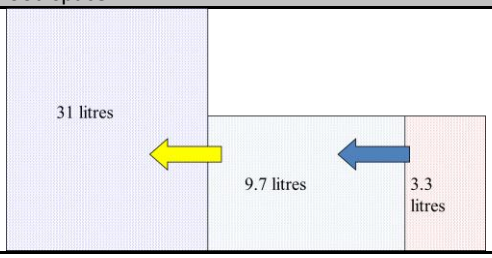
5 RULES OF FLUID REPLACEMENT

- Someone with serious intravascular volume depletion, hypotension and reduced cardiac output is in a shock, whether it was caused by blood loss (e.g. hemorrhage), plasma loss (e.g. major burns), or water loss.
- The aim here is to **restore intravascular volume** with a fluid that remains in the vascular compartment, and may even draw water from the intracellular space into the blood system. A fluid with a high oncotic pressure would do this job.
  - Blood remains the fluid of choice to treat someone with blood loss.

  - Colloid is the fluid of choice in resuscitation when blood loss is not pronounced, or whilst waiting for blood.
  - Any **crystalloid** will enter the vascular space, then distribute around the other compartments. By containing sodium, the main extracellular cation, saline will expand the **interstitial and intravascular** compartments more than dextrose will, most of which will enter the intracellular space.
- The right treatment for blood loss is to replace it with blood. Giving 2 liters of blood to someone will expand their intravascular compartment by 2 liters. None of this fluid will escape across the blood vessel walls (in the short term at least) and the other compartments are unaffected.
- Giving colloid into the vascular space results in an immediate expansion of the intravascular compartment by 2 liters (like blood).
  - Colloid does not escape from the vascular space, but does increase oncotic pressure markedly **causing water to be drawn into the vascular space** from the interstitial and intracellular reservoirs. Giving colloid not only expands the vascular space itself, but does so by moving water from other spaces
- Saline being a **crystalloid, does not remain within the vascular space**, but will diffuse into the interstitial space. The sodium it carries will not enter the intracellular space however, because of active sodium extrusion from the cell.
  - Saline will cause **immediate expansion** of the intravascular volume, **followed by equilibration** between the vascular and interstitial spaces, the osmolality of which are equal, but are now slightly greater than that of the intracellular space, due to the increased sodium load. This results in water movement from the intracellular space in order to equalize osmolality throughout all three compartments.
- 5% **Dextrose** is isotonic to plasma. Giving 2 liters of 5% dextrose will cause the immediate expansion of the vascular compartment but as its glucose content is rapidly metabolized, the remaining water will distribute itself between all compartments and **very little will remain within the blood space**. For this simple reason, dextrose is not a fluid of resuscitation.
- In summary:
  a. Replace plasma with colloid
  b. Replace ECF (extra-cellular fluid) depletion with saline
  c. Dextrose should be given in case of dehydration.
  d. The only thing that goes intracellulary is the potassium.

Figure 4: Effects of different types of IV fluids



2L of **colloid**: immediate expansion of intravascular space& increases oncotic pressure	+ draws water from other compartments
	
2L of **crystalloid**: immediate expansion of vascular compartment	Then: equilibration between the vascular and interstitial spaces, and draws water from intracellular space
	
2L of 5% **dextrose**: immediate expansion of vascular compartment	Then: glucose content is rapidly metabolized, remaining water will distribute itself between all compartments and very little will remain within the blood space
	

6 ABNORMALITIES

6.1 WATER

6.1.1 WATER EXCESS

- **Causes**:
  - Inappropriate use of hypotonic solutions such as 0.5% water leading to hypo-osmolar hyponatremia (iatrogenic)
  - Syndrome of inappropriate ADH (anti diuretic hormone) secretion.
    - Inappropriate secretion of ADH has many causes, such as: malignant tumors, CNS diseases, pulmonary disorders (pneumonia, lung abscess), medications, and severe stress e.g. major surgery)
- **Signs and symptoms:**
  - Symptoms of water excess develop slowly and if not recognized and treat ed promptly, they become evident by **convulsions** and **coma** due to **cerebral edema**.
  - Signs include: hypertension, tachycardia, raised JVP/ gallop, edema, pleural effusions, pulmonary edema, ascites and organ failure.

Box: Antidiuretic Hormone (ADH):

- ADH is released from the posterior pituitary gland.
- It is secreted in response to **high osmolality** in plasma or in response to **low volume.**
  - ADH secretion is influenced by volume receptors so that hypovolemia stimulates ADH secretion and water reabsorption. In the paradoxical situation where hypovolemia is accompanied by a fall in osmolality, ADH secretion will increase because the major stimulant is hypovolemia
- Role of ADH: to maintain normovolemia and the osmolality of plasma
  - The principle mechanism by which osmolality is maintained: changes in ADH secretion from the posterior pituitary.
- Anti-diuretic hormone secretion results in:
  - **Pure water reabsorption** from the collecting duct of the nephron via a pathway that involves the V2 receptor and aquaporin 2.
  - It increases the urine's concentration

- **Diagnosis** is established when **urine sodium >20 mEq/L** (in the absence of renal failure, hypotension, and edema)
- **Treatment**:
  - Water restriction by giving the patient <1 L/day, infusion of isotonic or hypertonic saline solution and use of
  - ADH- antagonist (Demeclocycline 300-600 mg b.i.d).

6.1.2 WATER DEFICIT:

- The most encountered derangement of fluid balance in surgical patients is **hypovolemia**.
- **Causes** include: bleeding, third spacing ⓘ, gastrointestinal losses, increase insensible loss (normal ≈ 10ml/kg/day), and increase renal losses (normal ≈ 500-1500 ml/day).
- **Signs and symptoms:**
  - Symptoms: thirst, dryness, lethargy, and confusion.
  - Signs:
    - Dry tongue and mucous membranes, sunken eyes, dry skin, loss of skin turgor, collapsed veins,
    - postural hypotension, Tachycardia, absence of JVP at 45o
    - Oliguria, organ failure
    - Depressed level of consciousness, and coma
- **Diagnosis**: confirmed by increased serum sodium (>145mEq/L) and increased serum osmolality (>300 mOsmol/L)
- **Treatment**:
  - Bleeding should be replaced by **IVF initially then by whole blood** or packed red cells depending on hemoglobin level. Each blood unit will raise the hemoglobin level by 1 g.
  - If fluid loss was caused by diarrhea or vomiting you give IVF (crystalloid usually).
  - If **sodium** is **low**, give a solution that contains sodium (e.g. 0.9% NaCl)
  - If **sodium** is **>145** mEq/L give **0.45% hypotonic** saline solution
  - If sodium is **>160** mEq/L give **D5**% water cautiously and slowly (e.g. 1 liter over 2-4 hours) in order not to cause water excess
  - You should treat anything that causes further water loss.

ⓘ Third spacing is the shift of fluid from the intravascular space to a nonfunctional space, or the loss of extracellular fluid from the vascular to other body compartments.

        - Third spacing replacement can be estimated within a range of 4-8 ml/kg/h.
        - Gastrointestinal and intraoperative losses should be replaced 1 cc/cc loss
    - IVF maintenance can be roughly estimated by 4/2/1 rule.

6.2 SODIUM

6.2.1 HYPERNATREMIA

- **It is established when serum sodium >145 mEq/L**
- **Causes** :
    1) **Excessive sodium load** (excessive normal saline (0.9%) or hypertonic solutions e.g. 3% NaCl - >145 of Na)
    2) Hyperaldosteronism; aldosterone promotes water & Na+ retention (rare)
    3) **Reduced water intake** by fasting, nausea and vomiting, or reduced consciousness as in Alzheimer's patient/elderly (they forget to drink)
    4) **Increased water loss** by sweating (pyrexia, hot environment), respiratory tract loss (increased ventilation, administration of dry gases) or burns.
    5) Inappropriate **urinary water loss** by diabetes insipidus (pituitary or nephrogenic) or diabetes mellitus
    6) Patients with **CHF**, **cirrhosis**, and **nephrotic** syndrome are prone to this complication
- **Symptoms**: similar to water excess symptoms, includes coma, convulsions and confusion.
- **Treatment** :
    - Water restriction and ↓ sodium infusion in IVF (e.g. 0.45% NaCl or D5% water).

6.2.2 HYPONATREMIA

- **Causes**:
    1) Hyperglycemia (it could be Pseudohyponatremia; diabetic)
        - Corrected Na+ = BS mg/dl x 0.016 + P (Na) (BS = blood sugar)
    2) Excessive IV sodium-free fluid administration (*hypotonic solutions*)
    3) Hyponatremia with volume overload "**hyper**volemic **hypo**natremia" usually indicates impaired renal ability to excrete sodium.
- **Treatment**:
    - Administering the calculated sodium needs in isotonic solution
    - In severe hyponatremia (Na+ <120 mEq/L) you give a hypertonic solution
    - Serum Na+ administration shouldn't be given at a rate > **10-12 mEq/L/hr,** because rapid correction may cause permanent brain damage due to the osmotic **demyelination** syndrome ⓘ
    - Before treating hyponatremia, you should check if it's true hyponatremia or pseudohyponatremia by checking the glucose levels.
        - The glucose levels should be corrected in case of pseudo-hyponatremia, no further treatment is needed.

ⓘ **Pseudo-hyponatremia**: low serum sodium concentration resulting from volume displacement by massive hyperlipidemia or hyperprotienemia or by hyperglycemia.

6.3 POTASSIUM

6.3.1 HYPERKALEMIA

- **Causes**:
  - Increase K+ infusion in IVF
  - Tissue injury, surgery
  - Metabolic acidosis (causes a shift of potassium from intracellular space into extracellular space)
  - Renal failure (↓ excretion)
  - Blood transfusion (RBCs contain high concentrations of K+)
  - Hemodialysis
- **Signs & symptoms:** arrhythmia ⓘ
- **Diagnosis**: established by ↑ serum K+ **>6 mEq/L** and **ECG changes** (bradycardia and peaked T wave)
- **Treatment**:
  - Insulin: 10 IU (shifts K+ back into intracellular compartment) + glucose: 1 ampule of Dextrose 50% (prevent hypoglycemia from insulin) – over 15 minutes
  - Calcium oxalate enemas (can also be given orally)
  - Lasix 20-40 mg IV
  - Dialysis (if needed)

6.3.2 HYPOKALEMIA

- Occurs when serum K+ **<3 mEq/L**
- The most common surgical abnormality.
- **Causes**:
  1) Inadequate replacement (e.g. during surgery)
  2) Diuretics (e.g. Lasix)
  3) Metabolic alkalosis (shifts K+ to intracellular compartment)
  4) Hyperaldosteronism (promotes K+ excretion in kidneys)
  5) Gastrointestinal tract losses:
     - Vomiting
     - Gastric aspiration/drainage (the flow of gastric content into the upper respiratory tract due to a ↓ antireflux reflex)
     - Fistulae (an abnormal connection between an organ, vessel, or intestine and another structure. It's usually the result of injury, surgery, infection or inflammation.)
     - Diarrhea
     - Ileus (disruption of the normal propulsive gastrointestinal track that causes obstruction which prevents bowel contents, such as stool, fluid and gas, from moving through the intestine, which becomes distended)
     - Intestinal obstruction
     - Potassium-secreting villous adenomas
  6) Urinary loss
  7) Renal tubular disorders (e.g. *Bartter syndrome*, renal tubular acidosis, amphotericin-induced tubular damage)
- **Symptoms**: 📄 weakness and fatigue (most common), muscle cramps and pain (severe cases), altered level of consciousness, arrhythmias
- **Treatment**: K+ replacement (KCl solution)

6.4 CALCIUM

6.4.1 HYPERCALCEMIA

- **Causes**: *hyper*parathyroidism and malignancy.
- **Symptoms**: confusion, weakness, lethargy, anorexia, vomiting, epigastric abdominal pain (due to pancreatitis), and polyuria (due to nephrogenic diabetes insipidus).
- **Diagnosis** is established by measuring the **free Ca >10 mg/dl.**
- **Treatment** includes normal saline infusion
  - If Ca >14mg/dl with ECG changes: additional diuretics, calcitonin, and mithramycin (antineoplastic antibiotic that has been discontinued) might be necessary

6.4.2 HYPOCALCEMIA

- **Causes**: *hypo*parathyroidism after thyroid or parathyroid surgeries, other less common causes include:
  - pancreatitis (depletion of Ca due to saponification of fat),
  - necrotizing fasciitis,
  - high output GI fistula, and
  - massive blood transfusion (citrate in the blood binds Ca)
  - Low vitamin D
  - Pseudo-hypocalcemia (low albumin and hyperventilation)
    - Hypoalbuminemia; the relation between Ca^{++} and albumin is the binding of the Ca^{++} to albumin which makes the calcium less free. In hypoalbuminemia total calcium is ↓ while ionized is unaffected.
    - Hyperventilation → respiratory alkalosis → ↑ Ca binding & ↓ free Ca
- **Symptoms**:
  - **Numbness** and tingling sensation circumorally or at the finger-tips.
  - **Tetany** and seizures may occur at a very low calcium level.
- **Signs** include tremor, hyperreflexia, carpopedal spasms and positive Chvostek sign.
- **Diagnosis**: serum Ca **< 8.5 mg/dl** (2.1 mmol/L)
- **Treatment**: should start by treating the cause. Calcium supplementation with calcium gluconate or calcium carbonate IV or orally. Vitamin D supplementation especially in chronic cases.

Figure 5: Carpopedal spasm

Figure 6: Chvostek sign; tapping the zygomatic arch causes the facial muscles to twitch

6.5 MAGNESIUM

6.5.1 HYPERMAGNESEMIA

- Mostly occurs in association with renal failure, when Mg+ excretion is impaired.
- The use of antacids containing Mg+ may aggravate hypermagnesaemia.
- Treatment includes rehydration and renal dialysis
- Hypermagnesemia and hypophosphatemia are all conditions of renal failure

6.5.2 HYPOMAGNESAEMIA

- Usually there are no symptoms but when you want to correct Ca or K levels they don't get corrected.

- Mg plays a role in the pumps and the muscular junctions
- When Mg decreases the Ca and K will decrease as well even though they were in their normal levels.
- But when correcting the magnesium everything will go back to normal
- The majority of magnesium is intracellular with only <1% in the extracellular space.
- It happens from inadequate replacement in depleted surgical patients with major GI fistula and those on TPN.
- Magnesium is important for neuromuscular activities. (cannot correct K nor Ca)
- In surgical patients hypomagnesaemia is a frequently missed common electrolyte abnormality as it causes no major alerting symptoms.

6.6 PHOSPHATE

6.6.1 HYPERPHOSPHATEMIA

- Mostly associated with renal failure and hypocalcaemia due to hypoparathyroidism, which reduces renal phosphate excretion.

6.6.2 HYPOPHOSPHATEMIA

- **Causes**:
  - Inadequate intestinal absorption,
  - Increased renal excretion,
  - Hyperparathyroidism,
  - Massive liver resection
  - Inadequate replacement after recovery from significant starvation and catabolism.
- **Symptoms**: muscle weakness and inadequate tissue oxygenation due to reduced 2, 3- bisphosphoglycerate levels.
- Early recognition and replacement will improve these symptoms.

> *Mg and phosphate abnormalities occur with chronic diseases, before replacing them check the renal system, caused all the time by renal failure*

7 ACID BASE BALANCE

> *When the H^+ concentration increases, the pH value will decrease and blood will become acidic. If the H^+ concentration decreases the blood will become alkaline.*

ⓘ Normal values:
pH = 7.36 – 7.4
H^+ concentration = 36 – 40 mmol/L
$PaCO_2$ ~ 40 mmHg
Bicarbonate con-centration **$[HCO3^-]$** = 20-28, average 24 mmol/L

- Hydrogen ion (mainly intracellular) is generated in the body by:
  1) Protein and CHO metabolism (1 mEq/kg of body weight)
  2) Predominant CO2 production
- Mechanisms to maintain the normal value of pH in the intracellular fluid:
  1) **Proteins** which include hemoglobin : Protein buffers include basic group, and acidic protein buffer groups, that act as hydrogen ion depletors or donors to maintain the pH level at 7.4
  2) **Phosphate**: when H concentrations increase, it binds to H ions and is excreted in the urine with sodium
- Mechanisms to maintain the normal value of the PH in the extracellular fluid:
  - **The buffer system**: bicarbonate/carbonic acid system:

- pH levels depend on CO2 and HCO3 mainly
  $H^+ + HCO_3^- \leftrightarrow H_2CO_3 \leftrightarrow CO_2 + H_2O$
- Hydrogen ions and the bicarbonate form carbonic acid which forms CO_2 and water under the enzyme carbonic anhydrase.
- So if hydrogen ions increase in the plasma, CO_2 production will increase therefore the pH will decrease.
- **Respiratory compensation**: In acidosis, pH changes will stimulate the respiratory center in the brain stem → hyperventilation → PCO_2 will decrease and the pH levels will get back to normal.
- **Metabolic compensation**: when acid accumulates: the kidneys increase urinary excretion of acids and reabsorption of bicarbonate (in the proximal tubules in the kidneys).

8 ACID BASE DISORDERS

Normal values for anion gap vary according to the source.

8.1.1 METABOLIC ACIDOSIS

- Low pH due to H+ ions accumulation and HCO_3 ions decrease
- **Causes**:

To know the cause of metabolic acidosis you have to calculate the anion gap:
- **AG = Cations (Na + K) – Anions (Cl + HCO_3)**
- Normal value is **12 mmol** (8-16)

  - High anion gap (AG >16):
    - **Lactic acidosis** caused by shock (any cause), severe hypoxemia, severe hemorrhage/anemia, liver failure
    - **Diabetic ketoacidosis**
    - Acute or chronic renal failure
    - Poisoning (ethylene glycol, methanol, salicylates)
  - Non-anion gap (AG = 8-12):
    - Increased bicarbonate loss by **diarrhea**, intestinal fistulae, renal tubular acidosis (types I-IV)

8.1.2 METABOLIC ALKALOSIS

- High HCO_3 → high pH
- **Causes**: (1) **H+ ions loss** (vomiting, NGT, Lasix) (2) Hypokalemia (3) **HCO_3 retention**.

If you lose K, you will get alkalosis. If you gain you will get acidosis.

Vomiting, NGT: Gastric secretions are rich in HCl. The secretion of HCl by the stomach usually stimulates bicarbonate secretion by the pancreas.

Diuretics cause chloride depletion and by increased delivery of sodium ions to the collecting duct, which enhances potassium ion and hydrogen ion secretion

Hypokalemia when alone without hyper-aldosteronism causes only mild alkalosis

8.1.3 RESPIRATORY ACIDOSIS

- **Causes**: (anything that causes hypoventilation)
  - Common surgical causes of respiratory acidosis
  - Central respiratory depression
  - Opioid drugs
  - Head injury or intracranial pathology
  - Pulmonary disease
  - Severe asthma
  - COPD
  - Severe chest infection

8.1.4 RESPIRATORY ALKALOSIS

- **Causes**: (anything that causes hyperventilation):
  - Pain
  - Apprehension/hysterical hyperventilation
  - Pneumonia
  - Central nervous system disorders(meningitis, encephalopathy)
  - Pulmonary embolism
  - Septicemia
  - Salicylate poisoning
  - Liver failure

Type of A- B disorder	**Acute (Uncompensated)**			**Chronic (Partially compensated)**		
	PH	PCO2	HCO3	PH	PCO2	**HCO3**
Respiratory acidosis	↓↓	↑↑	Normal	↓	↑↑	↑
Respiratory alkalosis	↑↑	↓↓	Normal	↑	↓↓	↓
Metabolic acidosis	↓↓	Normal	↓↓	↓	↓	↓
Metabolic alkalosis	↑↑	Normal	↑↑	↑	↑	↑

9 MCQ'S

1) What is the composition of 0.9% Saline?
   a) 130 mEq sodium, 109 mEq chloride, 28 mEq lactate.
   b) 154 mEq sodium, 154 mEq chloride.
   c) 513 mEq sodium, 513 mEq chloride.
   d) 855 mEq sodium, 855 mEq chloride.
2) Which of the following is correct regarding the composition of the body fluid compartments?
   a) The major intracellular cation is sodium.
   b) The major intracellular anions are proteins and phosphates.
   c) The major extracellular cation is potassium.
   d) The major extracellular anion is magnesium.

🗝 Answers: 1:b, 2:b

BLOOD & BLOOD PRODUCTS TRANSFUSION

1 INTRODUCTION

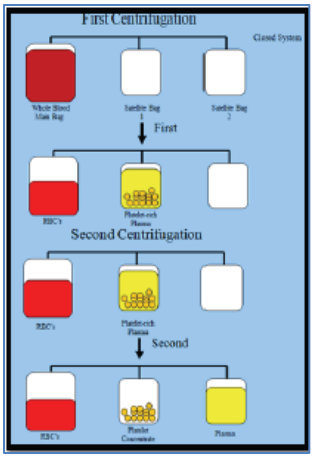
1.1 HISTORY OF TRANSFUSIONS

- Blood transfused in humans since mid-1600's
- 1828 – First successful transfusion
- 1900 – Landsteiner described ABO groups
- 1916 – First use of blood storage
- 1939 – Levine described the Rh factor

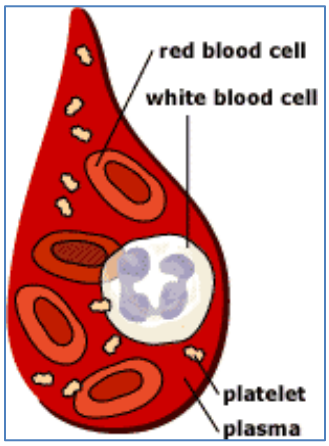
1.2 OBJECTIVES

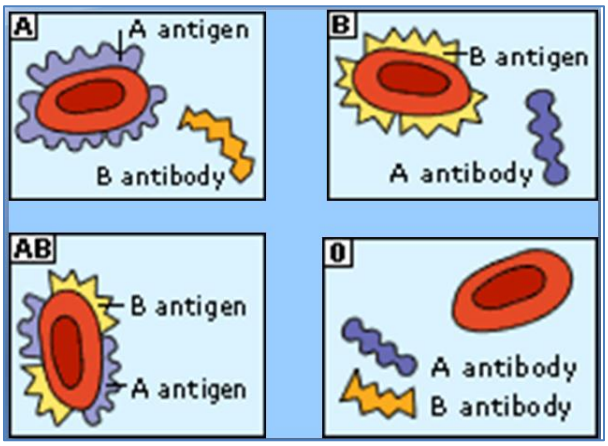
- Blood components
- Indications for transfusion
- Safe delivery
- Complications

1.3 BLOOD COMPONENTS ⓘ



- Prepared from whole blood collection
- Whole blood is separated by differential centrifugation
  - Red Blood Cells (RBCs)
  - Platelets
  - Plasma
    - Cryoprecipitate
    - Others (include Plasma proteins—IV Ig, Coagulation Factors, albumin, Anti-D, Growth Factors, Colloid volume expanders)
  - WBCs
- Differential centrifugation
  - First Centrifugation > Seperates RBCs from platelet rich plasma
  - Second Centrifugation > Seperates platetlets from plasma





2 TYPES OF BLOOD TRANSFUSIONS

- Indication to use WBCs: (granulocyte) transfusion for patients with severe leukocytosis or immunocompromised who don't respond to antibiotics
- Important notes:
  - Why do we need RBCs for anemic patients? Because RBCs have Oxyegn carrier capacity "we notice shortness of breath and tachycardia in anemic patients".
  - Whole blood transfusion is excellent when it's fresh, but if it's frozen (storage) there will be risk of coagulopathy.
  - Storage of whole blood hypocalcaemia (precipitate Ca^{+2}) + affect coagulation factors especially factor V & VIII. ⓘ
  - In surgery, they order RBCs + plasma + platelets
  - Platelets, fresh frozen plasma and cryoprecipitate are given to improve hemostasis.
  - Before going through with the blood transfusion, we take a blood sample from the donor to:
    - Do screening test to avoid transmitted infection (viruses)
    - Blood group ABO testing (do it in RBCs, whole blood but not in plasma or platelets)
    - Cross match test (Rh test) [If there is a Rh group antigen, it is (+ve), if there is not then it is (-ve)]

Because of the high risk of infections in blood transfusions, we should not give blood if unnecessary; instead, we give colloids or crystalloids (volume expanders) to compensate the volume loss.

Blood is only given when the Hb level is low (to increase O carrying capacity) e.g. Anemia > its earliest sign > Resting tachycardia

2.1.1 WHOLE BLOOD

- **Storage:**
  - 4° centigrade for up to 35 days, transported in 1-10 centigrade ⓘ
  - With Mannitol, Adisol or Nutrisol, it lives up to 42 days
- **Indications**ⓘ
  - Massive Blood Loss / Trauma / Exchange Transfusion (thalassemia, sickle cell anemia) - (patient needs volume expansion)
- **Considerations**
  - Use filter as platelets and coagulation factors will not be active after 3-5 days
  - Donor and recipient must be ABO identical ⓘ
- **Why should we consider filter in blood transfusion?**
  - To avoid any clot or debris and transmission of a virus
  - We do it for whole blood, FFP and RBC.
  - Platelets do not have to undergo filtering otherwise they will be damaged.
  - Filter size = 170 micro
  - In case of immunocompromised patients or in severe infections, the filter size is usually = 20-40 micro

2.1.2 RBC CONCENTRATE (PACKED)

- **Storage**
  - 4° for up to 42 days, can be frozen
- **Indications**
  - Many indications—i.e. anemia, hypoxia, mild bleeding,..etc.

Packed cells should never be given directly, they must be diluted with saline (imagine packed cells as a bag of honey which is hyperosmolar)

- **Considerations**
  - Recipient must not have antibodies to donor RBC's (note: patients can develop antibodies over time) > identical ABO
  - Usual dose 10 cc/kg (will increase Hb by 2.5 gm/dl)
  - Usually transfuse over 2-4 hours (slower for chronic anemia)
- Also, never dilate the cells with Ringer's lactate. It has Ca^{+2} and that kills the RBCs.
- Remember that the life span of RBCs depends on the preservative used.

2.1.3 PLATELETS

- **Storage**
  - Up to 5 days at 20-24° (its half-life is 1-7 days)
- **Indications**
  - Thrombocytopenia, Plt <15,000
  - Bleeding and Plt <50,000 (because it increases volume expansion)
  - Invasive procedure and Plt <50,000 (because it increases volume expansion)
- **Considerations**
  - Contain Leukocytes and cytokines
  - 1 unit/10 kg of body weight increases platelet count by 5000-10000 ⓘ
  - Donor and recipient must be ABO identical
  - Platelets are stored at room temperature, so no need to warm it. (Unlike whole blood & RBCs, which we have to warm up to avoid hypothermia)
  - Platelets don't need warming, filtering (it will inactivate it and damage it) or ABO testing.

2.1.4 PLASMA AND FFP

- **Contents—**Coagulation Factors (1 unit/ml)
- **Storage**
- **FFP:**
  - 12 months at 18° or colder
  - We should give it to the patient in 2 hours when we order it because it has a short half-life.
- **Indications**
  - Coagulation factor deficiency, fibrinogen replacement, DIC, liver disease, exchange transfusion, massive transfusion
- **Considerations**
  - Plasma should be recipient RBC ABO compatible
  - In children, should also be Rh compatible
  - Usual dose is 20 cc/kg to raise coagulation factors approximately 20%

2.1.5 LEUKOCYTE REDUCTION FILTERS

- Used for prevention of transfusion reactions
- Filter used with RBCs, Platelets, FFP and Cryoprecipitate
- Other plasma proteins (albumin, colloid expanders, factors, etc.) do not need filters
- NEVER use filters with stem cell/bone marrow infusions
- May reduce RBCs by 5-10%

3 RBC TRANSFUSIONS

3.1 PREPARATIONS

- **Typing**
  - Typing of RBCs for ABO and Rh are determined for both donor and recipient
  - If Rh antigen factor is present, it is (Rh +ve). If not, it is (–ve).
- **Screening**
  - Screen RBCs for atypical antibodies
  - Approximately 1-2% of patients have antibodies
  - Viral screening ⓘ
- **Crossmatching**
  - Donor cells and recipient serum are mixed and evaluated for agglutination to prevent hemolysis reaction after the transfusion
  - Duration: 30-45 minutes for the results to be ready
- In case of ER, we don't have enough time to do cross matching, so we just give the patient (O –ve)= universal donor.

3.2 ADMINISTRATION

- **Before administering the blood, we should check the blood pack for**ⓘ
  - Patient's name
  - Hospital number
  - ABO and Rh compatibility
  - Expiration date
  - Should be checked by 2 people in the operation room
- **The most common cause of death after blood transfusion is ABO incompatibility and the most common cause of that is a labeling error.**
- **Dose**
  - Usual dose of 10 cc/kg infused over 2-4 hours (slowly) >> to avoid massive blood transfusion
  - Maximum dose of 15-20 cc/kg can be given to hemodynamically stable patients
- **Procedure**
  - Use IV line of size 20 gage (or larger) cannula to prevent hemolysis and breakdown of RBCs, and we need to check if its working before we use it on the patient.
  - Warm the blood to prevent hypothermia > either by putting it in warm water or by a warming machine
  - May need Premedication (Tylenol and/or Benadryl) > first possible complication is increase in temperature (hyperthermia) so we give this medication to adjust it.
  - Filter use—routinely leukodepleted
  - Monitoring—VS q 15 minutes (vital signs every 15 minutes), clinical status
  - Do NOT mix with medications

4 TRANSFUSION COMPLICATIONS ⓘ

- **Complications**
  - Rapid infusion may result in Pulmonary edema, hypothermia, especially if it is not warmed
  - Transfusion Reaction
  - Transmitted diseases can be transmitted during blood transfusion

4.1 ACUTE TRANSFUSION REACTIONS

Adverse Effect	Frequency	Comments
Acute Hemolytic Rxn	1 in 25,000	Red cells only
Anaphylactic hypotensive	1 in 150,000	Including IgA
Febrile Nonhemolytic	1 in 200	Common
Allergic	1 in 1,000	Common
Delayed Hemolytic	1 in 2,500	Red cells only
RBC alloimmunization	1 in 100	Red cells only
WBC/Plt alloimmunization	1 in 10	WBC and Plt only

4.1.1 ACUTE HEMOLYTIC TRANSFUSION REACTIONS

- **MOST important complication**
- Occurs when incompatible RBCs (usually ABO or Rh) are transfused into a recipient who has pre-formed antibodies
- Antibodies activate the complement system, causing intravascular hemolysis > hemoglobinuria
- This hemolytic reaction can occur with as little as 1-2 cc of RBCs ⓘ
- Labeling error is the most common problem
- Can be fatal
- Symptoms occur within minutes of starting the transfusion and they include:
  - High fever/chills
  - Hypotension
  - Back/abdominal pain
  - Oliguria
  - Dyspnea
  - Dark urine
  - Pallor
- What to do? If an AHTR occurs ⓘ
  - STOP TRANSFUSION
  - ABCs
  - Maintain IV access and run IVF (NS or LR)
  - Monitor and maintain BP/pulse

- Give diuretics
- Obtain blood and urine for transfusion reaction workup
- Send remaining blood back to Blood Bank

- Blood Bank Work-up of AHTR ⓘ
  - Check paperwork to assure no errors
  - Check plasma for hemoglobin
  - Repeat crossmatch
  - Repeat blood group typing
  - Blood culture
- Monitoring in AHTR
  - Monitor patient clinical status and vital signs
  - Monitor renal status (BUN, creatinine)
  - Monitor coagulation status (DIC panel– PT/PTT, fibrinogen, D-dimer/FDP, Plt, Antithrombin-III)
  - Monitor for signs of hemolysis (LDH, bili, haptoglobin)

4.1.2 FEBRILE NON-HEMOLYTIC TRANSFUSION REACTIONS

- Definition--Rise in patient temperature >1°C (associated with transfusion without other fever precipitating factors)
- Occurs with approximately 1% of PRBC transfusions and approximately 20% of Plt transfusions
- What to do? If an FNHTR occurs
  - STOP TRANSFUSION
  - Use of Antipyretics—responds to Tylenol
  - Use of Corticosteroids for severe reactions
  - Use of Narcotics for shaking chills
- Future considerations:
  - May prevent reaction with leukocyte filter
  - Use single donor platelets
  - Use fresh platelets
  - Washed RBCs or platelets:
    - PRBCs or platelets washed with saline
    - Indicated to prevent recurrent or severe reactions
    - Washed RBC's must be used within 24 hours
    - RBC dose may be decreased by 10-20% by washing
- In short, stop transfusion check ABO compatibility and papers, if there are no precipitating facotrs, give Tylenol and continue the blood transfusion.

4.1.3 ALLERGIC NON-HEMOLYTIC REACTIONS

- Etiology
  - May be due to plasma proteins or blood preservatives/anticoagulants
- Presents with urticaria and wheezing
- Treatment ⓘ
  - Mild reactions—Can be continued after Benadryl
  - Severe reactions—Must STOP transfusion and may require steroids or epinephrine
- Prevention—Premedication (Antihistamines)

4.1.4 TRANSFUSION RELATED ACUTE LUNG INJURY

- Clinical syndrome similar to ARDS
- Occurs 1-6 hours after receiving plasma-containing blood products
- Caused by WBC antibodies present in donor blood that result in pulmonary leukostasis
- Treatment is supportive
- High mortality
- There will be damage to the alveoli by the donor's WBCs and antibodies, causing pulmonary leukocytosis. The lung will be white because of this reaction.

4.1.5 COAGULOPATHY WITH MASSIVE TRANSFUSIONS ⓘ

- Coagulopathy may occur after transfusion of massive amounts of blood (trauma/surgery) – giving the patient 1 unit of blood volume within 2 hrs (unit of blood volume = 70 ml/kg)
- Coagulopathy is caused by failure to replace plasma – stored blood has less coagulating factors, which leads to coagulopathy
  [Thrombocytopenia due to decreased platelets number compared to the increased blood vloume > causes more bleeding)
- Electrolyte abnormalities
  - Due to citrate binding of Calcium (hypocalcemia)
  - Also due to breakdown of stored RBCs
  - May cause metabloic acidosis (electrolyte imbalance)
- When these complications occur, you should start supporting the platelets (because they will decrease in number as well as the coagulation factors) and replace it.
- In case of trauma or major surgery where there is active bleeding, large amounts of blood will be transfused to the patient and as we said this will lead to massive transfusion, which in turn will cause diluted platelets and coagulation factors. So Platelets and Fresh Frozen Plasma should be ordered from the blood bank to prevent these complications. They're also ordered if the patient is a known case of thrombocytopenia or coagulation disorder.

Blood can be returned to the blood bank as long as it wasn't warm, whereas platelets and FFP should be used when they're ordered (so only order it when you need it).

4.1.6 BACTEREMIA

- More common and more severe with platelet transfusion (platelets are stored at room temperature)
- Organisms
  - Platelets—Gram (+) organisms, ie Staph/Strep
  - RBCs—Yersinia, enterobacter
- Risk increases as blood products age (use fresh products for immunocompromised)

4.2 CHRONIC TRANSFUSION REACTIONS

- Delayed, might take 2-20 days for the reaction to start
  - Alloimmunization
  - Transfusion Associated Graft Versus Host Disease (GVHD)
  - Iron Overload
  - Transfusion Transmitted Infection

4.3 TRANSFUSION ASSOCIATED INFECTIONS ⓘ

- Hepatitis C
- Hepatitis B
- HIV
- CMV: CMV can be diminished by leukoreduction, which is indicated for immunocompromised patients

5 SUMMARY

	Platelets	FFP	Packed RBC	Whole Blood
Storage	Up to 5 days at 20-24° [warm]	at -18 degree or colder up to 12 months [cold]	4° for up to 42 days [cold]	4° for up to 35 days [cold]
Needs of warming	NO NEED	NEED	NEED	NEED
Needs of filter	NO NEED	NEED	NEED	NEED
Needs of ABO	NO NEED	NO NEED	NEED	NEED
Indication	• Thrombocytopenia & Plt (<15,000) • Bleeding and Plt (<50,000) • Invasive procedure and Plt (<50,000) • 1 unit of platelet	• Coagulation Factor deficiency • fibrinogen replacement • DIC • liver disease • exchange transfusion • massive transfusion	• mild bld loss • severe anemia & hypoxia to Improve O capacity	• Massive Blood Loss • Trauma • Exchange Transfusion (e.g thalacemia)
Notes	transfusion can increase platelet count up to 5000-10000 • Half life of platelets in the body 1-7days	• There is a risk of virus transmitted disease • 1 unit of FFP can increase coagulation factor up to 2-3% • Given within 2 h of order	• Usual dose (10 cc/kg) will increase Hgb by 2.5 gm/dl • Dose administration infusion (slowly) over 2-4 Hrs	• Filter = 170 micro
CROSSMATCH should be done before any transfusion (take 30-45 min) Disadvantages of Blood storage: 1- Precipitation of Ca^{++} (Hypocalcaemia). 2- Affecting on coagulation factors mainly 5 & 8.				

6 MCQS

1. RBCs can be stored at a temperature of:
   a. 1c
   b. 4 c
   c. 10 c
   d. 18 c

2. For a 70 kg patient, 1 unit of platelets transfusion increases platelets count by approximately:
    a. 500-1000
    b. 5000-10000
    c. 15000-20000
    d. 25000-30000

3. Regarding disseminated intravascular coagulation:
    a. Platelet count is normal
    b. Coagulopathy profile is within normal
    c. There is high platelet consumption
    d. Cannot occur secondary to sepsis
    e. Occurs if the patient loses 1.5 liter of blood

4. Standard screening tests on donors' blood include all the following EXEPT:
    a. Hepatitis C
    b. Hepatitis B
    c. Rubella
    d. Syphilis

5. Blood transfusions may cause all of the following except:
    a. Microcirculation thrombosis
    b. Transmission of malaria
    c. Allergic reaction
    d. Bronchospasm
    e. Increase platelets count

🗝 Answers → 1;B , 2;B , 3;C , 4;D , 5;E

NUTRITION

1 INTRODUCTION

- The best interference for a case of malnutrition is either medical or surgical. About 30% of admitted patients, despite treatment, will not get better because of malnutrition itself.

1.1 WHAT IS NUTRITION?

- It's providing your body with the basic nutrition needed in order for it to function properly.
- If you do not take adequate amounts of minerals and macromolecules (Carbohydrates + Proteins + fats), it will cause the body to function poorly, and if the patient is already ill that makes it more specific.
- **The basal metabolic rate [BMR]** increases tremendously during illness [e.g. infections, malignancies, altered hormonal states, etc..]. In this state, more energy is burnt than consumed [imbalance] making the body prone to develop malnutrition. It affects all bodily systems.
- (Imbalance = what's lost is higher than what's gained, same as in starvation)
- In short, **malnutrition** is the condition that occurs when the body is not being given enough nutrients. The most dangerous part of malnutrition is when it involves protein loss. When you have a traumatic accident, for example, a burnt patient, the body will try to consume protein as the main source of calories. Glycogen stores will last for two days [~48hrs] after that; fat starts to be consumed as the main source of energy instead.

2 TYPES OF MALNUTRITION

1. **Kwashiorkor:** adequate calories but not enough protein:
   - Here, you get your calories from macromolecules other than proteins [lipids-fat, and carbohydrates]
2. **Marasmus:** a condition of low calorie, and low [but not all the time] protein state:
   - Here, you have inadequate calories.
   - Protein intake can be normal or low.

2.1 KWASHIORKOR (PROTEIN MALNUTRITION):

- In liver cirrhosis patients, protein is not synthesized as it should be (they have third space ascites). Protein is also lost in kidney disease [nephrotic syndrome] patients and burnt patients.

2.1.1 CLINICAL MANIFESTATIONS OF KWASHIORKOR:

- Hypoalbuminia
- Peripheral edema
- Muscle atrophy
- Altered Immunity

2.2 MARASMUS: NO PROTEIN, NO CARBOHYDRATE

- Usually seen in ICU patients, post major surgeries, cancer, and burn patients.

2.2.1 CLINICAL MANIFESTATIONS OF MARASMUS:

- Very obvious sign in: Weight loss (very low BMI)
- Bradycharia
- Low body Temperature
- Dysphagia
- Anorexia

Mostly: in cancer patients, GIT diseases (Crohn's disease: thickening of the small intestine), major surgery and alcoholism (high calories, zero nutrition).

Mixed	Kwashiorkor	Marasmus
➢ Depleted somatic and visceral proteins. ➢ Patients appear cachectic and severely malnourished. • **e.g.** Chronic hypercatabolic patients and prolonged starvation ○ Trauma	➢ Normal somatic proteins, depleted visceral proteins. ➢ ↓ Serum Albumin and transferrin. ➢ Patients appear normal or overweight. ➢ **Protein malnutrition** • **e.g.** Hypercatabolic critical care patients ○ Chronic diarrhea, chronic kidney disease, trauma, burns, hemorrhage, and liver cirrhosis	➢ Depleted somatic proteins, normal visceral proteins (↓Weight). ➢ Normal Albumin and transferrin. ➢ Patients look thin and malnourished. ➢ **Protein-calorie malnutrition** • **e.g.** Patients with mild to moderate starvation, common severe burns, systemic infections, cancer , conditions in which patients do not eat, like in anorexia nervosa

3 HOW CAN WE EVALUATE MALNOURISHED PATIENTS?

- First, look at the weight, BMI, fat and protein storage. Based on that, calculate the losses.
- Remember, if someone loses more than 10% in the past 6 months, that's a severe sign of malnutrition (3% within a month = severe).
- Vitamin deficiency = severe malnutrition (comes after protein malnutrition)
- **Basic energy expenditure**: when you wake up doing nothing, that amount of calories you require is basic energy (70% of your entire calorie requirement). It depends on the patient's weight and height.
- **Calorie sources**: carbohydrates, fat & protein. The average person will have about 60 % from carbohydrates [glucose], 20-40 % of fat and15% from protein.

4 WHEN TO FEED?

- 24 to 48 hr for **post-admission** patients.
- > 7 days for NPO patients (**TPN**).
- 1-2 weeks for **pre-surgery** patients.
- 3rd day for **post-surgery** patients.

5 WHAT TO FEED?

- Energy 25-30 kcl
- CHO 55% ⓘ
- Protein 1-1.5 g/kg ⓘ
- Lipid 20% 0.5-1.5 g/kg
- Electrolyte
- Antioxidant

Note:
- **Fat:**
1 gram = 9 cal
- **Protein:**
1 gram = 4 cal
- **Carbohydrates:**
1 gram = 4 cal
- **Alcohol:**
1 gram = 7 cal

Suppose you're eating meat all day, or Carbohydrates... If you burn 1 gram of Carbs, it will give you 3-4 cal. If you burn protein, it'll give you the same thing. Which would be better to take? Do you prefer getting calories from carbohydrates or protein? Let us see.

- Protein is only utilized in the body to build cells, enzymes, and muscle. Any extra nutrients [protein in this case] will be converted through **gluconeogenesis,** from amino acids to **glucose**. Glucose will be stored as glycogen. So no matter how much protein you eat; it will be converted to glucose. This way your body (kidney and liver primarily) will be working harder. Therefore, it is not a very good idea to increase your protein intake unless you are a body builder. Otherwise, it would be a waste of time and effort.

6 WHAT ABOUT FLUIDS?

- If you take 45 ml, it will give you adequate fluid for every calorie you consume.
- **A very useful universal formula for patients with healthy kidneys**:
  - For first 10 kg, every kilogram will need 100 ml
  - Second 10 kg, every kilogram will need 500 ml
  - Third 10 kg will be around 900 ml

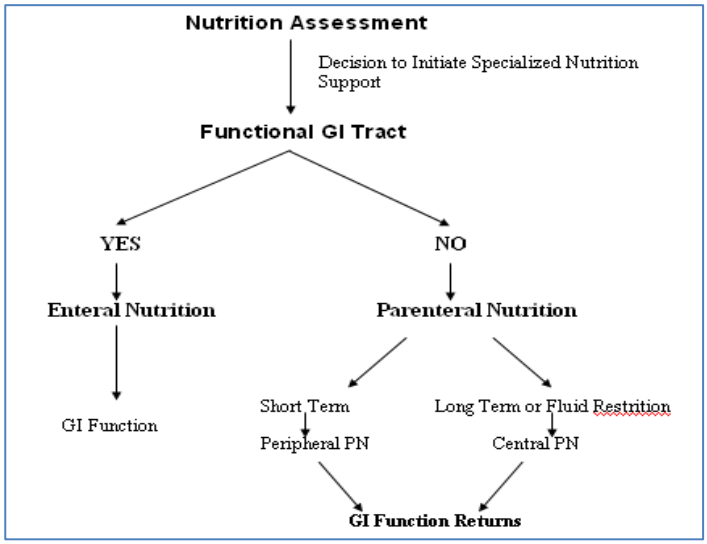
(It is very useful for children)

7 ROUTE OF ADMINISTRATION

- First, if the GI tract is functioning we use **enteral route**. If not (loss of movement happens), you will end up giving them **parenteral (IV).** In IV, there are two sub-routes:
  - Peripheral line
  - Central line (subclavian or jugular vein): the solution given is very high in osmolarity in relation to the plasma (2000) [normally plasma osmolarity is around 300]

- **Enteral route**: only if GIT is working (5 ways)
  - Oral
  - Naso-gastric tube
  - Naso-duodenal
  - Naso-jujenal [from the nose to the jujenum, bypassing the esophagus,

stomach and duodenum]
- Directly into the stomach



7.1 EARLY ENTERAL NUTRITION AFTER BOWEL RESECTION

- **Start oral diet within post-operative week, if hemodynamically & clinically stable**
  - Sips of isotonic oral rehydration solution
  - Small quantities of foods containing complex of CHO & protein (600-1000kcal/d individualized)
  - Limit oral intake if GI losses are high
- **Tube feeding if intolerant to food items**
  - Polymeric enteric formulary → advance slowly
  - Use of nasal or surgical placed gastric or SB feeding tubes

8 WHAT ARE THE COMPLICATIONS OF BOTH ENTERAL AND PARENTERAL FEEDING?

Enteral feeding	Parenteral feeding
• Aspiration • Pneumonia • Diarrhea	• Catheter sepsis • Hyperglycemia ⓘ • Pneumothorax & hemothorax • Electrolyte imbalance • Azotemia • Increase LFT ⓘ

8.1 WHO NEEDS PARENTERAL?

- When a patient is unable to feed himself for more than 5 days
- Intestinal obstruction
- Major surgery
- Short bowel syndrome
- Before that, make sure that all electrolytes are balanced! Why? (Especially K^+ and phosphate)

Refeeding syndrome is a metabolic disturbance that occurs as a result of reinstitution of nutrition to patients who are starved or severely malnourished.

- If he had low K^+ and phosphate, and he's given high GI = major drop in K^+ and Phosphate and he will die (Re-feeding syndrome)

8.2 WHY DON'T WE USE CENTRAL LINES?

- Due to certain complications, which include infection, pneumothorax, and catheter embolism.
- It's avoided in severe necrotizing pancreatitis (only) because of the fistula that will be formed in the intestine. So whatever is ingested is going to be leaked out of the system.
- Also, in patients with nausea and vomiting issues, it will cause aspiration pneumonia.

8.3 CENTRAL AND PARENTERAL NUTRITION

Central Nutrition	Parenteral Nutrition
• Subclavian line	• Peripheral line
• Long period	• Short period < 14days
• Hyperosmolar solution	• Low osmolality < 900 mOsm/L
• Full requirement	• Min. requirement
• Minimum volume	• Large volume
• Expensive	• Thrombophlebitis
• More side effects	

Peripheral Parenteral Nutrition (**PPN**) can be infused through central line

Central total Parenteral Nutrition (**TPN**) CANNOT be infused through the peripheral line.

9 QUESTIONS TO BE ASKED

- **Q: In which cases should we insert NG tube?**
  - Patients with dysphagia and stroke pt. (**long term**)
  - In patients who need nutrition for more than 6 weeks, it will not be a good idea; because it might cause ulceration and irritation.
- **Q: What is the complication of using NG tubes?**
  - Aspiration pneumonia (since the pt. is not moving, the food will move on to the lungs)

10 MCQS

1. All of the following are used to assess the nutritional status of the patient except:
   a. Platelet count
   b. Lymphocyte count
   c. Body weight
   d. Serum albumin
   e. Triceps skin fold
2. Which of the followings is (are) an indication(s) of nutritional support:
   a. Anorexia nervosa
   b. Intestinal fistula
   c. Malignancy
   d. All of the above
3. Metabolic changes after surgery include:
   a. Decreased glycogen breakdown
   b. Decreased lipolysis
   c. Decreased gluconeogenesis
   d. Decreased body weight

🗝 Answers: 1;A , 2;D, 3;D

GENERAL COMPLICATIONS OF SURGERY

1 INTRODUCTION

- All surgeons expect speedy, uneventful recovery
- Always recognized the risk of complications
- Affects result of surgery: poor scar, hernia
- Prolongs hospital stay and cost
- Increased morbidity/ mortality
- Medico-legal issues

1.1 METHODS OF REDUCING POST-OPERATIVE COMPLICATIONS:

- Good pre-operative evaluation
- Optimizing the general condition of patients: **a surgeon should delay his surgery until patients health status reaches optimality or relative optimality, even if that means delaying surgery, especially when is not a emergency surgery. So Medical problems and Nutritional issues must be corrected before surgery.**
- Minimizing preoperative hospital stay: **reduces likelihood of complications like hospital associated infections and DVT.**
- Good surgical technique
- Early mobilization

1.2 PHASES OF POST-OPERATIVE CARE:

- Recovery room
- Surgical ward
- On discharge

1.3 COMPLICATIONS DEVELOPING IN THE RECOVERY ROOM:

- **The complications in this stage are mostly due to cardiopulmonary disease. These happen when patients are recovering from anaesthesia, so Anaesthesiologists are people in charge of these problems.**
- Airway obstruction
- Acute pulmonary complications
- Cardio-vascular complications
- Fluid derangements
- Reactive haemorrhage is the most important post-operative complication in the recovery room either: Slipped ligature or Dislodgement of clot ⓘ

1.4 "GENERAL" COMPLICATIONS:

- Nausea/ vomiting: this maybe due to effects of drugs given to the patients. This usually isn't a significant problem, antiemetics can be given to stop vomiting in sever cases.

- Persistent hiccups: -gastric distension: gastrointestinal peristalsis can be greatly reduced and as a result gas can build up in the stomach causing gastric distension and irritation of the diaphragm (diaphragmatic irritation causes hiccups). This can be corrected by decompressing the stomach with a nasogastric tube. Otherwise, renal failure must be excluded.
- Headache - spinal anaesthesia
- IV site- bruising, haematoma, phlebitis, vein thrombosis, air embolism, infection

2 PULMONARY COMPLICATIONS

- Largest single cause of post-op. morbidity
- 2nd most common cause of death in over 60 age
- Higher risk to patients with chronic pulmonary disease (COPD)

2.1 ATELACTASIS:

- Inability to breath deeply/ cough up secretions: happens in cases when patient breath shallowly and don't caugh. Secretions build up and collapse airways. This is complicated by process like Paralysis of cilia (due to anesthicts), impaired diaphragmatic movement, abdominal distension, pain
- Bronchus/bronchiole obstructed by secretions
- Distal alveolar space close (atelectasis), solidify
- Usually occurs within 24 hours
- Tachypnoea, tachycardia, mild fever **(most common cause of increased temperature after operation),** ↓ breath sound on affected side, ↓PaO2 ⓘ
- Chest X-ray- areas of opacification
- If left untreated: Infection- lobar or bronchopneumonia can develop
- Prophylaxis: stop smoking, physiotherapy for COPD
- Delay surgery if chest infection
- Treatment: encourage deep breathing/cough, mobilization, analgesia, chest physiotherapy ⓘ
- If severe hypoxia develops- intubation, suction, bronchoscopy

2.2 PULMONARY INFECTION:

- Follows atelectasis, gastric aspiration
- Strep. pneumo., H. influenzae or gram negatives are the most common causatives
- Pyrexia, tachypnoea, greenish sputum
- ↓ breath sounds, coarse crepitations, bronchial breath.
- Chest X-ray: patchy fluffy opacities
- Treatment: antibiotics, encourage to cough
- Severe cases: O2, bronchoscopy, ventilation

2.3 RESPIRATORY FAILURE:

- Definition: Inability to maintain normal PaO_2 & $PaCO_2$ levels
- Normal PaO2= 11.6 -13 kPa
- Resp. failure PaO2 < 6.7 kPa
- Central cyanosis
- ABG- key to early recognition

- Treatment: Intubation and ventilation

2.4 ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS):

- Characterized by: Impaired oxygenation, diffuse lung opacification and lung stiffness (↓ compliance)
- Signs: Tachypnoea, ↑ventilatory effort, confusion, hypoxia
- Causes: Systemic/lung sepsis, massive Blood transfusion, aspiration of gastric contents
- Pathophysiology: Endotoxin activated leucocyte→ oxygen-derived free radicals, cytokines & chemical ↑capillary permeability →interstitial & alveolar oedema
- CXR- bilateral diffuse fluffy opacities
- Treatment is ventilation PEEP, treatment of sepsis, hypovolaemia
- Mortality: 50%

2.5 PLEURAL EFFUSION:

- Causes: usually happens after surgery only if the patient has another form of pulmonary pathology like: collapse, consolidation, infarction, tumour deposit.
- Also as a result in abdominal pathology: subphrenic abscess
- Approach: Small effusions left to reabsorb, while large effusions aspirated for culture/ cytology.

2.6 PNEUMOTHORAX:

- Insertion of central venous line is the most common cause of post-operative pneumothorax
- **CXR after insertion central venous line is necessary to exclude this complication.** ⓘ
- Positive pressure ventilation- rupture of pre-existing bullae
- Drained by underwater seal

3 CARDIAC COMPLICATIONS:

- Likelihood of anaesthetic/surgery complications are increased in patients with cardiovascular disease
- Severe aortic/mitral valve dis.- carefully monitor iv fluid administration
- Aortic stenosis impairs heart response to increased post-operative demand
- Whenever possible, treat these before surgery

3.1 MYOCARDIAL INFARCTION:

- Usually history of preceding cardiac disease
- Patients my experience Gripping chest pain.
- Sometimes hypotension is the only sign. *This is greatly due to the anaesthetics/post operative analgesics, where these drugs mask the other symptoms of ischemia/MI.*
- If ischemia is suspected: ECG changes, Cardiac enzymes should be obtained, and Cardiologist should be consulted.
- 1/3rd postoperative MI are fatal

3.2 ARRHYTHMIAS:

- Sinus tachycardia: hypovolaemia, hypotension, pain, fever, restlessness
- Sinus bradycardia: anaesthic agents, pharyngeal suction
- Atrial fibrillation may need medications

3.3 POST-OPERATIVE SHOCK:

- Hypovolaemic: Inadequate fluid replacement, bleeding
- Cardiogenic: acute MI, arrhythmias
- ↑pulse, ↓BP, sweating, pallor, vasoconstriction,↓ urine
- Septic:
  - **Early**: hyperdynamic circulation, bounding pulse, fever, rigor and warm extremity.
  - **Later**: hypotension and peripheral vasoconstriction

3.4 CARDIAC FAILURE:

- Happens in context of Ischaemic or valvular diseases, arrythmia
- Causes: CF is commonly caused by excessive fluid administration in a patient with limited Cardiac reserve.
- Signs: Progressive dyspnoea, hypoxaemia, and – diffuse pulmonary congestion on x-ray
- Treatment:
  - Avoid fluid overload
  - CVP monitoring
  - Diuretics, cardiac inotropes
  - Cardiologist consultation

4 URINARY COMPLICATIONS:

- Associated with: Groin, pelvic, perineal surgery, operations under spinal/epidural anaesthesia
- Causes: Pain, effect of anaesthetic drugs, lying/sitting position, BPH
- Males > females, especially when men have prostatic problems
- Signs: Palpable distended bladder
- Treatment: Catheterization

4.1 URINARY TRACT INFECTIONS:

- Most common nosocomial infection, including in postoperative patients.ⓘ
- Pre-existing UTI, urinary retention, cathterization
- Frequency, dysuria, fever, flank tenederness
- Urine culture
- Treatment: Adequate hydration, urinary drainage, antibiotics

4.2 RENAL FAILURE:

- ARF: protracted inadequate renal perfusion
- Causes: Hypovolaemia (most common cause), sepsis, nephrotoxic drugs like certain antibiotics

- Patients with pre-existing renal disease, jaundice are the most susceptible
- Prevention: adequate IV fluid, urine >0.5ml/kg/hr
- Treatment:
  - replace fluid loss+ 500ml
  - restrict dietary protein to <20Gm/day
  - u/e monitoring, haemodialysis
- Polyuric phase: monitor of fluid intake and u/e
- Recovery 2-4 weeks
- Mortality up to 50%

5 NEUROLOGICAL COMPLICATIONS:

- Cerebrovascular accidents (CVA): sudden ↓ in BP during/ post surgery, hypertensive patients. Carotid endarterectomy, cardiac surgery
- Psychiatric disturbence: elderly, dementia due to cerebral atrophy, use of sedatives/ hypnotics
- Acute toxic confusion: sepsis, hypoxia, uraemia, electrolytes imbalance
- Sleep deprivation particularly in ICU
- Delirium tremens: agitation, tremors, hallucinations

6 DEEP VENOUS THROMBOSIS (DVT):

- Virchow's triad: stasis, ↑coagulability, vessel wall injury
- Risk factors: old age, obesity, prolonged surgery, pelvic/ hip surg, malignancy, past DVT, varicose veins, pregnancy, use of oral contraceptive pills
- Presentation: painful swollen tender calf & fever.
- Diagnosis: Duplex ultrasonography
- Prevention: Compression stockings, mechanical compressions of calf during surgery, subcutaneous heparin
- Treatment: iv bolus/ infusion heparin, LMWH, Warfarin for 3-6 months (INR 2-3 times normal)

7 PULMONARY EMBOLISM:

- Massive PE: severe chest pain, pallor & shock
- CP resuscitation, heparinization, CT angiography, streptokinase/ urokinase (if 6 days post suregry)
- Small PE: chest pain, tachypnoea, haemoptysis
- CXR, ECG , V/Q scan, CT
- Haparinization
- Warfarin for 3-6 months

8 WOUND INFECTION:

- The most common complication
- Incidence 1% (clean) surgeries to 30% (dirty) casses
- Haematoma formation common before infection
- Manifests within 7 days of surgery
- Fever, tachycardia, increased pain at operation site
- Red, tender, swollen, discharging wound
- Remove few sutures to drain the wound

- Antibiotics, if septicaemic

9 WOUND DEHISCENCE:

- Involves abdominal wall. Incidence <1%
- Partial (deep layer), Complete (deep+ skin)
- Serosanguinous discharge, evisceration
- Manifests within 2 weeks
- Risk factors: Obesity, resp. disease, infection, malnourishment, renal failure, malignancy, diabetes, steroid use,& poor surg. Technique
- Re-suture under GA. Develops hernia later

SURGICAL INFECTIONS

1 INTRODUCTION

1.1 DEFINITION

- Infection is invasion of the body by pathogenic microorganisms and reaction of the host to organisms and their toxins.
- **Surgical infections:** Infections that require surgical intervention as a treatment or develop as a result of surgical procedure.
    - A major challenge
    - Accounts for 1/3 of surgical patients
    - Morbidity and mortality *(e.g. septicemia due to post-op infection)*
    - Increased cost to healthcare *(longer hospital stays)*

Exotoxins: toxins secreted by living bacteria that act on distant sites e.g. *Cl. tetani toxin acts on the nervous system*

Endotoxins: structural molecules released when the bacteria is broken down

1.2 PATHOGENESIS

The interaction between microbes and host related factors.

- Microorganism related factors:
    a. Adequate dose
    b. Virulence: pathogenic versus opportunistic organisms
    c. Pathogenicity:
        i. **Exotoxins**: specific, soluble proteins, remote cytotoxic effect e.g. Clostridium tetani, Streptococcus pyogenes
        ii. **Endotoxins**: part of gram-negative bacterial wall, lipopolysaccharides e.g. E. coli
        iii. **Resist phagocytosis** i.e. by protective capsule e.g. Klebsiella and Strep. pneumonia
- Host related factors ⓘ
    a. Suitable environment e.g. hematoma, seroma, foreign body, closed wound, septic technique, long surgery, inadequate drainage
    b. Susceptible host
        i. Skin/mucous membrane breach (surgery/trauma)
        ii. Compromised immunity: cellular (phagocytes) or humoral (antibodies) e.g. elderly, diabetes, drugs (corticosteroids, chemotherapy), radiotherapy

If a patient comes with a wound that has been there >6 hrs, the wound was highly contaminated, or the surgeon was not able to completely remove the necrotic tissue, closing the wound would create a suitable environment for infection.

1.3 CLINICAL FEATURES

1. Local: (apparent in superficial infections)
    - ▶ Signs of inflammation: pain, heat, redness, swelling, loss of function.
    - ▶ Can occur with, or progress to, systemic symptoms
2. Systemic: fever, chills, tachycardia

1.4 PRINCIPLES OF TREATMENT

1. Debridement: remove necrotic, injured tissue
2. Drainage: for abscess, or infected fluid
3. Excision: infection source (e.g. appendectomy)
4. Supportive measures:

a. Antibiotics
b. Immobilization *(if in a limb)*
c. Limb elevation *(to avoid fluid collection)*

1.5 MICROBIOLOGY

Organisms	Properties, Common Infections & Treatment
1. **Staphylococci (Gram +ve)**	• Inhabitants of skin • Sensitive to penicillinase-resistant β-lactam antibiotics • MRSA is resistant to penicillinase-resistant β-lactam antibiotics • Infection characterized by **suppuration** (thick pus formation): o Staph. aureus: **Surgical Site Infection (SSI)**, nosocomial, superficial infections o Staph. epidermidis: opportunistic e.g. wound (SSI), **endocarditis** (especially with prosthetic valves) • Rx: non-MRSA → *cloxacillin, oxacillin* o MRSA = **Vancomycin** ⓘ
2. **Streptococci (Gram +ve)**	• Aerobes/anaerobes • Flora of the mouth pharynx, bowel 1. Streptococcus pyogenes (β hemolytic): **90%** of infections e.g. lymphangitis, **cellulitis**, **rheumatic fever** 2. Strep. viridens: endocarditis, urinary infection 3. Enterococci: urinary infection, intra-abdominal infections
3. Gram –ve Rods	• Most fall into the family Enterobacteriaceae • Most are facultative anaerobic
a. Enterobacteriaceae	**Escherichia, Proteus, and Klebsiella** • Susceptible to a broad variety of antibiotics e.g. 2nd generation **cephalosporins** • Common in **mixed** surgical infections **Enterobacter, Morganella, Providencia, and Serratia** • Greater resistance to antibiotics • Rx: **3rd generation cephalosporins, extended-spectrum penicillins, monobactam, carbapenem, aminoglycosides or quinolone**
b. Pseudomonas, Acinetobacter species	• Obligate aerobic gram –ve • Hospital-acquired ⓘ: pneumonia, peritoneal cavity or severe soft tissue infections • Rx: **ceftazidime** (anti-pseudomonal 3rd gen cephalosporin), cefepime (4th gen cephalosporin), **imipenem**/cilastatin, meropenem, **ciprofloxacin**, acylureidopenicillin, or an aminoglycoside
4. **Anaerobes** (all ⓘ)	• Inhabitants of GIT (**colon**) & the mouth • Most common: **Bacteroides fragilis** • Rx: **metronidazole**, **clindamycin**, imipenem, meropenem, ertapenem, the combinations ticarcillin/clavulanate, ampicillin/sulbactam & piperacillin/tazobactam
5. **Clostridia (Gram +ve)**	• Anaerobe, rod-shaped microorganisms • Live in bowel & **soil** • Produce **exotoxin** for pathogenicity • Most important ⓘ: o Cl. perfringens, Cl. septicum: **gas gangrene** o Cl. tetani: **tetanus** o Cl. difficile: **pseudomembranous colitis**

MRSA: Methicillin-resistant staphylococcus aureus

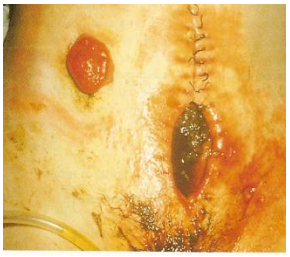
ⓘ A patient 5 days post-appendectomy presents with pain and redness over the site of the wound associated with purulent discharge, fever and chills. The most likely organism: **E. coli**

ⓘ An ICU patient, intubated, and catheterized develops an infection. Most likely organism: **Pseudomonas**

2 SPECIFIC INFECTIONS

2.1 SURGICAL SITE INFECTIONS

- 38% of all surgical infections *(very common ⓘ)*
- **Definition**:
  - Infection within **30 days** of operation
  - Infection within **1 year if prosthetic** device used *(e.g. vascular graft, artificial heart valve, or mesh for hernia repair)*
- **Classification** ⓘ:
  1. Superficial SSI: skin & subcutaneous plane (**50%**)
  2. Deep SSI: subfascial and muscle plane (20%)
  3. Organ space SSI: intra-abdominal, other spaces (30%)
- **Microbiology** ⓘ:
  1. **Staph. aureus** is the most common organism
  2. **E coli**, Enterococcus & other Enterobacteriaceae → deep infections
  3. **B. fragilis** → intra-abdominal abscess
- **Risk factors**:
  - Patient-related: age, malnutrition, obesity, immunocompromised,
  - Surgery-related: poor surgical technique, prolonged surgery, preoperative shaving and type of surgery *(clean/contaminated)*
- **Diagnosis**:
  - Superficial SSI: erythema, edema, discharge and pain *(take a swab of any discharge)*
  - Deep SSI: no local signs, fever, pain, hypotension. Needs investigation *(e.g. CBC, blood & urine cultures, CXR, CT scan)*
- **Treatment**: surgical / radiological intervention
- **Prevention**:
  - Pre-op:
    1. Treat pre-existing infection *(if there is an active infection e.g. UTI, delay surgery until infection resolves, unless surgical emergency)* ⓘ
    2. Improve general nutrition
    3. Shorter hospital stay *(to avoid hospital acquired infections)*
    4. Pre-operative shower *(with anti-septic wash to risk of infection)*
    5. Hair removal (*Surgical site should NOT be shaved the day before the surgery. It's recommended to shave it in the OR, or use other methods of hair removal)*
  - Intra-operative:
    1. Aseptic technique
    2. Good surgical technique *(e.g. do not leave a hematoma or bleeders)*
    3. Normothermia
  - Post-operative:
    1. Wound dressing in 48-72 hours
    2. Early drain removal
    3. Blood sugar control



Pus appears with superficial SSI if skin is opened and with deep SSI if superficial layer of muscle is exposed

ⓘ In abdominal surgeries, the most common organisms causing surgical infections are: **B. fragilis & E. coli** *(can be a mixed infection)*

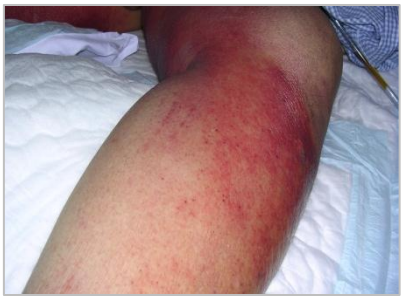
A patient who has undergone an ileostomy presents with signs of infection.

Most likely organism: **E. coli**

Management: Take a swab for culture, AND THEN start empirical antibiotics

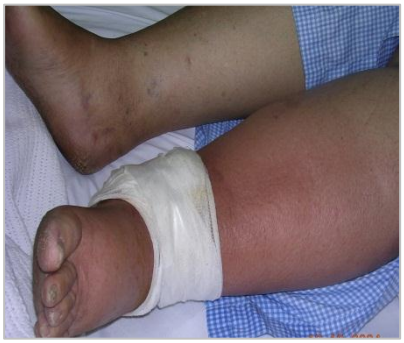
2.2 SOFT TISSUE INFECTIONS

2.2.1 ERYSIPELAS



- **Superficial** spreading cellulitis & **lymphangitis** ⓘ (*limbs to groin = spread toward lymph nodes*)
- Redness, sharply defined irregular border
- Follows minor skin injuries
- Organism: Strep. pyogenes
- Rx: **Penicillin**, Erythromycin

2.2.2 CELLULITIS



- Inflammation of skin & subcutaneous tissue
- Non-suppurative
- Organism: Strep. pyogenes
- Common sites: limbs
- Affected area is red, hot & indurated
- Rx:
  - Rest, limb elevation
  - Penicillin, Erythromycin
  - Fluocloxacillin (if staph. suspected)

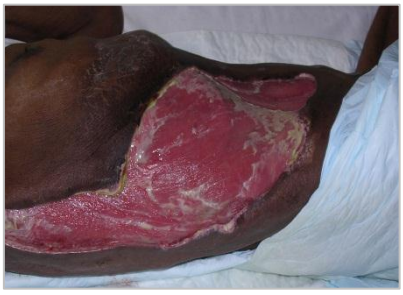
2.2.3 ABSCESS

- Localized collection of pus
- Superficial on the trunk, head and neck → S. aureus, (less commonly streptococci) ⓘ
- In the axillae → gram-negative
- On the perineum → mixed aerobic & anaerobic gram-ve
- Abscess may be mistaken for cellulitis when located deep
- Rx: drainage, antibiotics

2.2.4 FURUNCLE & CARBUNCLE

- Furuncle: infection of hair follicle / sweat glands
- Carbuncle: extension of furuncle into subcutaneous tissue
  - Common in diabetics
  - Sites: back, back of neck
  - Rx:
    - Drainage and antibiotics
    - Control diabetes

2.2.5 NECROTIZING FASCIITIS



- Necrosis of superficial fascia, overlying skin
- Risk factors: elderly, diabetic, immunosuppressed

Erysipelas vs. Cellulitis:

Both are skin infections that present as redness, warmth and edema.

Erysipelas: involves the upper dermis & superficial lymphatics. Therefore, lesions are raised and sharply defined. It is more common in children.

Cellulitis: involves the deeper dermis and subcutaneous fat. It is more common among young children & elderly.

Furuncle and carbuncle are both caused by Staph. aureus

- Organism: "polymicrobial" → **Streptococci**, Staphylococci, Gram-ve rods & anaerobes
- Sites:
  - Limbs
  - Perineum (Fournier's)
  - Abdominal wall (Meleny's)
  - Trunk (in elderly, diabetics, immunosuppressed)
- Starts as cellulitis → edema → systemic toxicity → shock ⓘ
- Appears less extensive than actual necrosis ⓘ
- Investigation: aspiration, Gram's stain, CT, MRI
- Treatment:
  - IV fluids
  - IV antibiotics (broad spectrum: ampicillin, cephalosporins, clindamycin, metronidazole, aminoglycosides)
  - Surgical: **repeated debridement** ⓘ, dressings, skin grafting

> 👁 An immune-compromised patient presents with a small area of cellulitis, **severe pain** and fever post-op. On examination, patient looks very ill and was hypotensive.
>
> Most likely Dx: necrotizing fasciitis
>
> Differentiation from cellulitis: **physical findings do not correspond to severe pain**
>
> Most likely organism: **Streptococci**
>
> Management: immediate surgical debridement

2.2.6 CLOSTRIDIAL MYONECROSIS (GAS GANGRENE)

- Cl. Perfringens, Cl. Septicum, Cl. Novyi (exotoxins)
- Risk factors:
  - *Bone trauma (i.e. open fractures)*
  - Large wounds of muscle (contaminated by soil, foreign body)
  - Drug abusers
- Clinical picture:
  - Pain, crepitus (air felt under skin), swelling, seropurulent discharge, foul smell, myonecrosis
  - Toxemia, tachycardia, ill looking
- Investigations: X-ray = gas in muscle and under skin
- Rx:
  - Antibiotics: Penicillin, clindamycin, metronidazole
  - **Repeated debridement** ⓘ, drainage, amputation, hyperbaric oxygen

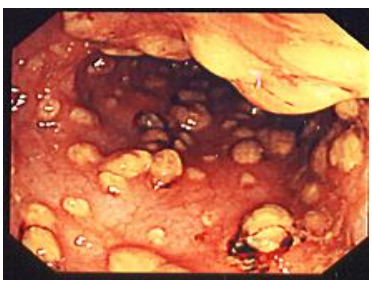
> ⓘ After debridement, the wound should be left open to allow healing from the inside out, and prevent bacteria from growing in their preferred anaerobic (closed) environment.

2.2.7 TETANUS

- Organism: Cl. Tetani (produces neurotoxin)
- Risk factors: penetrating wound (rusty nail, thorn)
- Clinical picture:
  - Trismus: first symptom; stiffness in neck & back
  - Respiration & swallowing become progressively difficult
  - Reflex convulsions along with tonic spasm
  - Death by exhaustion, aspiration or asphyxiation
- Rx is prevention: wound debridement, IV antibiotics (penicillin)
  - **T toxoid:** if previously immunized and booster taken >10 years ago

2.2.8 PSEUDOMEMBRANOUS COLITIS

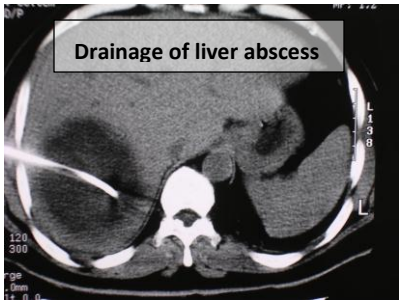
- Organism: Cl. Difficile
- Overtakes normal flora in **patients on antibiotics** ⓘ
- Clinical picture: watery diarrhea, abdominal pain, fever

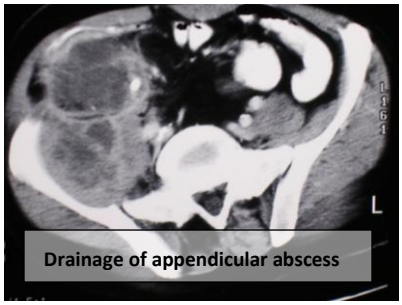


- Investigations:
  - Sigmoidoscopy: membrane of exudates (pseudomembranes)
  - Stool: culture and toxin assay
- Treatment :
  - Stop offending antibiotic *(e.g. clindamycin)*
  - Oral vancomycin/ metronidazole ⓘ
  - Rehydration, isolate patient

2.3 BODY CAVITY INFECTIONS

Primary peritonitis	Secondary peritonitis
Spontaneous	Inflammation/ rupture of viscera
Streptococci, pneumococci 🗎	Polymicrobial
Children **with ascites**	Peptic ulcer perforation, pancreatitis, ruptured spleen, bladder, appendix, diverticulitis
Haematogenous/ lymphatic spread	Direct spread
Rx: antibiotics	Investigations: blood, radiological Treat the original cause





2.4 PROSTHETIC DEVICE-RELATED INFECTIONS

- Examples:
  - Artificial valves and joints
  - Peritoneal and hemodialysis catheters
  - Vascular grafts
- Organism: Staphylococcus aureus
- Rx: Antibiotics, washing of prosthesis or removal

2.5 HOSPITAL-ACQUIRED INFECTIONS

- Definition: occurring **within 48 hours** of hospital admission, **3 days of discharge** or **30 days** following an operation
- 10% of patients admitted to hospitals
- Spent 2.5-times longer in hospital (UK)
- **Highest prevalence in ICU**
- Organism: **Enterococcus, Pseudomonas**, E coli, Staph. aureus
- Sites: **Urinary** ⓘ, surgical wounds, respiratory tract, skin, blood, GIT

3 ANTIBIOTICS

3.1 CLASSES OF ANTIBIOTICS

Class	Examples	*Coverage*
Penicillins	Penicillin G, Piperacillin	*Gram +ve*
Penicillins with β-lactamase inhibitors	Tazocin (piperacillin + tazobactam) Methicillin, cloxacillin	*Anti-pseudomonal* *Anti-staphylococcal*
Cephalosporins	Cephalexin 1st, Cefuroxime 2nd, Ceftriaxone 3rd	*1st & 2nd gen.: Gram +ve cocci 3rd gen.: Gram –ve rods*
Carbapenems	Imipenem, Meropenem	*Gram +ve, gram –ve & anaerobes*
Monobactam	Aztreonam	*Gram –ve, aerobic*
Aminoglycosides	Gentamycin, Amikacin	*Gram –ve rods e.g. E. coli*
Fluoroquinolones	Ciprofloxacin	*Gram +ve, Gram –ve, pseudomonas*
Glycopeptides	Vancomycin	*MRSA*
Macrolides	Erythromycin, Clarithromycin	*Erythro ≈ penicillin, Clarithro = extended*

3.2 ROLE OF ANTIBIOTICS

- Chemotherapeutic agents that act on organisms
  - Therapeutic: To treat existing infection
  - Prophylactic: To reduce the risk of wound infection

3.2.1 THERAPEUTIC USES

Pseudomembranous colitis	Oral vancomycin/ metronidazole
Biliary-tract infection	Cephalosporin or gentamycin
Peritonitis ⓘ	Cephalosporin/ gentamycin + **metronidazole/ clindamycin**
Septicemia	Aminoglycoside + ceftazidime, tazocin or imipenem
Septicemia due to vascular catheter	Flucloxacillin/ vancomycin
Cellulitis	Penicillin, erythromycin

3.2.2 PROPHYLACTIC USES

- Administration of antimicrobial(s) prior to surgical procedures to reduce the number of microbes that enter the tissue or body cavity.
- Antibiotics are selected according to microbes likely to be present at the surgical site.
- Surgical wound classification ⓘ:

Class		Site	Infection	Antibiotics?
I	Clean	Thyroid surgery, breast biopsy	< 5%	No
ID	Graft/mesh used			MUST
II	Clean-contaminated	Minimal contamination e.g. Biliary, urinary or GI tract surgery	11%	Yes
III	Contaminated	Gross contamination e.g. Bowel surgery	17%	Yes
IV	Dirty	Surgery through established infection e.g. Peritonitis	> 27%	MUST

- Prophylaxis in class ID, II, III, IV
- Antibiotic is given **just before** patient sent for surgery
- Duration of antibiotic is controversial (one dose-24 hour regimen)

4 MCQS

1. To prevent infection developing in a lower limb wound sustained 8 hrs ago In a road accident , the single most action is :
    a. Adequate wound debridement
    b. Application of topical antibiotic before Wound closure
    c. Giving broad spectrum antibiotic
    d. Immediate wound closure
2. A 34 y/o man had bacterial infection and treated with antibiotics. During his course, he started to develop fever, which was associated with abdominal pain and watery diarrhea. The most probable diagnosis is:
    a. Food poisoning
    b. Pseudomembranous colitis
    c. Inflammatory bowel disease
    d. None of the above
3. The first thing to do in treating the previous case is:
    a. Start metronidazole
    b. Start vancomycin
    c. Isolation and rehydration
    d. Stop the offending antibiotic

🗝 Answers: 1:a, 2:b, 3:d

STERILIZATION

1 TABLE OF CONTENTS

2 EARLY CONCEPTS OF INFECTION/ASEPSIS AND STERILIZATION

450 BC (Hippocrates)-	Wine & boiled H2O used to irrigate wounds.
1450 BC (Moses)-	Sterilization by fire.
200 AD (Galen)-	Boiled the instruments in the care of wounded gladiators (soldiers).
1545 (Fracastorious)-	Proclaimed that diseases were spread: by direct contact, by handling infected articles that infected people handled previously & by airborne transmission.
1774 (Scheele)-	Discovery of **Chlorine.**
1818 (Thenard)-	Discovery of **Hydrogen peroxide.**
1837 (Schwan)-	Beginning of **sterilization by heat.**
1847 (Semmelweis)-	Used chloro-lime for puerperal sepsis prevention. Introduced washing of hands between patients.
1850-1862 (Louis Pasteur)-	Found out that heat can kill germs (Pasteurization) and theorized that fermentation caused by particles of living matter are so small that they could be carried freely in the air.
1854 (Schroeder & Dusch)-	Introduced the use of filters in sterilization of high temperature pressure.
1859 (Wurtz)-	Discovered **Ethylene oxide**.
1860 (Kuchenmeister)-	Discovered **Phenol** as sterilizing agent.
1860 (Joseph Lister) -	Advocated carbolic soaks, hand sprays, wound dressings, sutures.
1867 (Lister)-	Antiseptic principle in the practice of surgery. Discovery of phenol for infection prevention after operation.

1876 (Koch R.)-	Discovered bacillus anthracis as the cause of disease.
1879 (Chamberland)-	The first autoclave was introduced.
1886 (Ernst Von Bergmann & associates)-	Discovered steam sterilizer under pressure as it is known today to kill heat resistant microorganisms.
1894 (Reinecke)-	Sterilization action of 90% **alcohol.**
1894 (William Stewart Halsted)-	Pioneered the widespread use of **rubber gloves** during surgery.
1900-	All sterilization equipment designed in USA & Europe.
1908 (Grossich)-	Sterilization **by Iodine tincture.**
1927 (Schrader & Bossert)-	Examination of sterilization action by Ethylene Oxide.
1929-	**EO gas** as anti- bacterial agent was introduced.
1933 (Underwood C.)-	Completion of high-pressure steam sterilizer.
1940-	EO gas in industries & hospitals is used for sterilization.
1945-Gamma radiation-	Introduced & used on commercial basis for the sterilization.
1949 (Philips & Kaye)-	Build up theory of E.O. gas sterilization.
1963 (Stone Hill)-	Development of **Glutaraldehyde** (Cidex).
1980 - Antibiotics	Are given before certain types of surgery to prevent infection.
1993-	**Plasma Sterilizer** was introduced.
1999-	**OPA Cidex** was introduced.

3 TERMINOLOGIES

Sterilization ⓘ	The process by which all living microorganisms both pathogenic & non-pathogenic including spores are killed.
Sterile	Absence of all microorganisms including bacteria, mold spores and viruses.
Asepsis	Freedom from infection or the absence of microorganisms that cause diseases.

Sepsis	"Opposite of asepsis" Generalized reaction to pathogenic microorganism, evident clinically by signs of inflammation & systemic manifestation of febrile condition.
Aseptic Techniques	Practices that restrict microorganisms in the environment, on equipment, supplies & prevent the normal body flora from contaminating the surgical wound. Methods by which contamination with microorganisms is prevented.
Bactericidal	**Agents capable of killing or inactivating bacteria.**
Antiseptics	Substances that render microorganisms on living tissue inactive by preventing growth. Combat sepsis. Disinfect body surfaces, on skin & tissue & inhibit the growth of endogenous bacteria.
Disinfection	Any process, which renders inanimate objects free of pathogenic bacteria.
Disinfectants	Agents that kill all growing or vegetative forms of microorganisms, thus completely eliminating inanimate objects.
Contamination	Introduction of microorganisms to a sterile field.

- The prevention of infection in health care areas is largely dependent on the following
- Rigorous adherence to the principles of aseptic techniques by all personnel who perform and assist in any invasive procedures on patients.
- Sterility of all items directly used in such procedures.
- Disinfection of all surfaces and other items in the immediate environment.
- Take note:
- Surgical instruments and heat sensitive items are sterilized by the method recommended by the manufacturer.
- No disposable items designed for sterile single use should be reprocessed.
- Sterilizing agent to be in contact with every part / surface of each item to be sterilized for the specified period of time at the specified temperature.

There is no degree of sterility.

An item is either sterile or non-sterile; it can never be relatively sterile. If you are not sure that this item is not sterile, don't use it.

4 METHODS OF STERILIZATION

- Physical Methods:
- Dry Heat-Hot air ovens, infra-red ovens (Not available in KKUH)
- Moist heat- Steam Autoclave- (Available in KKUH)
- Cool Chemical Methods:
- E.O. Sterilizer- (Available in KKUH)
- Plasma Sterilizer (Sterrad)- (Available in KKUH)
- Liquid Chemicals
- Other Methods

4.1 PHYSICAL METHOD

Moist heat, at a raised atmospheric pressure

- **Steam sterilization** is the most inexpensive and effective method of sterilization. Steam under pressure permits permeation of moist heat to porous substances by condensation and results in destruction of all microbial life. **Ex.** Steam autoclave (steam under pressure)
- Usual method of sterilizing surgical instruments, dressing, drapes, swabs, laps sponges and culture media.

1. Steam autoclave:
    a. An autoclave is a closed chamber in which items or objects are subjected to steam at high pressures and temperatures above 100ºC.
    b. Types of autoclaves:
        i. Downward Displacement Autoclave: Air is removed in two stages and sterilization is effected by an atmosphere of pure steam. Minimum exposure time is required for sterilizing instruments is 50 minutes at 131ºC or 60 minutes at 136ºC. (Not available in KKUH)
        ii. High Vacuum / High Pressure Autoclave: Air is removed by powerful pump. Steam penetrates the load instantaneously and very rapid sterilization of dressings, instruments, raytec swabs, lap sponges, other surgical items & packs is possible in 15 to 40 minutes at 134ºC. (Available in KKUH)
    c. Preparation of items BEFORE sterilization:
        1) Decontamination (wash and decontaminate them)
        2) Disassembly
        3) Washing (wash them again)
        4) Drying
        5) Packing
        6) Loading in sterilizer
2. Ultrasonic Washer: For delicate instruments like in vascular or neurosurgery
3. Automated Washer: Washer & Dryer.

4.1.1 THE STEAM STERILIZATION PROCESS-5 DISTINCT PHASES

1. **PHASE I** -The loading phase - in which the objects or items are packaged and loaded in the sterilizer.
2. **PHASE II** -The heating phase - in which the steam is brought to the proper temperature and allowed to penetrate around and through the objects in the chamber.
3. **PHASE III** -The destroying phase or the time temperature cycle - in which all microbial life is exposed to the killing effect of the steam.
4. **PHASE IV** -The drying and cooling phase - in which the objects are dried and cooled “because if u touch it and it's still moist you’ll contaminate item” , filtered air is introduced into the chamber, the door is opened and the objects are removed and stored.

5. **PHASE V** -The testing phase - in which the efficiency of the sterilization process is checked. All mechanical parts of sterilizers, including gauges, steam lines and drains, should be periodically checked by a competent biomed engineer.

4.1.2 **MAKING** OF STERILE PACKAGES:

- Sterile packages / items should be left untouched and allowed to be cooled before storage to avoid condensation inside the packs.
- Sterile packages must be handled as little as possible to reduce the risk of contamination.
- Event Related Sterility - An item that has been properly cleaned, sterilized, stored & handled will remain sterile unless it is opened or an event happens that compromises sterility.

4.1.3 **STORAGE** OF STERILE PACKAGES: ⓘ

- Sterile packages should be stored on open shelves:
- The lowest shelf should be 8 inches off the floor.
- The highest shelf should be 18 inches from the ceiling.
- All shelves should be at least 2 inches from the walls.
- Sterile packages must be stored and issued in correct order.
- Sterile items are good for either 30 days or 6 months to a 1 year depending solely on how the packages are wrapped and what type of wrappers are used. When it's process in the central supply department it should be with 2 wrappers> standard.
- This is called the shelf life which refers to the length of time a package maybe considered sterile.
- Storage room must be subjected to regular adequate pest control to prevent contamination from rodents, ants and cockroaches.
- Traffic is restricted to CSSD (Central Sterilization Supply Department) personnel and trainees only.

4.1.4 CAUSES OF FAILURE TO DELIVER A STERILE LOAD:

1. Faults in the autoclave:
    a. Poor quality steam
    b. Way it is operated
    c. Failure to remove air and condensate
    d. Faulty gauges and timings
    e. Leaking door seals
2. Errors in loading:
    a. Large packs
    b. Excessive layers of wrapping materials
    c. Over packing
3. Recontamination after sterilization due to:
    a. An inadequate air filter and leakage into the chamber
    b. Wet or torn packs
    c. Incorrect storage

It is necessary to test autoclaves regularly with Geobacillus stearothermophilus, which is one of the most heat tolerant species of bacteria.

If sterilization in an autoclave does not destroy the Geobacilus spores, the autoclave is not working properly.

4.1.5 METHODS OF TESTING EFFECTIVENES OF AUTOCLAVES:

- Bowie Dick test Pack- a pack with a chemical indicator both on the outside and inside to verify that steam has penetrated the pack & to test air leaks.
- Mechanical- chart and gauges usually carried out by Biomed Engineer.
- Chemical- by the use of autoclave tapes, strips and card. A daily test in an empty chamber using a heat sensitive tape. This is for high vacuum/high pressure autoclaves.

Testing the effectiveness of steam autoclaves:

- **First-** They run it empty for one cycle. (Dummy Run) – to warm up the machine. **Second-** They put inside in the middle of the chamber, the Bowie Dick Test Pack and run it again and finish the whole cycle. Oh high pressure- to test leaks and presence of air. (Yellow turns black)
- **Third-** They load it with items and trays for sterilization (little bit lower pressure). It is done once daily.
- **Fourth-** Live Organism- done once in every Saturday morning in CSSD (Central Sterilization Supply Department), KKUH

4.2 COLD (CHEMICAL) METHOD

4.2.1 ETHYLINE OXIDE (EO):

- Well established technique for sterilizing heat labile articles.
- Colorless gas at ordinary temperatures
- Has an odor similar to that of ether
- Has an inhalation toxicity similar to that of ammonia dioxide or fluorinated hydrocarbons (Freon).
- In general, an exposure period of 4 to 7 hours is necessary for complete E.O. sterilization. *Temperature for sterilizing is 21º C to 60º C (70º F to 140º F).
- Used for sterilizing vascular and bone grafts, delicate instruments, plastic articles such as disposable syringes, surgical instruments such as cystoscopes, catheters, bacteriological media and vaccines.

Advantages	Disadvantages
•Used only if materials are heat sensitive and unable to withstand sterilization by saturated steam under pressure. •Easily available and effective against all types of microorganisms. •Penetrates through masses of dry materials; does not require high temperatures, humidity or pressures. •Non- corrosive and non- damaging to items.	•**Lengthy** process with long exposure and aeration periods"7-8 hours" but it's very effective also . •Expensive and more complex process because after sterilization we have to aerate the item to remove residues of Co2. •Produce **serious burns** on exposed skin if not immediately removed. •Insufficiently aerated materials can cause irritation, burns of body tissues, hemolytic of blood and diluents used with EO cause damage to some plastics. •Toxic and may cause **Cancer**. *Precautions should be taken to protect personnel.

4.2.2 PLASMA STERILIZATION:

Plasma Autoclave (Now replaces EO autoclave)

- Low Temperature Hydrogen Gas Sterilizers.
- Employs 1.8 ml. of 58 % hydrogen peroxide vaporized in a sterilization chamber after a vacuum is created, the vapor is converted into plasma by means of radio –frequency energy.
- Spore testing should be performed at the same interval as testing of other sterilizers.
- Advantages of plasma sterilization include speed and safety of use, and the process does not require aeration.
- Used for moisture and heat sensitive devices, such as cameras, scopes and fiber-optic cables, microsurgical instruments , glass, ceramic & some electrical equipment.

4.2.3 LIQUID (CHEMICAL) STERILIZATION:

- When used properly liquid chemo sterilizers can destroy all forms of microbial life including bacterial, fungal spores, tubercle bacilli and viruses.
- Liquid chemicals can be used for sterilization when steam, gas or dry heat is not indicated or available.
- Common liquid agents causing disinfection/sterilization:
    - Aqueous Formaldehyde- Oldest chemo sterilizers known to destroy spores; rarely used due to its pungent odor.
    - 2% Aqueous Glutaraldehyde (Cidex)- Colorless liquid chemical with pungent odor.
    - OPA Cidex-(0.55% ortho-phthalaldehyde)-Clear, pale-blue liquid (pH, 7.5), contains 0.55%the non-glutaraldehyde solution for disinfection of flexible endoscopes and other medical devices.
    - Alcohol- 70% Isopropyl Alcohol- Effective & rapidly acting disinfectants. *Alcohol gel preparations today have been introduced & long standing effect, fast in action & more users friendly.
    - Chlorexidine- Useful skin antiseptic & highly active against vegetative bacteria. Used in hand scrubbing .
    - Hypochlorite- Broad spectrum chlorine disinfectant effective against viruses, fungi, bacteria & spores. *Disinfectant of choice against hepatitis B virus.

4.3 OTHER METHODS OF STERILIZAION

1. Gamma radiation:
    a. Radioactive material, such as a Cobalt-60 source, emits radiation (gamma rays).* Pure energy that is generally characterized by its deep penetration & low dose rates.
    b. Gamma Radiation effectively kills microorganisms throughout the product and its packaging with very little temperature effect.
    c. Used on commercial basis for the sterilization of a wide variety of pre-packaged hospital items and devices.

d. Total sterilizing time is measured in days.

2. Flash sterilization:
   a. Should be used in selected clinical situations & in a controlled manner. *Use of flash sterilizer should be kept to a minimum & only for emergent use.
   b. Flash sterilization should be used only when there is insufficient time to process by the preferred wrapped or container method, and should not be used as a substitute for insufficient instrument inventory.
   c. Flash sterilization should not be used for implantable devices.

5 PRINCIPLES OF ASEPTIC TECHNIQUES

5.1.1 DEFINITION:

Aseptic techniques are sets of practices / procedures performed under careful, controlled conditions in order to minimize contaminations of pathogens.

- **Most strictly applied in the O.R.** because of direct & extensive disruption of skin & underlying tissues.
- These practices ensure safe & effective ways in establishing & maintaining sterile field in which surgery can be performed safely.
- Aseptic techniques help to **prevent or minimize surgical site infection.**
- Tears in barriers & expired sterilization dates are considered breaks in sterility.

All items used within the sterile field must be sterile.

Scrubbed personnel should function within a sterile field.

5.1.2 THE SURGICAL TEAM CONSISTS OF:

- Members are either:
  - Sterile members or scrubbed personnel (work directly in the surgical field.) e.g. Surgeons, Scrub nurse, O.R. Technician
  - Non-sterile members or unscrubbed personnel, e.g. Anesthetists, Circulating nurses, Anesthesia Technicians, X-Ray Technician and students
- Surgical team members must wear the scrub suit attire with the surgical cap, surgical face mask before performing surgical hand scrub.
- Surgical hand scrubbing should be performed prior to the donning of sterile gown & sterile gloves.

5.1.3 SURGICAL HAND SCRUBBING:

- Surgical Hand Scrub is performed before come in contact with sterile field.
- The first surgical hand scrub should be at least 5 minutes and the subsequent hand scrub should be at least 2 to 3 minutes.
- Keep nails shorts. No rings & other jewelry, no artificial nails.
- Principle to be applied: "Fluid flows in the direction of gravity." Hands are held higher than elbows.

The sterility is limited to the portions of the gowns directly viewed by the scrubbed person.

5.1.4 DONNING OF GOWNS/STERILE GLOVES:

a. Gown should not touch any unsterile parts.
b. Gloves outer side is not touched by bare hands.

Cuffs should be considered unsterile due to its tendency to collect moisture & it is not an effective barrier. Therefore, cuff should always be covered by sterile gloves.

- Scrub nurse may assist other personnel in donning sterile gown & sterile gloves.
- Gowns are considered sterile only on the: ⓘ
  1) Front of gown from chest to the level of the sterile field.
  2) Sleeves of gown from 2 inches above the elbow to the cuff.

c. Areas of the gown considered non-sterile: ⓘ
   1) Gown's neckline
   2) Under the arms
   3) Shoulders
   4) Back

5.1.5 SKIN PREPPING:

d. Surgical site is cleaned with appropriate antiseptics containing Povidone-Iodine 70%, Alcohol 70 % & Chlorexidine 0.5%.
e. Apply antiseptic at the line of proposed incision site in concentric circles moving towards the periphery.
f. Cotton tipped applicators with antiseptics are needed to clean the umbilicus thoroughly.
g. Antimicrobial tincture or paint may be applied according to surgeon's preference.

It's better to use both Iodine & Alcohol, starting first with Iodine then using Alcohol.

5.1.6 SURGICAL DRAPES:

h. Surgical Drapes are sterile materials used to maintain the sterility of the operation field, as they create a sterile field within them.
i. Surgical Drapes establish an aseptic barrier minimizing the passage of microorganisms from non-sterile to sterile areas.
j. Sterile surgical drapes should be placed on the patient, parts of O.R. table & equipment included in the sterile field, leaving only the incision site exposed.

5.1.7 DRAPING PROCESS:

k. Only the scrubbed personnel should handle sterile drapes by cuffing the draping material over the gloved hand.
l. When draping, the surgical drapes should be compact, held higher than the O.R. table & draped from the prepped incision site to the periphery.
m. Tables are only sterile at the table level.
n. Once the drape is placed, it should not be moved or re-arranged & only the top surface of the draped area is considered sterile.

An inch safety margin is usually considered standard on package wrappers, whereas the sterile boundary on a wrapper used to drape is at the table edge.

5.1.8 STERILE WOUND DRESSING:

- Dressing material should only be opened during wound dressing time.
- Wound or surgical site should be cleaned & dried before application of the dressing material.
- Dressing material should be applied before surgical drapes are removed to avoid contamination of the incision.

5.1.9 GENERAL POINTS OF EMPHASIS:

- When opening the sterile items, unscrubbed personnel should open the wrapper flap farthest away from them first and the nearest wrapper last to prevent contamination by passing an unsterile arm over a sterile item.
- After a sterile package or container is opened, the edges are considered unsterile.
- Either the entire bottle contents should be poured into the receptacle (Jug) or the remainder should be discarded.
- Pouring should be done at the edge of the table & not at the middle.
- Contaminated items must be removed immediately from the sterile field.
- Surgical patient's operative site is the center of the sterile field & all the scrubbed personnel should remain close to this area without wandering around the room. Movements can cause contamination of the sterile field.
- Surgical team should move only from sterile areas to sterile areas. If they change positions, they should turn back to back or face to face and maintain a safe distance close to the sterile field.
- When delivering sterile supplies onto the sterile field, never contact or reach over any portion of the sterile area. Non-sterile items should not cross above a sterile field.
- Non-sterile personnel should always face the sterile field on approach and should never walk between 2 sterile fields.
- O.R. personnel with colds & URTIs should avoid working inside the theater or else wear double masks.

Edge of the bottle cap is considered contaminated once the cap has been removed from the bottle.

Fluid/solution should be poured slowly to avoid splashing. Splashing can cause strike through and contamination of the sterile field.

The general margin of safety is considered to be a **minimum of 12 inches.**

6 MCQS

1. **Which of the followings is correct regarding sterilization?**
    a. Chemical sterilization is the commonest type to be used
    b. The scrub up team must keep hands above waist
    c. Disinfection is considered enough for surgical instruments
    d. Suspicion of organisms spread during an operation is not harmful
2. **Which one of the following is the definition of sterilization:**
    a. The documentation of instrument after using then packing and loading them.
    b. The elimination of violable microorganisms including viruses , bacteria ,fungi except spores.
    c. The process by which all living microorganisms both pathogenic & non-pathogenic including spores are killed.
    d. The universal precautions that is applied to all patients when there is exposure of blood product
3. **To avoid contamination of the surgical wound. The sterile wound dressing pad should be applied:**
    a. After cleaning and drying the wound before removing the drape
    b. After surgical drapes are removed
    c. Before leaving the operating theater
    d. Without cleaning and drying the surgical wound

🗝 **Answer Key:** 1;B , 2;C , 3;A

PRINCIPLES OF SURGICAL ONCOLOGY

1 INTRODUCTION

1.1 TYPES OF TUMORS

1.1.1 BENIGN

- Encapsulated
- No invasion (controlled growth)
- No metastasis
- Can be removed locally
  - In lipoma, we only have to resect it by simple excision, whereas in liposarcoma, we have to remove the skin and adjacent tissue as well.

1.1.2 MALIGNANT

- Non-encapsulated
  - Sometimes, there is a capsule but it's a "false capsule", meaning it's a fibrous capsule from the same tissue.
- Usually does invade (uncontrolled growth)
- Metastasis
- Loss of contact inhibition ⓘ
  - Normally, cells stop growing and reproducing once their plasma membrane comes into contact with another. Cancer cells lack this contact phenomenon. They continue to grow into other cells taking over and often destroying the other cells, creating a tumor.
- Has two main types:
  - Carcinoma
    - Arises from epithelial tissue
    - Ex: Adenocarcinoma of the stomach, transitional cell carcinoma of the bladder, squamous cell carcinoma of the skin, follicular carcinoma of the thyroid.
  - Sarcoma
    - Arises from connective tissue
    - Ex: lipoma(Benign), liposarcoma (Malignant), Fibroma(Benign) Fibrosarcoma (Malignant), Myoma (Benign),myosarcoma (Malignant), *Rhabdomyosarcomas* are tumors of the skeletal muscles, *Leiomyosarcomas* are smooth muscle sarcomas.

1.1.3 TERATOMA

- Arises from the embryonic "totipotential cells", which are capable of developing into any variety of cells.
- Commonly found in germ cell areas (testes and ovaries)
- Could be benign or malignant
- Has the potential to produce new tissues in the organ affected

ⓘ **The difference between metastasis and direct invasion:**

- Direct invasion: tumor enlarges to invade the adjacent organ with continuity of primary tumor. (e.g. bladder cancer goes to colon or uterus).

- Metastasis: tumor invades other organs with discontinuity of primary tumors.

ⓘ Benign growth is controlled whereas malignant growth is not. That's why it:

- can invade the same organ(non-encapsulated), go to adjacent organs, or go to lymph or blood
- can metastasize e.g. cancer in lung goes to brain, cancer of colon goes to lung, cancer of prostate goes to vertebral column.

- Ex: Dermoid ovarian cyst, a cystic teratoma that contains developmentally mature skin, complete with hair follicles, sweat glands, bone and cartilage, which are not normally found in the ovary.

1.1.4 HAMARTOMA

- Abnormal arrangement of normal tissue, “haphazardly arranged tissue” that resembles a neoplasm.
- Benign
- Capable of producing complications
- Ex: Angiomyolipoma of the kidney, composed of blood vessels, smooth muscle cells and fat.

2 GRADING AND STAGING

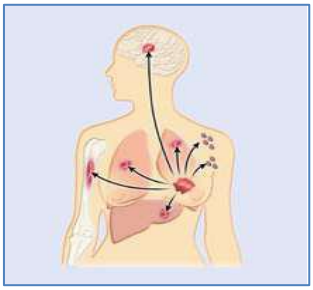
2.1.1 GRADING

- It describes the histological characteristics of cancer cells, mainly the cell layers (e.g. grade I, II, III…) ⓘ
- Grade of Differentiation describes the characteristics of cancer cells in reference to their resemblance to the cell of origin ⓘ
  - Well differentiated
  - Moderately differentiated
  - Poorly differentiated
    - Indicates that the cancer is rapidly growing with no time for the cells to be differentiated.
    - Most of them are more susceptible to chemotherapy agents b\c they’re weak due to the rapid development and growth.
  - Anaplastic
    - E.g.: if we found an enlarged lymph node but we did not know the origin, we send it to the lab. If it it’s a well-differentiated tumor, the pathologist will be able to identify the cell of origin.
    - However, in poorly differentiated or anaplastic tumors, the pathologist will not be able to identify the cell of origin, he will only be able to confirm the malignancy.

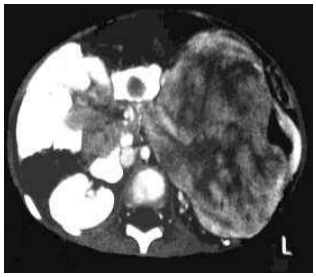
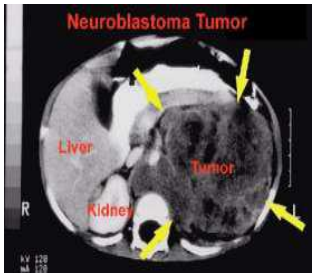
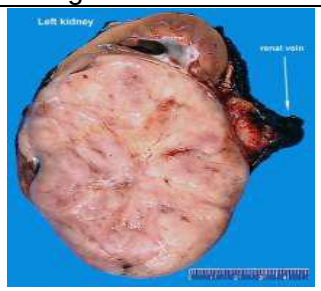
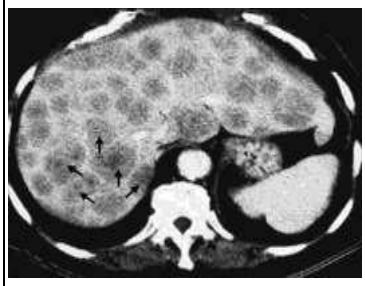
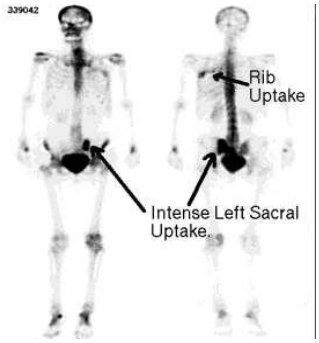
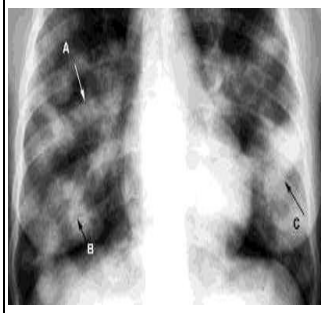
📄 The cell usually differentiates from being a "blast" in the beginning to it becoming a "cyte". The blast stage means it is still growing, and if we see a "cyte", it’s closer in morphology to the mother cell.

2.1.2 STAGING

- Describes the primary tumor, the relation of the primary tumor with:
  - The organ of origin
    - Bladder cancer can go to the uterus, small intestines, peritoneum, and rectum (local). Can also go to the liver, chest, brain
    - E.g.: tumor in the stomach can go to the duodenum, tumor in kidney can go to posterior abdominal wall, bladder cancer goes to colon, uterus, lateral pelvic wall .
  - The adjacent organs
  - The distant organs and lymph nodes:
    - In general, the organs that get metastasized the most are the liver, lung and bone,
    - But the brain is rarely metastasized due to presence of BBB and can be metastasized from bronchogenic carcinoma.
    - Types of Spread:



1. Lymphatic
   - Regional & distant lymph nodes
   - E.g. Colon cancer manifesting as a lump in the neck
   - Lump in the neck >> 1st sign of metastasis of cancer in the colon, stomach and testes.
2. Hematogenous
   - Common areas of metastasis: Liver, lung, bones
   - Brain isn't a common target of metastasis because of the BBB that can block out the cancer cells. However, small-cell lung cancer CAN metastasize to the brain. It spreads very quickly and also produces hormones like ACTH from the lung.
3. Transcoelomic
   - Dissemination of malignant tumors throughout the peritoneal (abdominal & pelvic) cavity
   - E.g." Krukenberg tumor", stomach cancer metastasis to the ovary, despite the lack of any anatomical relations between both (lymph nodes nor blood vessels nor direct).
4. Intraperitoneal seeding during surgical manipulation
   - Implantation e.g. needle tracks, wounds…
   - Very rare
   - Needle biopsy should be obtained for diagnosis

		
Local Invasion	**Black areas: Hot spots in bone**	**False capsule**
		
Cannonball Metastases		

- Types of Staging
  - Classical staging:
    - Stage I and II confined to the organ
    - III =direct invasion
    - IV= metastasis
  - TNM Classification is more specific: e.g. T1, No, Mo
    - T – Tumor : T1,2,3…., Tis, Ta, Tb…
    - N – Node : N0, 1, 2, 3 ….
    - M – Metastasis : M0,1,2,3…
- Why do we stage malignant tumors?
  - To decide the treatment
    - Treatment for primary tumors is different from secondary ones and localized is different from metastasis.
    - E.g. you can't tell the patient he has cancer in the kidney when you don't know if there's metastasis to the liver. This way you have exposed the patient to unnecessary treatment when operated on (because there is no benefit of the operation, since you didn't check for metastasis)
  - To plan the treatment
    - Multimodality treatment
    - Sometime they're referred to the tumor board to plan the treatment (surgery, radiotherapy, chemotherapy)
    - Duration of treatment depends on the case
  - To assess the prognosis
    - E.g. if we have a patient with a localized kidney tumor and another with a metastasized kidney tumor, the second patient has poorer prognosis in comparison
    - "Our expectations, according to Statistics but not necessarily applied to the patient himself ". So when we talk about certain tumors and its high percentage for bad prognosis, this is a statistical study for a population. But when we talk for a person, s/he has 50% of having bad or good prognosis.
- Remember that every organ has its own different staging (e.g. Duke classification for colon cancer only)

There's no mention of lymph nodes or distant metastasis in the classical staging. That's why the TNM classification has been added.

3 PRESENTATION OF MALIGNANT TUMORS

- Asymptomatic (incidental finding)
- Symptoms related to the primary tumor
  - E.g. Bleeding per rectum or intestinal obstruction for colon cancer
  - Dysphagia for esophageal cancer
  - Hematuria for bladder tumor
  - Hemoptysis for lung cancer
- Symptoms related to the secondary tumors
  - E.g. 60 y/o female had sudden low back pain, after investigations, she was discovered to have breast cancer
  - Hemoptysis- patient might have cancer in the kidney and the patient doesn't have any problem in urination (secondaries)
  - Minimal fall > pathological fracture – discovered to have bone metastasis

ⓘ In the GIT, weight loss & cachexia depends on the level of tumor, at which the food is blocked. So it's more evident in the esophagus (highest level), than in the stomach and the colon (lowest level).

- Weight loss & Cachexia ⓘ
  - Late manifestations of most malignant tumors (advanced stage) except in GI and lung cancers (bronchogenic carcinoma)
- 1st presentation comes from the secondary and not from the primary
Presentation of malignant tumors:
  - Seizure > metastasis to brain
  - Colon cancer > can present as bleeding per rectum – abdominal distension – intestinal obstruction
  - PE= enlarged liver: nodular, rough, smooth, hard.. (to know character of pain)

4 INVESTIGATIONS

- Investigate for the primary tumor
  - For primary we have to define histological features
  - In 99% of the cases, we have to know the tissue diagnosis in order to determine the tumor type
    - Define the histology
    - Define the local extension
    - Depends on the site
- Investigate for the secondaries:
  - Look for metastasis
  - Usually liver, lung and bones
- Both will define the diagnosis & stage
  - Accordingly, the treatment plan will be determined
  - Treating Malignant tumors exposes the patient to major surgeries, dangerous chemotherapy or troublesome radiotherapy. So make sure that it is malignant then define the type of this tumor (each malignancy has a specific way of treatment)

4.1.1 CYTOLOGY:

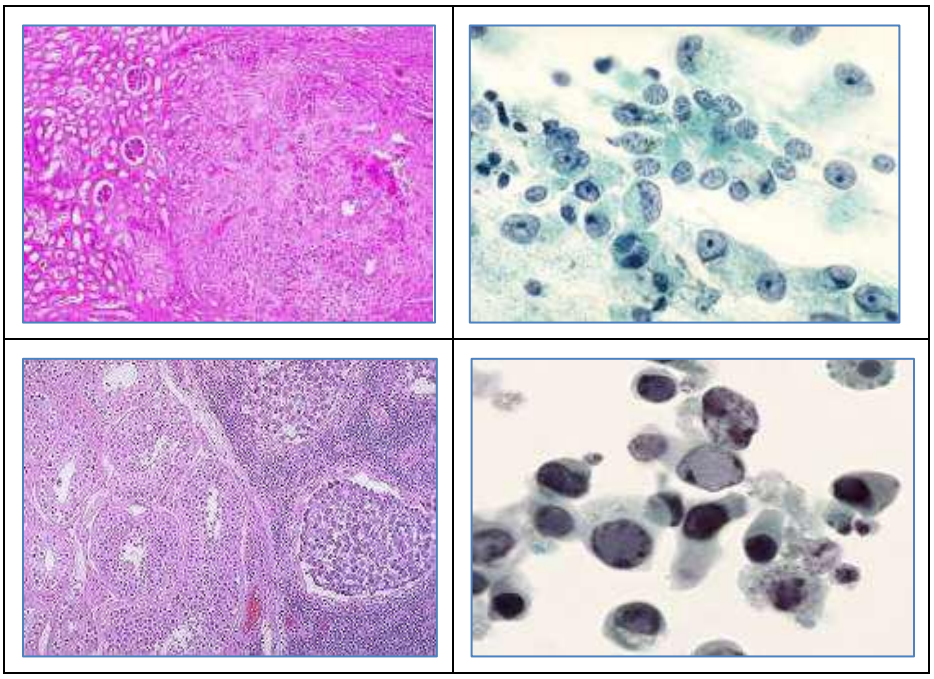
- Morphology of individual cells
- Many ways of obtaining it
  - Exfoliative (fluid): urine – sputum the epithelial layer Multiplies and the superficial cells fall down so try to collect & get benefit from it “without any effort from doctor “as in sputum or urine sample”.
  - Fluid aspiration: ascites, pleural fluid, cyst acidic fluid or plural effusion draw out and send to cytology
  - FNA: taking cells from solid tumors, Fine needle aspiration (FNA), very common nowadays: in solid tissue and draw out cells, then stain the cells on the slide and look under the microscope for any malignant cells.

4.1.2 BIOPSY

- Examination of the tissue
  - Fine-needle aspiration
  - Core biopsy
    - E.g. Tru-cut: core of tissue removed for histological examination
    - Usually done if the lump is apparent and distinct and localized
    - Commonly done through endoscope
  - Incisional

- Removes a small accessible piece of the lesion for histological examination (forceps, needle...)
- Many ways of obtaining it
  - Like in ulcer, you take a small sample by a knife then send it to histology
  - Needle
    - ✓ E.g. if having breast cancer for example under x-ray, US or CT control
  - Gastroscope
    - ✓ If we suspect a gastric ulcer to be malignant OR colonoscope

- Excisional:
  - Complete removal of a discrete lesion without a wide margin and without it being considered curative of the malignancy
  - E.g. Remove breast lump for histology
  - Sometimes, this cannot be done because the tumor is disseminated or cannot be removed alone



- The difference between benign and malignant cells:
  - Malignant cells are characterized by deeply stained nuclei (darker), divided nuclei that are larger in size in comparison to the cytoplasm, and the shape of the cells is not identical (polymorphism, the cells in different stages of growth).

5 TUMOR MARKERS

- Substances present in the blood or tissue fluid in a concentration related to the presence of a tumor
- Most markers are cells from normal cells or malignant cells (primitive)
  - Most are non-specific

- Important in diagnosis (general findings + tumor markers>> Dx)
  - E.g. patient with testicular tumor "clinically" and was found to have a high level of the tumor marker>> the patient has teratoma not seminoma
- Important in follow up
  - E.g. patient has testicular tumor and high α –fetoprotein,, after removing the tumor, α –fetoprotein is decreased. If after 6 months, the α –fetoprotein goes back up, that indicates recurrence of the tumor.
- Important for screening
  - The early detection, incidence of disease
    - Males over 40 years old do PSA
    - Mammography for carcinoma of the breast
    - PAP smears for cervical carcinoma
    - Others: CEA, α-fetoprotein, HCG

- Sometimes pathologists use histochemical stains for specific tumor markers in tissue, and by this we can determine the type of tumor.
- Patients with high PSA, biopsy showed no indication of malignanacy > false +ve
- Patient has malignancy but PSA level was normal > false –ve
- To detect relapses

6 HORMONES AND CANCER

- **Hormones related to tumor growth:**
  - Usually sex hormones (testosterone, estrogen)
  - They may have a relation to tumor growth
  - Hormone receptors are involved
  - The concept can be used in treatment
    - E.g. In breast cancer, ask the histologist to find any estrogen receptors. That will affect the treatment plan and prognosis. Also the prostate needs testosterone to live, so if we block the testosterone secretion by drugs, the tumor will stop growing
    - Growth of the prostate and the malignant cells are dependent on the testosterone. So we control the malignancy by either removing the primary producing organ of the tumor, which is the testes, or blocking one of these pathways.
  - When tissue is taken from a cancerous breast and gets sent in to the lab, we may find estrogen receptors which could be treated with antiestrogen (Tamoxifen), thus decreasing the effect of estrogen on the breast. This way we're minimizing growth of the malignant cells.
- **Hormones may be produced by tumors:**
  - Originally hormone producing organ e.g. adrenals (Cushing's…)
  - Originally non hormone producing organ e.g. lung (bronchogenic carcinoma)

7 MCQS

1. **A patient comes with an enlarged cervical lymph node, which of the following is unlikely to be the primary site?**
   a. Bronchus
   b. Stomach
   c. Colon
   d. Mouth
   e. Laryngopharynx

2. **To detect hematogenous spread of a tumor, all the followings should be done EXCEPT:**
   a. Chest radiograph
   b. Cystoscopy
   c. Abdominal CT
   d. Bone scan

3. **Which of the following tumors has the least potential of malignant transformation?**
   a. Renal angiomyolipoma
   b. Ovarian embryonic carcinoma
   c. Osteosarcoma
   d. Mesothelioma

🗝 **Answers: 1:c, 2:b, 3:a**

PRESENTATION AND MANAGEMENT OF CARDIAC SURGICAL DISEASES

1 OBJECTIVES OF THE LECTURE

- Overview of diseases of the heart, where surgery can play a role.
- Understanding of the Basic Principles of Cardiac Surgery.
- Information regarding pre-operative, operative and post-operative care in cardiac surgery.

2 INTODUCTION

2.1 CARDIAC DISEASES

- Coronary Artery Disease
- Valvular Heart Diseases
- Congenital Heart Diseases
- Miscellaneous :
  - Aortic Diseases
  - Pericardial Disease
  - Cardiac Tumors
  - Trauma

2.2 MODES OF PRESENTATION OF CARDIAC DISEASES

- Chest pain
  - IHD (Ischemic Heart Disease)
  - Aortic disease:
    - Dissection
    - Aneurism
- Shortness of Breath
- Palpitations
- Peripheral Edema
- Congestive Cardiac Failure
- Cyanosis and Clubbing in Congenital Defects
- Uncommon presentations
  - Pleural Effusion
  - Hemoptysis

Chest pain and Shortness of breath are the most common presentations of cardiac patients.

Life threatening causes of chest pain: IHD, Pulmonary Embolism, aortic dissection, tension pneumothorax

2.3 COMMON CARDIAC OPERATIONS

- Coronary Artery Bypass Grafting (CABG) (most common)
- Valve Replacement / Repair
- Repair of congenital defects like ventricular septal defect (VSD) or atrial septal defect (ASD)
- Heart Transplantation

3 ISCHEMIC HEART DISEASE

3.1 CLINICAL MANIFESTATIONS:

- Asymptomatic
- Symptomatic:
  - Angina pectoris: stable- unstable
  - Myocardial infarction
  - V.S.D., Ischemic mitral regurgitation, Ventricular aneurysm, Heart failure, Conduction defects.

3.2 Risk factors:

1. Smoking
2. Diabetes mellitus
3. Hypertension
4. Hyperlipidemia
5. Hereditary factors.

3.3 LABORATORY INVESTIGATIONS:

- Routine investigations
- Cardiac enzymes
- E.C.G.
- Echocardiography
- Coronary angiography ⓘ

3.4 INDICATIONS OF SURGERY: ⓘ

1. Failure of medical therapy or percutaneous intervention.
2. Left main coronary artery disease.
3. 3-vessel disease with left ventricular dysfunction; three coronary arteries are affected.
4. Mechanical complications of myocardial infarction; include: 1. Tamponade .2. Wall rupture .3. Chordae tendinae rupture .4. Valve weakening
5. Associated valve disease; patient with IHD + valve problems = refer to surgery.

3.5 CORONARY CONDUITS: ⓘ

1. Arterial: Internal thoracic (mammary) artery
2. Venous: Long saphenous vein.

3.6 TYPES OF SURGERY:

1. Conventional: the heart is stopped using the heart lung machine, and cardioplegic arrest. The machine is used to maintain blood and oxygen supply. Used in valvular and congenital cardiac surgeries (because we have to open the heart)

Complications of IHD

- Angiography is used to decide the type of Rx:

1- Medical

2- Angioplasty 80%

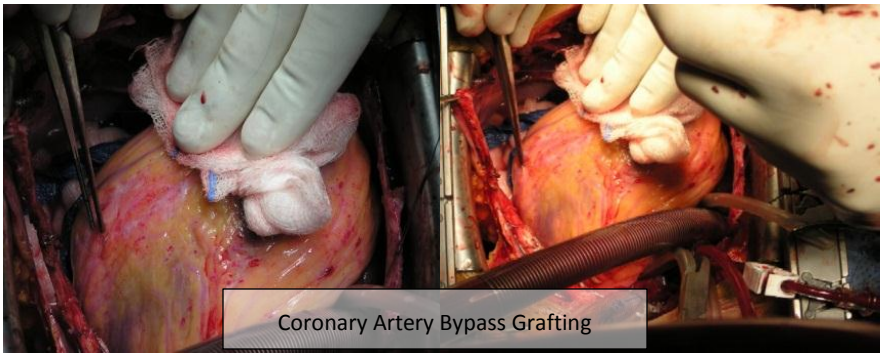
3- Surgery 20%

(2+3 Revascularization therapy)

Percutaneous intervention: angioplasty, balloon dilatation and stenting.

- Left main coronary artery: this is the main stem of the left coronary circulation. If the blockage is before it branches to left anterior descending and circumferential artery then its indicated for surgery.

2. Off-pump (beating heart surgery); When working on the coronaries, we don't need to stop the heart because they're external features. But we must stabilize the area.



Arterial grafts are better than venous; they have longer patency (In 10 years, 95% arterial grafts are patent, but only 50% of veins remain patent.

-Veins are normally under low pressure, so if they are used as coronary grafts, they are prone to high pressure from the aorta and atherosclerosis.

The internal mammary artery is preferred (it is a smooth muscle artery, as opposed to the radial artery which is a muscular artery and may undergo spasm).

Venous graft's patency may be improved by using antiplatelet medication and statins. But they are still not as patent as the internal mammary artery.

4 VALVULAR HEART DISEASES

4.1 MITRAL STENOSIS:

- Etiology: Rheumatic, Congenital
- Investigations: E.C.G., X-ray chest, Echocardiography
- **Indications for surgery**:ⓘ
  - Symptoms: exertional dyspnea, pulmonary hypertension, hemoptysis
  - Severe mitral stenosis: area less than 1 cm.
  - Left atrial thrombus.
- **Treatment**:ⓘ
  - Medical
  - Balloon valvuloplasty (dilatation in stenosis w/o regurgitation)
  - Closed mitral commissurotomy (doesn't need heart-lung machine)
  - Open mitral commissurotomy (needs heart-lung machine)

4.2 MITRAL REGURGITATION:

Mitral and Aortic are the most common diseased valves, sometimes the tricuspid as well.

- Etiology: Rheumatic, Degenerative, Endocarditis, Ischemic, Traumatic
- **Indications for surgery**:ⓘ
  - Symptomatic
  - Dilated left ventricle
  - Diminished ejection fraction
- **Treatment**:ⓘ 1.Medical 2.Mitral valve repair 3.Mitral valve replacement.

4.3 AORTIC STENOSIS:

Mitral valve replacement.Open mitral commissurotomy and mitral valve replacement are the only surgical procedures in the treatment list

Closed mitral commissurotomy is a surgical procedure but it is not preformed anymore.

- Etiology: Rheumatic, Congenital, Degenerative.
- **Indications for surgery**:ⓘ
  - Symptoms (angina, shortness of breath, syncopal attacks)
  - Severe aortic stenosis
- **Treatment**:ⓘ 1-Medical 2-Aortic valve replacement

4.4 AORTIC REGURGITATION:

- Etiology: Rheumatic, Endocarditis, Connective tissue disorders, Aortic dissection
- **Indications for surgery**:ⓘ
  - Symptomatic patients.
  - Progressive left ventricular dilatation.

4.5 PROSTHETIC VALVES: TYPES, MERITS AND DEMERITS

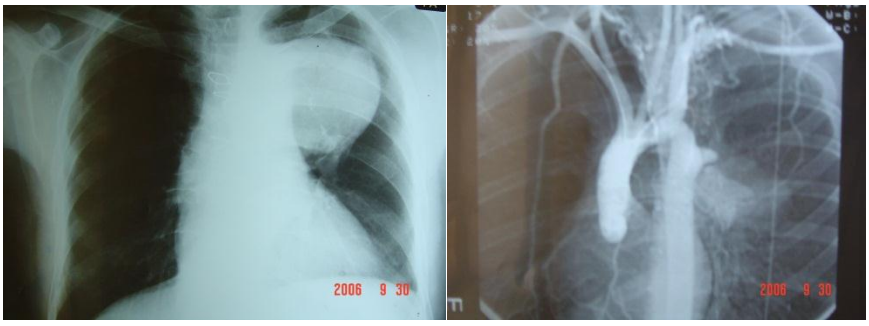
1. **Tissue Valves (Bio prosthesis)**
   a. No need to use long term anticoagulation.
   b. Limited and unpredictable durability
   c. When to use tissue valves?
      i. Old patients
      ii. Patient with contraindication to anticoagulants i.e. bleeding disorders
      iii. Non-compliant patients to anticoagulants e.g. psychiatric patients
      iv. Pregnant woman due to the teratogenic effect.

2. **Mechanical Valves**
   a. Anticoagulation for life ⓘ
   b. Prolonged durability

∗ Complications of prosthetic valves:
   1. Thrombosis
   2. Bleeding complications (1,2 Anticoagulant related complications)
   3. Infective endocarditis
   4. Paravalvular leak
   5. Degeneration of biological valves

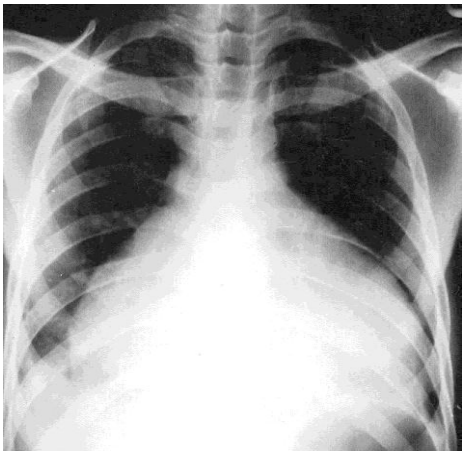
5 THORACIC AORTIC DISEASE

1. Thoracic aortic aneurysm
   a. Symptoms are usually due to pressure on surrounding structures.
2. Aortic dissection:
   a. Tear in the intima allowing blood to enter and flow in a false channel.
   b. There are 2 lumens separated by the dissecting membrane.



6 PERICARDIAL EFFUSION

- Progressive accumulation of fluid inside the pericardial cavity, may compress the cardiac chambers.
- Etiology:
  - Traumatic
  - Pericarditis
  - Malignancy
  - Uremia, post irradiation
  - Postoperative.
- Investigations:
  - Plain x-ray chest
  - Echocardiography
  - CT scan
- Management:
  - Treat the cause
  - Aspiration
  - Pericardiostomy (if the fluid is not accessible)



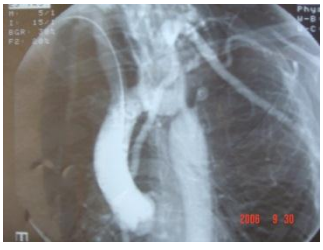
7 CARDIOTHORACIC EMERGENCY

1. Chest pain (causes):
   a. Myocardial ischemia
   b. Pulmonary embolism
   c. Aortic dissection
   d. Tension pneumothorax
   e. Rupture esophagus
2. Acute dyspnea (causes):
   a. Myocardial infarction
   b. Pulmonary embolism
   c. Spontaneous pneumothorax
   d. Bronchial asthma
   e. F.B. (foreign body) aspiration
   f. Stuck mechanical valve.
3. Chest trauma:
   a. Flail chest
   b. Traumatic hemo/pneumothorax
   c. Hemopericardium

8 CONGENITAL HEART DISEASES

8.1 ACYANOTIC:

1. Patent ductus-arteriosus
2. Co-arctation of the aorta
3. Pulmonary stenosis
4. Atrial septal defect (ASD)
5. Ventricular septal defect (VSD)



8.2 CYANOTIC:

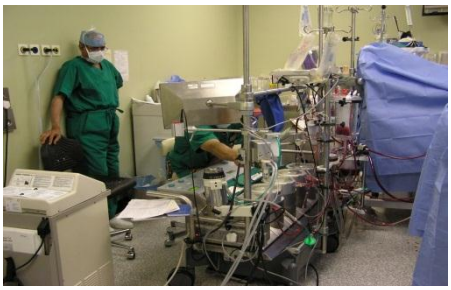
1. Tetralogy of Fallot (VSD, overriding aorta, pulmonary stenosis, RV hypertrophy)
2. Transposition of the great vessels
3. Tricuspid atresia
4. Total anomalous venous drainage
5. Truncus arteriosus

9 BASIC PRINCIPLES OF CARDIAC SURGERY

1. Adequate Exposure
   - Full or Partial Sternotomy / Thoracotomy / Robotic or Endoscopic
2. Bloodless Operative Field
   - Suction and re-transfusion / Snaring or clamping of bleeding vessels
3. Static Operative Target
   - Cardiac Arrest / Ventricular Fibrillation / Mechanical Stabilizers
4. Preservation of body perfusion
   - Use of Heart Lung Machine / Off-pump Techniques
5. Preservation of Myocardium
   - Off-pump Techniques / Hypothermia / Cardiac Arrest with cardioplegia

10 HEART LUNG MACHINE

- Components :
  1) Roller pumps
  2) Blood Reservoir (cardiotomy reservoir)
  3) Oxygenator
  4) Heater-cooler unit
  5) Tubing and Monitoring console etc



- Limitation/Problems :
  1) Requires full anticoagulation
  2) Can cause micro embolism
  3) Initiates Systemic Inflammatory Response

11 PREOPERATIVE ASSESSMENT

- Evaluation of patients referred for cardiac surgery aims to answer the following questions:
  - Is surgery appropriate for the condition?
  - Is the patient fit to undergo the planned operation?
  - Is there any comorbidities that may affect the operative management?
- Approach:
  1) History
  2) Physical examination
  3) Chest x-ray

4) E.C.G.
5) Laboratory investigations

- Pre-Operative Investigations for Cardiac Surgery
  - Full Blood Count
  - Blood Biochemistry
  - ECG
  - Chest X-ray
  - Pulmonary Function Tests.
  - Other test according to systemic review of patient
  - Echocardiography
  - Angiography
  - Carotid Duplex Scan
  - Peripheral Duplex Scan
- Usual Duration of Stay in Hospital
  - One day before surgery
  - 3-6 hours OR time
  - One day in ICU
  - 4-5 Days in Ward
  - Total 5-7 days

Carotid Duplex scan and Peripheral Duplex scan:

To check if other vessels are affected too

This is for a standard non complicated case.

Duration may increase due to complications and in older patients.

12 SUMMARY& MCQS

12.1 INDICATIONS OF CARDIAC SURGERY

IHD	1. Failure of medical therapy or percutaneous intervention. 2. Left main coronary artery disease 3. 3-vessel disease with left ventricular dysfunction 4. Mechanical complications of myocardial infarction. 5. Associated valve disease
Valvular heart disease	<u>Mitral stenosis</u> 1. When symptoms of severe SOB appear like: exertional dyspnea, pulmonary hypertension, hemoptysis 2. Severe mitral stenosis : less than 1 cm opening 3. Left atrial thrombus
	<u>Mitral regurgitation</u> Significant shortness of breath : Symptomatic, dilated left ventricle, diminished ejection fraction
	<u>Aortic stenosis</u> 1. Symptoms (angina, shortness of breath, syncopaal attacks) 2. Severe aortic stenosis
	<u>Aortic regurgitation</u> 1. Symptomatic patients 2. Progressive left ventricular dilatation
Thoracic aortic disease	1. Aortic aneurism 2. Aortic dissection
Pericardial effusion	Drainage by catheterization unless the fluid is not accessible

12.2 MCQS

1) For coronary artery bypass surgery, which one of the following conduits has proved to be the best in long term patency?

a) Gastroepiploic artery
b) Internal mammary artery
c) Radial artery
d) Reversed saphenous vein

2) Which one of the following coronary arteries is the most commonly involved in ischemic heart disease?

a) Left anterior descending artery (LAD)
b) Main coronary artery
c) Obtuse marginal branches
d) Right coronary artery

3) the most important pre-operative study in evaluating patients for coronary artery bypass grafting that will serve as a road map for the surgeon is?

a) Cardiac catheterization
b) ECG
c) Thallium scan
d) trans-thoracic echocardiography

🗝 Answers: 1;B , 2;A , 3;A

PRESENTATION AND MANAGEMENT OF COMMON THORACIC DISEASES

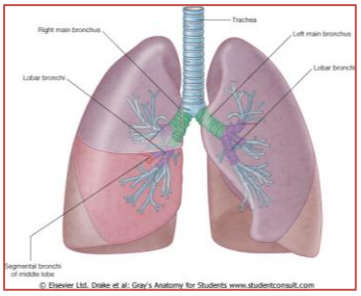
1 OVERVIEW: STRUCTURE AND DEVELOPMENT OF THE LUNGS

1.1 EMBRYOLOGY

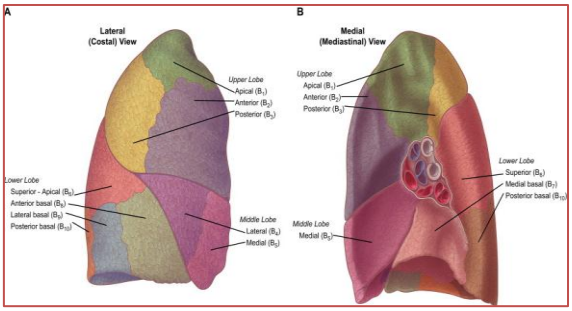
- Bronchial system
- Alveolar system

1.2 ANATOMY

- **Lobes and fissures:**
  - The right lung is divided into 3 lobes by the oblique and horizontal fissures.
  - The left lung is divided into 2 lobes by the oblique fissure



- **Segments**
- **Blood supply:**
  - Lungs don't receive any vascular supply from the pulmonary vessels (pulmonary artery or vein)
  - Blood is delivered to lung tissue via the bronchiole arteries
  - Vessels evolve from aortic arch
  - Travel along the bronchial tree
- **Airways:**
  - Trachea, primary bronchi, secondary bronchi, tertiary bronchi out to 25 generations
  - All comprised of hyaline cartilage
  - <u>Trachea:</u>
    - Begins where larynx ends (about C6)
    - 10 cm long, half in neck, half in mediastinum
    - 20 U-shaped rings of hyaline cartilage, keeps lumen intact but not as brittle as bone
    - Lined with epithelium and cilia, which work to keep foreign bodies/irritants away from lungs
  - <u>Bronchioles:</u>
    - First level of airway surrounded by smooth muscle; therefore can change diameter as in bronco-constriction and bronco-dilation
    - Terminal bronchioles
    - Respiratory bronchioles
    - 3-8 orders
  - <u>Alveoli</u>



Bronchopulmonary segments

Primary bronchi:

- Right primary bronchus is shorter, wider, and more vertical than the left primary bronchus. Therefore, when foreign bodies are aspirated, they often lodge in the right main bronchus.

Bronchopulmonary segments:

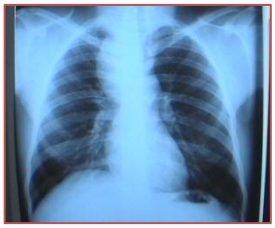
Each of the tertiary bronchi serves a specific bronchopulmonary segments.. There are 10 segments in the right lung and 8-10 segments on the left and each have their own artery. Each segment is a discrete anatomical and functional unit, so a segment can be surgically removed without affecting the function of the other segments.

2 LUNG DISEASES

Congenital	Infectious	Tumors
• Agenesis • Hypoplasia • Cystic adenomatoid malformation • Pulmonary sequestration • Lobar emphysema • Bronchogenic cyst	• Lung Abscess • Bronchiectasis • Tuberculosis • Aspergillosis • Hydatid cyst	• Malignant - Primary lung carcinoma - Secondary lung carcinoma • Benign

2.1 CONGENITAL

- **Agenesis**:
  - Absence of the lungs
- **Hypoplasia**:
  - Incomplete development of the lungs
- **Congenital Cystic Adenomatoid Malformation (CCAM):**
  - A cystic area within the lung that stems from abnormal embryogenesis. Usually an entire lobe of lung is replaced by non-functioning cystic area of abnormal lung tissue.
- **Pulmonary Sequestration:**
  - It consists of a nonfunctioning mass of normal lung tissue that lacks normal communication with the airways, and often receives its own arterial blood supply from the systemic circulation (esp. thoracic aorta). Most of the time it is located in the left lower lobe. Treated surgically to prevent infections.
- **Lobar Emphysema:**
  - Over-inflation of a pulmonary lobe (replacement of a whole lobe by bullae), which may compress the other remaining normal lobes. Air enters the lungs but cannot leave easily causing respiratory function to decrease. Treated surgically (lobectomy) in serious cases to allow normal lung to inflate.
- **Bronchogenic Cysts:**
  - They can be located:
    - In the mediastinum most commonly attached to trachea or below the carina (paratracheal or subcarinal)
    - Or within the lung parenchyma (intraparenchymal)



  - Clinical features:
    - They consist of semi-solid cartilaginous that secretes cheese like material, which is prone to infections. It may also result in hemorrhage and compression of the surrounding structures (i.e. trachea, aorta, and esophagus) patient then complains of SOB, stridor, cough and dysphagia.
    - Could be asymptomatic found accidentally on CXE as a smooth opacity.
    - They may transform to malignant adenocarcinoma.

CCAM:

Presenting clinical features include: respiratory distress and recurrent respiratory infections. The usual appearance of CCAM on CXR is a mass containing air-filled cysts (Swiss cheese pattern),

Pulmonary Sequestration:
Sequestrations are classified anatomically into intralobar and extralobar. Usually presents in adolescence or late childhood as repetitive chest infections that fails to respond to medical treatment. It appears on CXR as an opaque mass. Diagnosed by MRI/arteriography.

Lobar Emphysema:

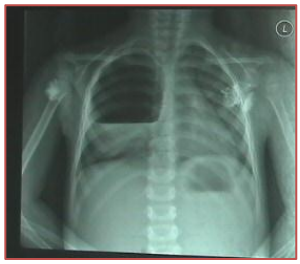
Diagnosed by respiratory symptoms and CXR, which shows over-inflation of the affected lobe (radiolucency).

- Treatment: surgical excision is done to confirm diagnosis, avoid complications such as malignancy, rupture, infection, and compression on vital organs.

2.2 INFECTIOUS

2.2.1 LUNG ABSCESS

- **Causes**:
  - As a complication of pneumonia, bronchial obstruction (by tumor or inhaled foreign bodies esp. in children) bacteremia, and septic emboli.
- **Clinical features**: copious production of foul smelling sputum
- **Investigations**:
  - CXR (air-fluid level)
  - CT scan



- **Treatment:**
  - Antibiotics
  - Drainage: internal and external
  - Pulmonary resection (surgical treatment)
    - *Indications*: ⓘ
      1. Failure of medical treatment
      2. Giant abscess (>6 cm)
      3. Hemorrhage (patient presents with hemoptysis)
      4. Inability to rule out carcinoma (e.g. a 65 y/o very ill smoker can have lung cancer superimposed by abscess)
      5. Rupture with resulting empyema
- Type of resections:
  - Lobectomy (main) or bilobectomy (2 lobes)
  - Pneumonectomy

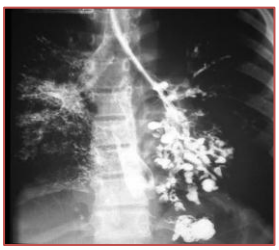
Clinical Features of Lung Abscess:

- Gradual onset
- Productive cough
- High fever
- Night sweats
- Weight loss & lethargy
- Chest pain (pleuritc)

Empyema= collection of pus in an anatomical cavity (e.g. pleural empyema)

2.2.2 BRONCHIECTATSIS

- **Definition**: Bronchial dilatation, usually affecting the lower lobes
- **Cause:**
  - Congenital (i.e. cystic fibrosis and immotile cilia syndrome)
  - Infection (repeated pulmonary infections and childhood infections)
  - Obstruction (by tumors/ inhalation of foreign bodies)
- **Clinical features:**
  - Cough mostly in morning with copious amounts of sputum
  - Dyspnea
  - Hemoptysis (50%)
  - Clubbing (it is a chronic disease)



- **Types:**
  - Cystic
  - Cylindrical
- **Investigations**:
  - Bronchogram (invasive)
  - CT scan (more accurate)
  - Bronchoscopy (not commonly used nowadays)
  - CXR (cystic formation)

Immotile Cilia Syndrome:

-Bilateral

-Lung transplant is needed in old age

Development of childhood vaccinations has reduced the incidence of bronchiectasis due to whooping cough, measles, and TB.

- **Treatment**:
  - Medical:
    - Resolve most cases (bronchodilators, antibiotics, and physiotherapy with postural drainage)
  - Surgical indications: ⓘ
    - Failure of medical treatment
    - Cystic dilatation (not cylindrical which is treated medically)
    - Localized disease
    - Not perfused (assessed by V/Q scan), most of cystic bronchiectasis are not perfused whereas most of cylindrical are perfused.

ⓘ E.g. a child inhales a foreign body → bronchial tree obstruction (> right main bronchus) → mom explains that her child was ok 6 months ago but now he has been getting repetitive chest infections/SOB/wheezing → suspect foreign body inhalation bronchiectasis

2.2.3 TUBERCULOSIS

- 30,000 new cases occur annually in USA
- **Cause**:
  - Pulmonary
  - Extra-pulmonary
- **Investigations:**
  - CXR (more in apex)
  - AFB sputum culture (if positive confirms TB)
  - Tuberculin skin test (latent TB)
  - Bronchoscopy
  - Chest CT scan (infiltration, abscess formation, lymph nodes)
  - Mediastinoscopy (caseating granuloma)
- **Treatment:**
  - Medical:
    - Effective in most cases
  - Surgical indications ⓘ
    - Failure of medical treatment
    - Destroyed lobe or lung
    - Pulmonary hemorrhage
    - Persistent open cavity with positive sputum
    - Persistent broncho-pulmonary fistula

ⓘ Cystic? Localized? Non-perfused? > Surgical

Cylindrical? Bilateral? Perfused? > Medical

Left bronchus syndrome:

Chronic condition that leads to unilateral post TB lung destruction as a result of untreated/resistant TB.

Fibrosis→loss of space→loss of ventilation on left side → left lung is smaller, infective, and bronchioectatic pulling the trachea towards it.

Don't operate on active open TB b/c of the risk of spread of infection. Manage them medically first for 4 weeks before surgery.

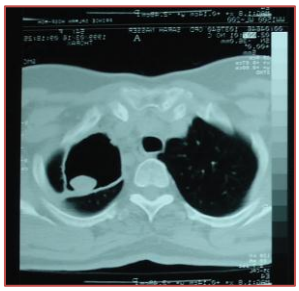
2.2.4 ASPERGILLOSIS

- **Cause**:
  - Aspergillus fumigatues, Aspergillus niger
- **Mode of Transmission**
  - Inhalation of airborne conidia
  - Contaminated water (during showering)
  - Nosocomial infections (hospital fabrics and plastics)
  - Esp. in immunocomprimsed individuals
- **Forms:**
  - Allergic (allergic bronchopulmonary aspergillosis)
  - Saprophytic (aspergilloma/mycetoma)
  - Invasive
- **Clinical features**
  - Aspergilloma/mycetoma
    - Comes with a warning sign of hemoptysis
    - At this stage, the doctor must act quickly because morbidity and mortality are very high in these patients

Saprophytic Aspergillosis:

Characterized by Asp infection without tissue invasion. The most common underlying causes are TB and sarcoidosis.

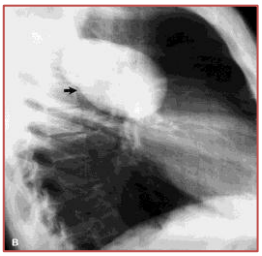
- Hemoptysis (patient with preexisting disease)
- Chronic productive cough
- Sometimes found accidentally on CXR



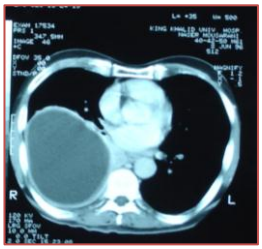
- **Investigations:**
  - Skin test
  - Sputum (fungal culture)
  - Biopsy (invasive)
  - CXR (radiolucent)
  - CT scan (cavity with aspergilloma complex and air crescent sign, DDx TB)
- **Treatment:**
  - Medical (anti-fungal)
  - Surgical indications:
    - A significant aspergilloma (with serious clinical features)
    - Hemoptysis
    - Types of resection: depends on the affected side
      - Segmentectomy
      - Lobectomy (mainly)
      - Pneumonectomy

2.2.5 HYDATID CYST

- **Definition**:
  - Parasitic infestation by **Echinococcus granulosus** (tapeworm)
  - Hosts: dogs, cats, and sheep (e.g. by eating raw contaminated sheep liver)



- **Clinical presentation:**
  - Asymptomatic (accidentally found)
  - Symptoms are the result of compression by the cyst (e.g. dyspnea)



- **Diagnosis**
  - Skin test (Casoni's reaction)
  - CXR
  - CT scan (a chronic cyst will appear calcified on CT)
  - High echinococcus titers and other serologic tests
  - Routine blood work (nonspecific)

- **Treatment**
  - Radical surgical excision (cyst resection or partial affected organ resection) coupled with chemotherapy using albendazole and/or mebendazole before and after surgery.
  - If multiple cysts are present in multiple organs surgery becomes impractical and chemotherapy is indicated.

Transmission:

Dog (definitive host) → sheep (intermediate host)→ human by eating raw sheep liver → enteric system→portal system→liver→IVC→ heart and lungs→systemic!

- The liver is the most common organ involved, followed by the lungs (brain, bones, kidneys... can also be involved)

Hydatid cyst layers:

1. The outer pericyst, composed of host cells that are formed as a reaction to the parasite (false layer).

2. The middle laminated membrane (external layer of cyst)

3. The inner germinal layer of cyst where the scolices are produced and contained.

2+3 form the true wall of the cyst

- Surgeon must be careful when doing this procedure, because each cyst contains millions of scolex (highly infective) so if ruptured it'll spill millions of scolex into surrounding cavities which leads to the formation of new cysts!
- Injection of scoliodal agents such as hypertonic 20% saline is used during surgery to kill scolex.
- Rupture of the cyst depends on the size of feeding bronchus, if it was big a small cyst can get ruptured, but if the feeding bronchus was small, the cyst won't rupture.

2.3 TUMORS

- Benign
- Malignant
  - Primary
  - Secondary

2.3.1 PRIMARY LUNG CARCINOMA

- **Incidence:**
  - Worldwide, lung cancer is the most common cause of cancer death.
- **Risk factor:**
  - Smoking (most important)
  - Others: radiation, industrial chemicals, diet, genetic factors, asbestos, and radon
- **Pathology:**
  - **Non-small cell carcinoma**
    - Adenocarcinoma
    - Squamous cell carcinoma
    - Large cell carcinoma
  - **Small cell carcinoma**
- **Classification:** *must differentiate between SCLC & NSLC because treatment approach is completely different.*

NSCLC	SCLC
• Epithelial origin • 75-80% 1. **Adenocarcinoma** (40%) ○ Peripherally located **2. Squamous cell carcinoma** (30%) ○ Centrally located **3. Large cell carcinoma** (9%) ○ Peripherally located • Treatment: ⓘ ○ Early**:** Surgery (+/- adjuvant chemotherapy) ○ Intermediate: Neoadjuvant chemotherapy + surgery ○ Late/metastasis**:** NON-surgical **(**chemo/radiotherapy + palliative management)	• Neuroendocrine origin • 20-25% • Centrally located • Poor prognosis • Patient usually presents with systemic disease (e.g. large mediastinal LAD) • Mostly discovered late when tumor has already metastasized • Treatment: ⓘ ○ NON-surgical (chemotherapy only +/- radiotherapy)

Definitions:

- Horner's syndrome:

Characterized by the classic triad of miosis partial ptosis, and loss of hemifacial sweating (i.e., anhidrosis).

- SVC obstruction syndrome:

SOB most common symptom and facial/arm swelling.

- Pancoast tumor:

Superior sulcus tumor injury of C8 & T1 causing shoulder pain that radiates to arm

- **Clinical Features:**
  - Asymptomatic → accidentally on CXR
  - Symptomatic
    - Lung manifestations (most commonly cough, hemoptysis, SOB...)
    - General manifestations (loss of appetite, fever, weight loss, fatigue)
    - Surrounding structures:
      - Recurrent laryngeal nerve (e.g. hoarseness)
      - Esophagus (dysphagia)
      - C8, T1 nerve (arm pain/numbness)
      - Sympathetic (esp. 1st sympathetic ganglion: Horner's syndrome)
      - Pleural pain
      - SVC obstruction syndrome
    - Distal Para-neoplastic syndrome:
      - PTH (hypercalcemia)
      - ADH (hyponatermia)
      - ACTH (Cushing's syndrome)
      - Hypertrophic pulmonary osteoarthropathy (Pain and swelling of joints that doesn't respond to medical treatment and improves once tumor is resected)
- **Investigations:**
  - CXR (find a previous CXR for comparison, if lesion is stable for more than 2 years, it is most likely benign)
  - Bronchoscopy (for central lesions as with squamous lung CA and SCLC)
  - Transthoracic needle aspiration (for peripheral masses, CT guided)
  - CT scan (best modality for **staging** extent of metastasis) ⓘ
  - MRI (poor modality in staging, its helpful to rule out involvement of major structures in the apex: brachial plexus, vertebral column, and spinal cord e.g. superior sulcus tumor)
- **Staging:**

NEW INTERNATIONAL REVISED STAGE GROUPING

Stage 0	TIS
Stage IA	T1, NO, MO
Stage IB	T2, NO, MO
Stage IIA	T1, N1, MO
Stage IIB	T2, N1, MO
	T3, NO, MO
Stage IIIA	T1-3, N2, MO
	T3, N1, MO
Stage IIIB	T4, Any N, MO
	Any T, N3, MO
Stage IV	Any T, Any N, M1

Lymph node staging:

N1 → inside the lung

N2 → outside the lung toward mediastinum hilum

N3 →supraclavicular or to the other side

Neoadjuvant chemotherapy:

Chemotherapy before the surgery (to downsize the tumor)

Adjuvant chemotherapy:

Chemotherapy after the surgery

- **Management**
  - Depends on:

- Stage (tumor size, LN involvement, metastasis to liver, bone, and brain) done by CT.
- Cell type
- Patient physical fitness (tumor might be of an early stage but the patient has other comorbidities)

- NSCLC:
  - **Surgical** (early stages) +/- adjuvant chemotherapy
  - Neoadjuvant chemotherapy + surgery (intermediate)
  - **Radiotherapy and chemotherapy** (late stages)
- SCLC:
  - **Chemotherapy** (treatment of choice)
  - **Radiotherapy**
  - NON-surgical (because tumor is usually discovered late, when metastasis is extensive and the patient develops systemic symptoms with massive mediastinal lymphadenopathy)

2.3.2 SECONDARY LUNG CARCINOMA

- Neoplasms that have spread from a primary lesion in another organ
- Secondary lung tumors appear as solitary lung nodules (well-marginated, single mass <3 cm, intraparenchymal opacity)
- **Solitary Lung Nodule DDx:** *(coin lesions)*
  - Primary Carcinoma
  - Tuberculous Granuloma
  - Mixed tumor
  - ° 2 Carcinoma (metastatic)
  - Miscellaneous
- **Hamartoma - Carcinoid**
  - Age: hamartomas occur primarily in adults >50 y/o
  - Sex: Males 3 times more likely than females
  - X-ray (usually peripherally located)
    - Size: usually small <4cm in diameter, rounded
    - Time: grows slowly
    - Calcification: sometimes with varying patterns

Benign Vs. Malignant solitary pulmonary nodules:

Benign →

Age <50, nonsmoker, size <2 cm, no growth over 2 year period, circular and regular shaped, central laminated/concentric calc.

Malignant →

Age >50, smoker, size >3cm, steady growth, irregular nodule or speculated margins, stippled/eccentric calc.

Hamartomas are the most common type of benign lung tumors, accounting for 75% of all benign lung tumors and most of them are asymptomatic.

3 TRAUMA

- RTA (road traffic accidents)
- Fracture ribs (simple/complicated)
  - Most common blunt thoracic injuries
- Haemothorax:
  - Accumulation of blood in pleural cavity
  - Appears as radio-opacity on CXR.
  - Cause is mostly traumatic
- Pneumothorax
- Flail chest:
  - Fractures of several adjacent ribs in two or more places producing a free unstable segment of chest wall that results in paradoxical movement. There is usually associated lung contusion.
- Lung contusion and ARDS (no surgery needed unless massive bleeding)

Traumatic pneumothorax:

Can result from either penetrating or non-penetrating chest trauma. Iatrogenic pneumothorax is one of the most common causes (e.g. during transthoracic needle aspiration procedure). Treat simple pneumothorax with a chest drain if large/symptomatic.

4 MEDIASTINUM

4.1 ANATOMY:

- Mediastinum is the space in the thoracic cavity between the lungs
- Boundaries →
  - Superior: thoracic inlet
  - Inferior: diaphragm
  - Anterior: sternum and costal cartilages
  - Posterior: thoracic spine
  - Lateral: mediastinal pleura
- Divisions:
  - Superior mediastinum (above the sternal angle)
  - Inferior mediastinum (below the sternal angle) subdivided into: Anterior, Middle, Posterior
- Access:
  - Mediastenscopy
  - Mediastenotomy

4.2 MEDIASTINAL MASS LESIONS:

Anterior Mediastinum	Middle Mediastinum	Posterior Mediastinum
5 TS ⓘ a. **T**eratoma b. **T**hyroid (retrosternal goiter) c. **T**B lymphadenitis d. **T** cell lymphoma e. **T**hymoma	**Cysts** ⓘ a. Pericardial cyst b. Bronchogenic cyst	**Neurogenic tumors** ⓘ (E.g. dumbbell tumor of neurofibroma, paravertebral mass…)

4.2.1 THYMOMA

- **Incidence**:
  - The commonest tumor of anterior mediastinum ⓘ
  - Peak 40-60 y/o
  - M: F (1:1) equally affected
- **Pathology**:
  - Classification:
    - Epithelial
    - Lymphocytic
    - Lymphoepithelial
    - Spindle cell
  - Benign Vs. malignant
  - Stages: I, II, III, IV
- **Clinical Features:**
  - Asymptomatic
  - Symptomatic
    - Mass effect → SVC syndrome, dysphagia, and cough…)

        - Systemic effect → associated autoimmune disorders, most commonly myasthenia gravis 40-50%
- **Investigations:**
    - For all cases →
        - CXR
        - CT scan (can be indicative of malignancy)
        - Biopsy
    - For selected cases →
        - Bronchoscopy, Esophagoscoy
        - Angiogram
- **Treatment:**
    - Benign → complete excision
    - Malignant:
        - Complete excision if possible
        - If non-resectable (i.e. invasive and large) → Preoperative (neoadjuvant) chemotherapy and/or radiotherapy may be used to decrease the size and improve resectability or incomplete resection.

5 CHEST WALL

- **Deformities:**
    - Pectus excavatum: caved-in or sunken appearance of the chest
    - Pectus carniatum: protrusion of the sternum & ribs (pigeon chest)
- **Infections** (e.g. abscess, empyema, costochondritis…)
- **Chest wall tumor:** Those that grow on the ribs and sternum (>benign)
- **Thoracic outlet syndrome**

Thoracic outlet syndrome: compression at the sup thoracic outlet placing pressure on nerves and vessels passing through, with signs and symptoms manifesting in arms and hands esp. pain.

5.1 PLEURA

- **Spontaneous pneumothorax**
- **Pleural effusion**: collection of fluid in the pleural cavity
- **Empyema:** collection of pus in the pleural cavity
- **Mesothelioma:** rare cancer, usually caused by asbestos exposure

5.1.1 PNEUMOTHORAX

- **Definition:** air in the normally airless pleural space
- **Classification:** Traumatic and non-traumatic (spontaneous)

Spontaneous Pneumothorax	Tension Pneumothorax
➢ ***Primary/simple pneumothorax*** - Occurs without any underlying lung disease - Spontaneous rupture of air-filled sacs on the lungs causing escape of air from the lung into pleural space and lung collapse ➢ ***Secondary/complicated pneumothorax***	- Life threatening condition. - Accumulation of air under positive pressure in the pleural space collapses the ipsilateral lung and shifts the mediastinum away. **Causes:** 1. Mechanical ventilation with associated barotrauma 2. CPR

- As a complication of underlying lung disease, most commonly COPD **Clinical features:** 1. Sudden ipsilateral chest pain 2. Dyspnea and cough 3. Decreased breath sounds over affected side 4. Hyperresonance over the chest 5. Decreased tactile fremitus 6. Mediastinal shift *toward* side of pneumothorax **Diagnosis**: <u>CXR</u> **Treatment:** ➢ *Primary spontaneous pneumothorax* - <u>If small and patient is asymptomatic →</u> a. Observation (should resolve spontaneously in 10 days) reassess with CXR b. Small chest tube may benefit some patients - <u>If larger and/or patient is symptomatic →</u> a. Administration of supplemental oxygen b. Chest tube insertion to allow air to be released ➢ *Secondary spontaneous pneumothorax* **a.** Chest tube drainage	3. Trauma **Clinical features:** 1. Hypotension and tachycardia 2. Distended neck veins 3. Shift of trachea away 4. Decreased breath sounds on affected side 5. Hyperresonance **Diagnosis:** clinically (no time for CXR!) **Treatment:** Medical emergency! If tension isn't relived patient is likely to die from hemodynamic compromise. <u>Immediately decompress the pleural space via large-bore needle or chest tube</u>

5.2 AIRWAY

- **Congenital tracheal anomalies**
- **Tracheal stenosis:** narrowing of the trachea that is caused by an injury or birth defect.
- **Tracheostomy**

5.3 SURGERY

- Thoracotomy
- Thoracoscopy
- Sternotomy
- Analgesia

6 MCQS

1. Which one of the following thoracic diseases is treated by chest tube insertion?

a. Caveating malignant lung mass
b. Cystic bronchiectasis
c. Large pneumothorax
d. Lung abscess

2. A 22 y/o female is referred for evaluation of 2-cm posterior mediastenal mass discovered on routine chest radiograph. What is the most likely diagnosis?

a. Bronchogenic cyst
b. Lymphoma
c. Neurogenic tumor
d. Thymoma
e. Adenocarcinoma

3. Which one of the following is the most common blunt chest injury?

a. Pneumothorax
b. Hemothorax
c. Sternal facture
d. Rib fracture
e. Pulmonary contusion

🗝 ***Answer key → 1;C , 2;C , 3;D***

PEDIATRIC UROLOGICAL CONDITIONS

1 INTRODUCTION

- Most common anomalies of all organ systems
- 10% of the population have some type of urogenital anomaly
- 14:1000 births have an antenatal diagnosis of urogenital anomaly
- The antenatal diagnosis is done by ultrasound after 28 weeks of gestation because at 24-28 weeks the urinary system starts to be clear on ultrasound

2 ANTENATAL HYDRONEPHROSIS (ANH)

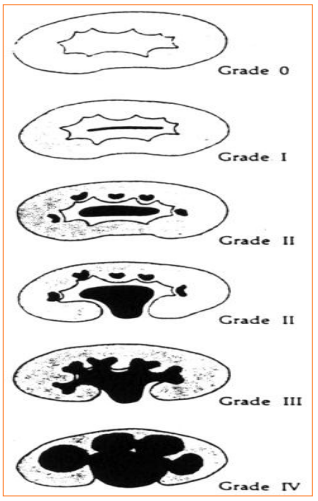
2.1 INTRODUCTION:

- This is hydronephrosis detected during pregnancy by ultrasound
- It is a condition which has a differential diagnosis and causes. It is not a diagnosis on its own!
- Causes:
  - Pelviureteric junction obstruction (41%)
  - Ureterovesical junction obstruction (23%)
  - Vesicoureteric reflux (7%)
  - Duplication anomalies (13%)
  - Posterior urethral valves (10 %)
  - MCDK (Multicystic dysplastic kidney)
  - Others (6%)
- Grades of ANH
  - Specific details of grading are not important but you should know:
    - Urine appears black on ultrasound
    - Grade zero: normal kidney
    - From I-IV: 1 is the simplest and 4 is the worst
    - Grading depends on level of dilatation by ultrasound
  - So if you see hydronephrosis in an antenatal ultrasound the
    - diagnosis is: antenatal hydronephrosis
    - the differential diagnosis is: (any of the below)

Hydronephrosis is dilation of the pelvicalyceal system.

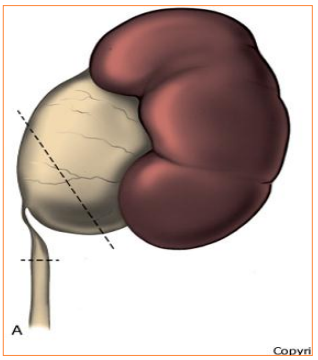
Hydroureter: dilatation of the ureter

Both renal pelvis and ureter: hydroureteronephrosis



2.2 1ST DDX: PELVIURETERIC JUNCTION OBSTRUCTION (PUJO)

- Dilation of the renal pelvis due to obstruction at the junction between the pelvis and ureter
- Because the obstruction is before the Ureter in these cases is usually not affected
- So on Ultrasound the renal pelvis is dilated and the ureter is normal
- It is the most common cause of ANH
- Etiology: (theories): Segmental muscular attenuation, Angulation, True Stenosis, Extrinsic compression, Crossing vessels; 20-30%.
- Associated findings:
  - Reflux in 5-10%

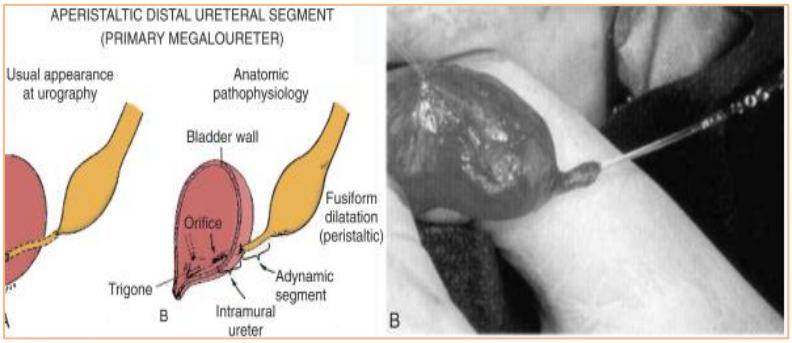


- Contralateral PUJO in 10%
- Contralateral agenesis in 5%

- Presentation:
  - More in males and occurs in the left side more than the right
  - Incidental in neonates by US or in infancy
  - If the diagnosis is missed during pregnancy or early infancy the child could come with symptoms like:
    - UTI
    - Pain
    - Mass
    - Hematuria
    - Stones
- Investigations include ultrasound, renal scans and VCUG (voiding cystourethrography)
- Indications of surgery:
  - Symptomatic patients
  - If the finding is incidental:
    - Neonates: worsening pattern or reduced renal function
    - Children: significant obstruction

2.3 2ND DDX: URETEROVESICAL JUNCTION OBSTRUCTION

- Also called megaureter or severely dilated ureter
- There is a narrow segment that causes the dilatation of the whole renal system
- Could be bilateral or unilateral but it is mostly unilateral
- It is different from PUJO in that the ureterovesical junction obstruction has a dilated ureter ⓘ

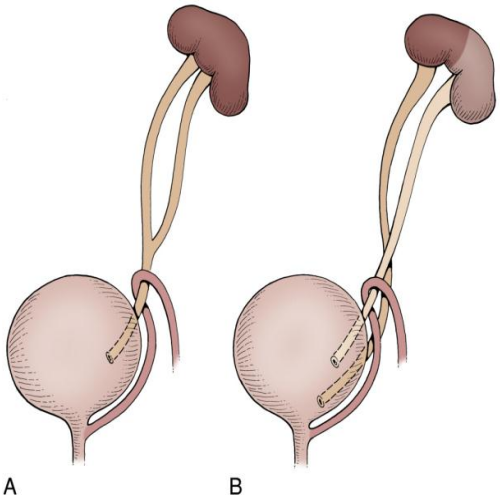


- Presentation:
  - Male 3:1 female
  - Left 3:1 Right
- Types:
  - Obstructive non refluxing
  - Obstructive refluxing
  - Refluxing non obstructive
  - Non refluxing non obstructive (adynamic ureter)
  - Treatment:
    - Obstruction: excision and reimplanting of the UVJ
    - Reflux: according to the same line of reflux management

2.4 3RD DDX: DUPLICATION ANOMALIES

2.4.1 RENAL AND URETERIC DUPLICATION

- Incidence is 1%, 1.6:1 F:M, 85% unilateral.
- Either two urethral buds meeting the meta-nephros or one ureteric bud that bifurcates.
- Associated with: reflux 43%, renal dilatation 29%, ectopic insertion 3%, infections and ureterocele.
- Duplication per se is of no clinical significance, but the associated anomalies may require intervention



A: incomplete duplication

B: complete duplication

- Usually only one ureter comes from each kidney to bladder: single system.

- Duplication Anomalies: 2 ureters coming from each kidney and going to bladder.

- Incomplete Duplication: both ureters meet in their way and one ureter go to bladder.

- Complete Duplication: 2 duplications.

- Embryological view:
  - Normally: One ureteral bud (early precursor of the ureter) meet future kidney.
  - In **Incomplete** (figure 1) ureteral duplication: Ureteral bud bifurcate into 2 after the generation they go to kidney as 2 ureters
  - In **complete** (figure 2) ureteral duplication: 2 separate ureteral buds come to meet metanephic kidney (future kidney)
  - If the both ureters coming to kidney and no reflex or obstruction > no harm to kidney but if there is obstruction as in uretrocele or ectopic ureter or reflex that will be harmful

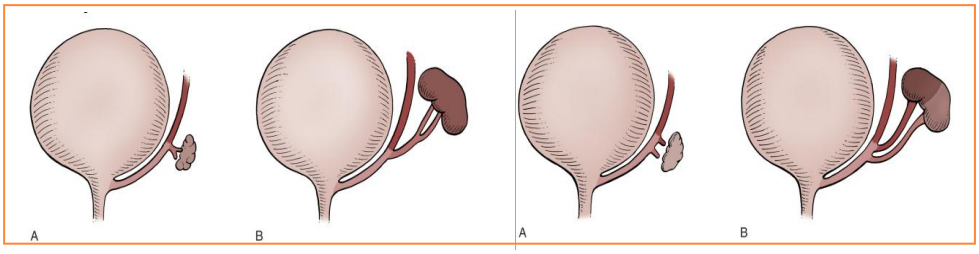


Figure 1 **Figure 2**

2.4.2 COMPLETE DUPLICATION FROM UPPER POLE OF KIDNEY TO LOWER POLE OF BLADDER

- Here a principle called the Weiger-Meyer Law takes place which states:

- The upper pole ureter (which drain the upper pole of the kidney) comes to lower part of bladder and lower pole ureter coming to upper part of the bladder

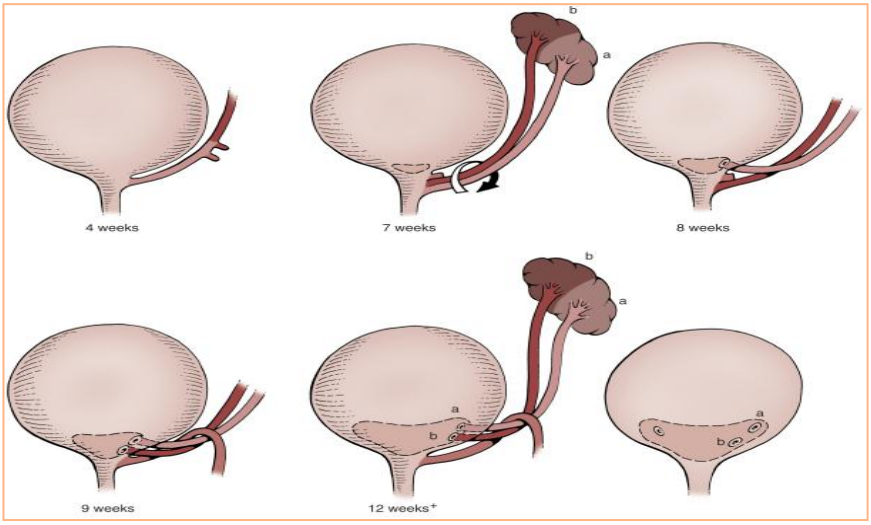
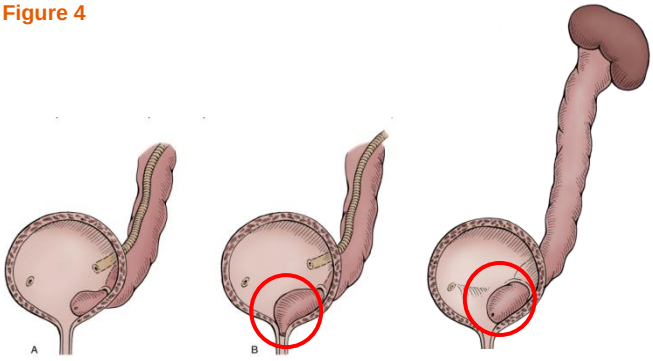


Figure 3

2.4.3 URETROCELE

- Commonest cause of urine retention in female infants ⓘ
- Cystic dilatation of the distal part of the ureter
  - This will lead to obstruction and the whole ureter will get dilated
- Associated with duplication anomalies (figure 3)
- This can be confused with Uretrovesical junction obstruction but the difference here is there is cystic dilatation which can be present in the bladder
- Sacculation of the terminal portion of the ureter has 2 types:
  - Orthotopic
    - Intravesical (inside bladder)
    - Simple OR adult type ureterocele.
  - Ectopic
    - Start in bladder and extended outside the bladder
    - Extravesical=duplex system OR infant type ureterocele.
  - In ectopic ureterocele it involve the upper pole system.

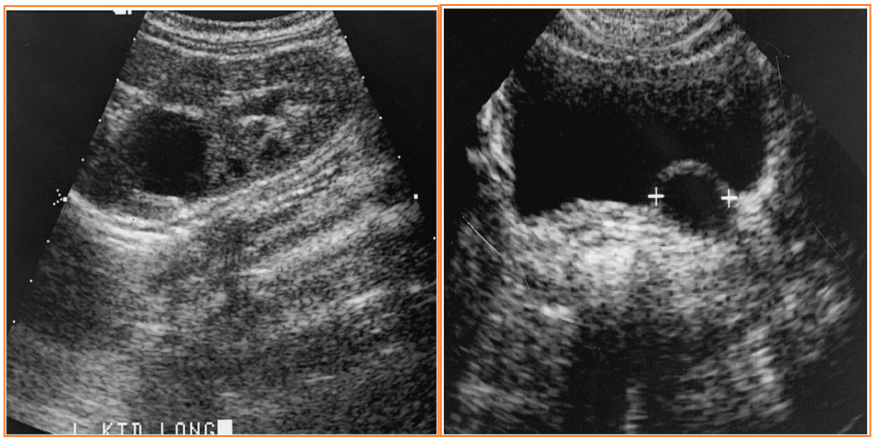
Figure 4



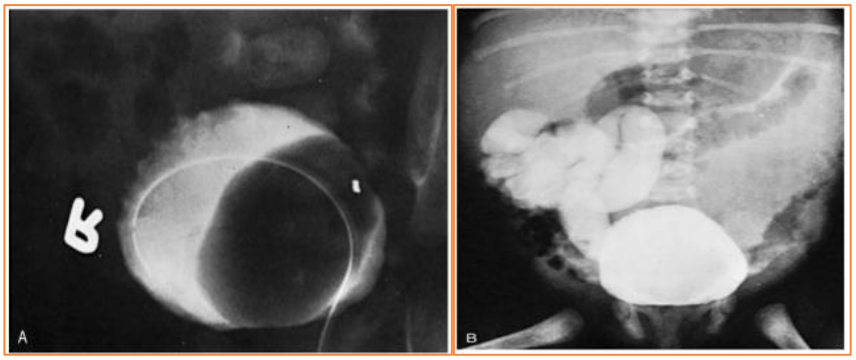
- Presentation:
  - Hydrouretronephrosis
  - 7:1 F:M, 10% bilateral, ectopic : orthotopic 4:1

- Usually detected by Antenatal (U/S) → we use MCUG (micturating cystourethrogram) to confirm the diagnosis ⓘ (figure 5)
- Symptoms include: urine retention, infection and presence of stones
- Intralabial mass:
  - One of the differential diagnosis for an Intralabial mass in females is a ureterocele

- Management:
  - Needs urgent intervention
  - Incision of ureterocele
  - Upper pole heminephroectomy
  - Excision of ureterocele and common sheath reimplant.



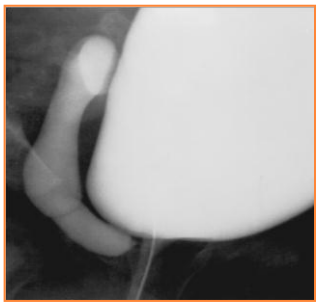
Cystic structure in bladder which is cystic dilation of distal part of ureter



MCUG, put catheter in bladder and use contrast if there is no abnormality the whole bladder will be white. But in ureterocele we will see filling defect ⓘ

Figure 5

2.4.4 ECTOPIC URETER

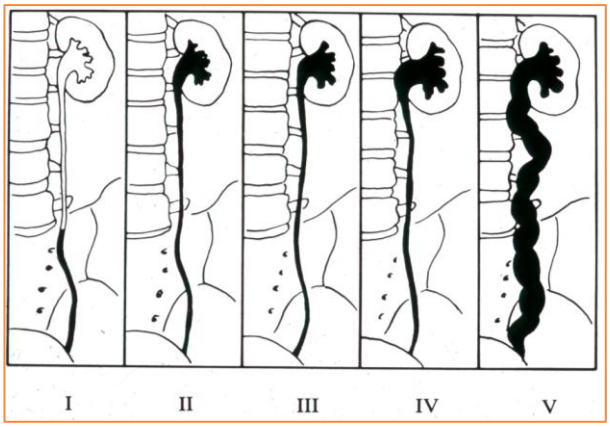


- Ureter does not implant in the bladder and stays outside
- Most commonly associated with duplex system and with ureterocele.
- Clinical picture depend on: associated anomalies, site and sex of the patient.
- Can be:
  - Simple ectopia: implanted in abnormal position

- Ectopic ureter: it is completely outside the bladder
- Investigations include IVP, VCUG, cystoscopy
- Renal scan asses the function of both renal poles in case of duplication.

2.5 4TH DDX: VESICOURETERIC REFLUX (VUR)

- There is a normal anti-reflux mechanism between the bladder and the ureter a "Flap Valve" which depends on: ⓘ
  - Ureter has an Oblique course as it enters the bladder.
  - Proper muscular attachments to provide fixation.
  - Posterior support to enable its occlusion.
  - Adequate submucosal length.
- The study to rule out reflux is MCUG ⓘ and it is also used for grading:
  - Normal: contrast in bladder
  - Grade I: confined to ureter, contrast is in the distal part of the ureter
  - Grade II: contrast reaches the kidney but there is no dilation
  - Grade III: Mild dilation of the renal pelvis and ureter without loss of calyces
  - Grade IV: moderate dilation but there is loss of calyces
  - Grade V: severe dilation and tortuous dilated ureter"



- **Resolution of reflux: Over 3 year follow up:**
  - 87% of Grade 1
  - 63% of Grade 2
  - 53% of Grade 3
  - 33% of Grade 4
- **Management:** The decision depends on :
  - The chance of spontaneous resolution (age and grade at presentation)
  - Breakthrough infection
  - Renal scarring and renal function
  - Compliance with medication
- **Medical management:**
  - **Patients with UTI (the most common presentation) and VUR is suspected.** ⓘ
    - Continue on prophylactic antibiotics after treatment till the VCUG is done.
  - **Patients for conservative management :**

Prophylactic dose: (1/3 of therapeutic dose only at night (24h), long term)

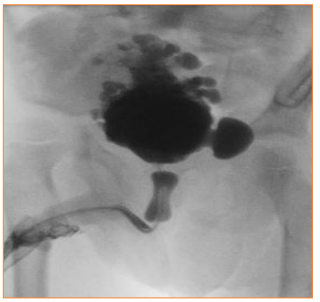
- Continue meticulously on prophylactic antibiotics and surveillance with urine culture and sensitivity, U/S ,and DMSA (dimercaptosuccinic acid) scan

2.6 5TH DDX: POSTERIOR URETHRAL VALVE (PUV)

Between anterior & posterior urethra during embryologically there is canalization from distal to proximal and from caudal to cephalic leading to complete tube without any narrowing,

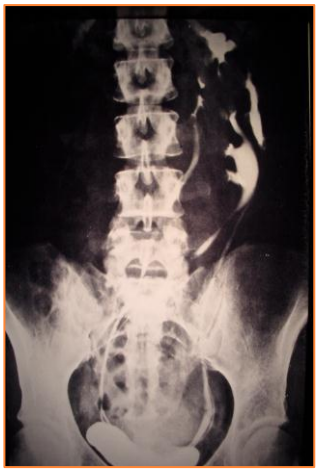
But in Posterior urethral valve incomplete canalization of urethra and leave small membrane (posterior urethral valve) which cause obstruction

- Incomplete canalization of the posterior urethra
- 1:5000 male infants.
- Most common cause of urine retention in male infants.
- 50% have renal impairment.
- The bladder and the kidneys developed under high pressure and resistance.
- The more proximal the valve the more sever the condition
- Associated findings:
  - Oligohydramnios ⓘ:
    - low amount of Amniotic fluid
    - No output of urine or little → Amniotic fluid
    - Low in Ultrasound because there is no secretion but <u>there is absorption</u>.
    - Obstruction of esophagus <u>no absorption</u> > Polyhydramnios.
  - Bilateral renal dilatation ⓘ
  - VUR: 40%
  - Valve bladder → loss of its Function and become abnormal bladder
    - The bladder is urogenic ⓘ because during pregnancy the detrusor muscle is replaced by collagen so no contraction of muscle occurs.
    - Baby start voiding during 24th week of gestation → so in Posterior urethral valve the baby will void against pressure so bladder will be large and trabeculated and urogenic
  - Renal impairment: in 30-50%, 25% of them will have renal transplantation in the future
  - The obstruction is not complete (narrowing or stenosis) because if its complete “severe” the patient will die in utero
- Presentation:
  - Antenatal (hydonephrosis)
  - Urine retention
  - UTI
  - Poor urinary stream
  - CRF; at late stage
- Investigations: Antenatal US, US, VCUG, Renal scan, renal function studies, Urodynamic studies.
  - MCUG: Posterior urethra dilated, normal anterior urethra ,bladder trabeculated and elongated (christmas tree bladder)



3 CONGENITAL ANOMALIES OF THE KIDNEY

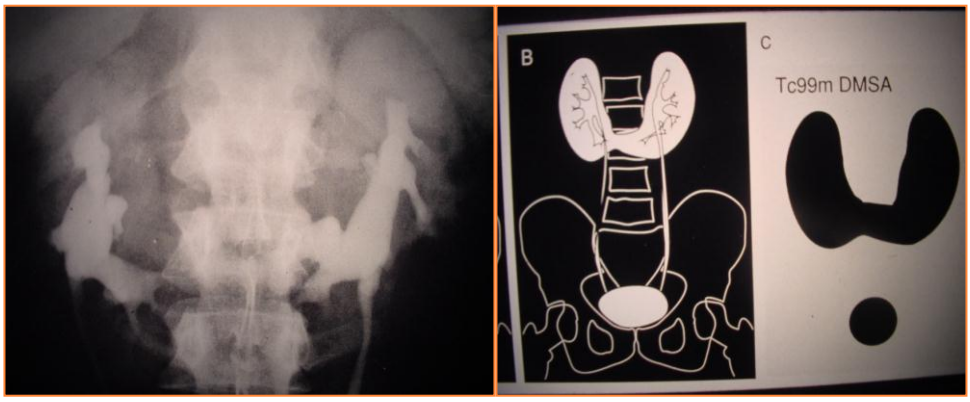
3.1 ANOMALY OF **POSITION**, NUMBER AND ROTATION:



Right to left ectopia

1. Simple ectopia:
   - A kidney that is outside the renal fossa.
   - Pelvic (commonest), lumbar, sacral.
2. Thoracic kidney. kidney in chest
3. Horseshoe kidney
   - 2 kidneys fused and connected together
   - 90% by the lower lobes
   - 10% upper lobes connected
   - the connection is either fibrous band or sometimes it's parenchymal tissue
4. Unilateral renal agenesis.
5. Bilateral renal agenesis.
6. Crossed renal ectopia with no fusion.
7. Crossed renal ectopia with fusion.
8. Malrotated kidney.

- Normal position of the kidney: retroperitoneal in flank area.
  - Anywhere except this place is called ectopia
  - Cross ectopia: kidney goes to other side



Horseshoe kidney

3.2 CYSTIC ABNORMALITIES:

1. Renal dysplasia
   - Congenital unilateral multicystic kidney.
   - Segmental and focal renal dysplasia.
   - Renal dysplasia associated with congenital lower tract obstruction.
2. Congenital polycystic kidney disease:
   - Infantile type
   - Adult type
3. Simple cyst
4. Calyceal cyst
5. Peripelvic cyst
6. Perinephric cyst

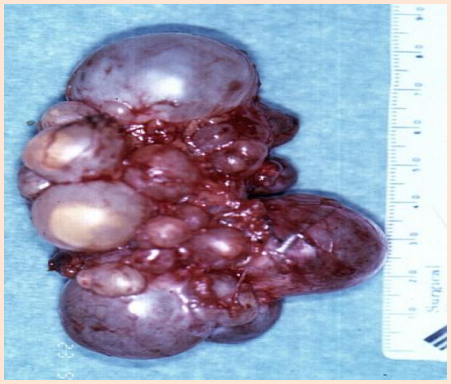


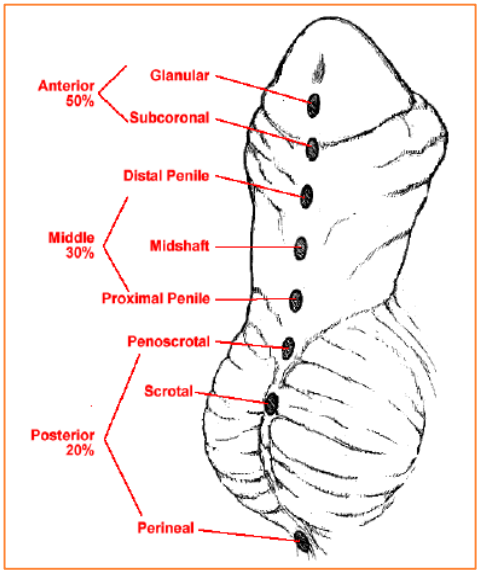
Figure: Multicystic dysplastic kidney

- One of the DDx of ANH
- Kidney is non functional
- The whole kidney replaced by cysts so no nephrogenic tissue
- Multicystic: means multiple cysts
- Dysplastic: no renal tissue.
- Polycystic: kidney is large usually bilateral

4 PRUNE BELLY SYNDROME

- Also called Eagle-Barrett Syndrome
- Consists of a triad of:
  1. **Absent abdominal wall muscles**
     - External oblique, Internal oblique, Transverse abdominal muscles
     - You can feel all the organs and you can even see the bowel movement because the muscle layer is either absent or thin (hypopalstic).
  2. **Bilateral undescended testis**
  3. obstructive **uropathy**
     - bilateral hydrouretronephrosis and large bladder

5 HYPOSPADIAS

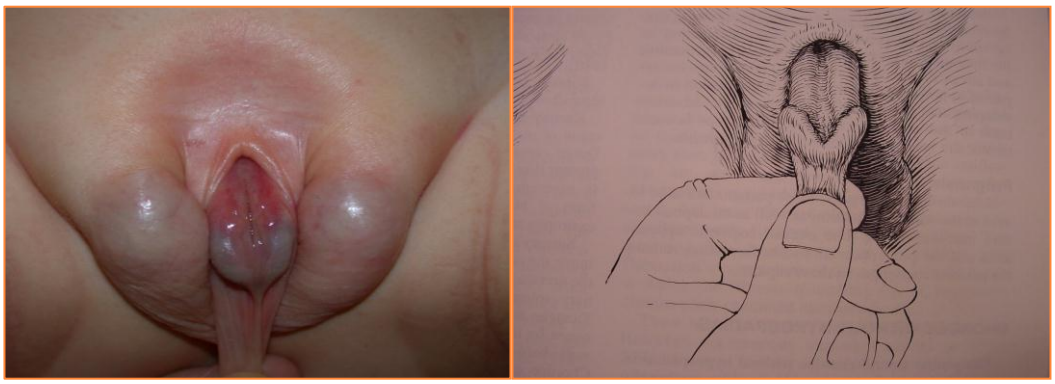


- Abnormally located meatus which contains the urethral opening
- The meatus usually opens in the tip of the glans penis and if it opens anywhere else it is considered an ectopic opening
  - In hypospadias the opening is towards the scrotum or ventral side
  - In Epispadias the opening is towards the abdomen or dorsal side
- Common (2%)
- Abnormal position of the EUM(external urethral meatus):
  - Distal hypospadias: (from mid shaft to Glans)
  - Proximal hypospadias (from proximal penile “proximal shaft” to the perineal)
- Treatment:
  - NO Circumcision ⓘ
  - Absolute contraindication because dorsal urethral skin we will be needed in repairing later on especially in proximal hypospadias

- Age to repair:6 to 9 months
- Requires one stage repair

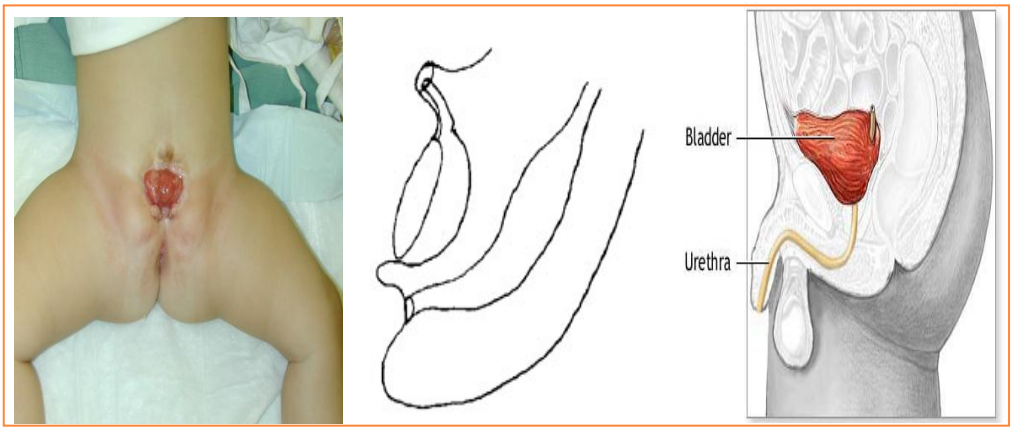
6 EPISPADIAS

- Very rare , Abnormal position of external urethral meatus in dorsal surface of the penis



7 BLADDER EXSTROPHY

- Bladder has 3 walls: Anterior, lateral and posterior.
- Anterior wall consist of abdominal muscles and skin
- In bladder exstrophy the anterior wall is absent (no anterior abdominal wall, no skin) so the lateral wall will be attached to skin to outside.
- the bladder is exposed to the outer environment
- We need to close bladder and to reconstruct abdomen
- Rare; 1:30000 live births with a 3:1 male : female ratio
- The results of improper development of anterior abdominal wall, pelvic girdle , and anterior wall of the bladder



ADULT UROLOGICAL DISORDERS

1 UROLOGICAL DISORDERS

1. Urinary Tract infections
2. Urolithiasis
3. Voiding dysfunction
4. Benign Prostatic Hyperplasia

1.1 URINARY TRACT INFECTIONS

1.1.1 INTRODUCTION

Lower Urinary Tract (less morbid)	**Upper Urinary Tract**
• Urethritis • Epididymitis/Orchitis • Prostatitis • Cystitis	• Acute Pyelonephritis • Chronic Pyelonephritis • Renal Abscess Pyelonephritis & Renal Abscess can lead to death. So, it's a serious condition.

Haematogenous spread: e.g. tooth abscess > pyelonephritis

In the past, they used to see more hematogenous spread like in TB> secondary TB in kidney.

-Adjacent invasion: e.g. Diverticulum ruptured in bladder >UTI.

- Routes of UTI: ⓘ
  - Ascending infection; 95%.
  - Haematogenous spread.
  - Adjacent invasion, imagine you have colon with diverticulum and diverticulum would rapture in bladder so u will have UTI
  - Lymphatics; rare.

1.2 URETHRITIS

Common in men; in young men usually the cause is STDs.

1.2.1 SYMPTOMS

- Urethral discharge
- LUTS in the form of dysuria
- Burning on urination
- Asymptomatic; 25% especially in women.

1.2.2 GONOCOCCAL VS. NON-GONOCOCCAL

- Diagnosis of the organism is established by :
  - Incubation periodⓘ: Gonococcal: 3-10 days vs. Non-gonococcal: 1-5 wks.
  - Urethral swab; send it to be cultured to identify the proper antibiotics which are affecting the organism.
  - Serum marker & antigen: Chlamydia-specific ribosomal RNA (usually done in chronic forms of disease)

	Gonorrhea	Chlamydia
Organism	**Neisseria gonorrhea**	**Chlamydia trachomatis**
Organism Type	Gram (-) diplococci	Intracellular facultative organism
Incubation Period	3-10 days	1-5 weeks
Urethral Discharge	Usually profuse, purulent	Usually Scant
Asymptomatic Carriers	40%-60%	40%-60%
Diagnostic Test	Ligand chain reaction, Gram stain Culture	Polymerase/ligand chain reaction, Culture, Immunoassay
Treatment	Ceftriaxone + Azithromycin or Doxycycline	Ceftriaxone or Azithromycin

The most common nonspecific urethritis is due to chlamydia.

1.3 EPIDIDYMITIS

- **Acute**: Pain, swelling, of the epididymis <6wk.
- **Chronic**: Longstanding pain in the epididymis and testicle, usually no swelling.

Young boy comes with bad testicular pain, must differentiate between 2 conditions:

1) Testicular torsion → urological emergency; patient should go to OR.

2) Epididymitis

- How can we differentiate b\w Epididymitis vs. Torsion?

	Epididymitis	Torsion
Family History	Older patient Gradual onset With urinary symptoms like burning sensation – hematuria e.g patient may say doctor I had blood in urine for 2 weeks now.	Usually young boys, just reached adolescence Acute pain – sudden in onset Usually without urinary symptoms
Physical Examination	Inflammatory sign (redness-warmth and swelling of the scrotum)	High raiding testis, testis is kidney-shaped , bean-shape, Horizontal lie Loss of cremasteric reflex
U/S	Because of infection > Hyperemia	No blood flow
Testicular Scan	Increased radiotracer uptake; hyperscan photogenic (black)	Photopenia (white area)
Urine for Culture	Younger: N. gonorrhoeae or C. trachomatis Older: E. coli (gram -ve rods)	

Young male has untreated urethritis, the consequences → blood flows up to urethra → goes to ejaculatory duct → goes to epididymis.

Epididymitis is caused by retrograde ascent of urinary pathogens from the urethra and bladder, via the ejaculatory ducts and vas deferens, leading to colonization and inflammation of the epididymis.

Cremasteric reflex is elicited by lightly stroking the superior and medial (inner) part of the thigh. The normal response is a contraction of the cremaster muscle that pulls up the scrotum and testis on the side stroked.

Examining a patient with torsion is very painful, but in epididymitis, raising the scrotum makes it better because it increases drainage → "Prehn's sign": pain relieved by elevation of the testicle.

Table 17–3. TREATMENT OF ACUTE EPIDIDYMO-ORCHITIS

Epididymo-Orchitis Secondary to Bacteriuria

1. Do urine culture and sensitivity studies
2. Promptly administer broad-spectrum antimicrobial agent (e.g., tobramycin, trimethoprim-sulfamethoxazole, quinolone antibiotic)
3. Prescribe bed rest and perform scrotal evaluation
4. Strongly consider hospitalization
5. Evaluate for underlying urinary tract disease

Epididymo-Orchitis Secondary to Sexually Transmitted Urethritis

1. Do Gram stain of urethral smear
2. Administer ceftriaxone, 250 mg IM once; then tetracycline, 500 mg PO qid for at least 10 days, or doxycycline, 100 mg PO bid for at least 10 days
3. Prescribe bed rest and perform scrotal evaluation
4. Examine and treat sexual partners

Adapted from Berger RE: Urethritis and epididymitis. Semin Urol 1983;1:143.

Figure 1: Guidelines for Treatment

1.4 PROSTATITIS

Prostate: produces about 80% of the semen

- Syndrome that presents with inflammation ± infection of the prostate gland including:
  - Dysuria, frequency
  - Dysfunctional voiding
  - Perineal pain
  - Painful ejaculation

The root of an ascending infection may go through ejaculatory duct to prostate and that's why some people get the infection.

-Difficult to treat because of the capsule and configuration of prostate. So you may give patients antibiotics for months.

Table 15–1. CLASSIFICATION SYSTEM FOR THE PROSTATITIS SYNDROMES

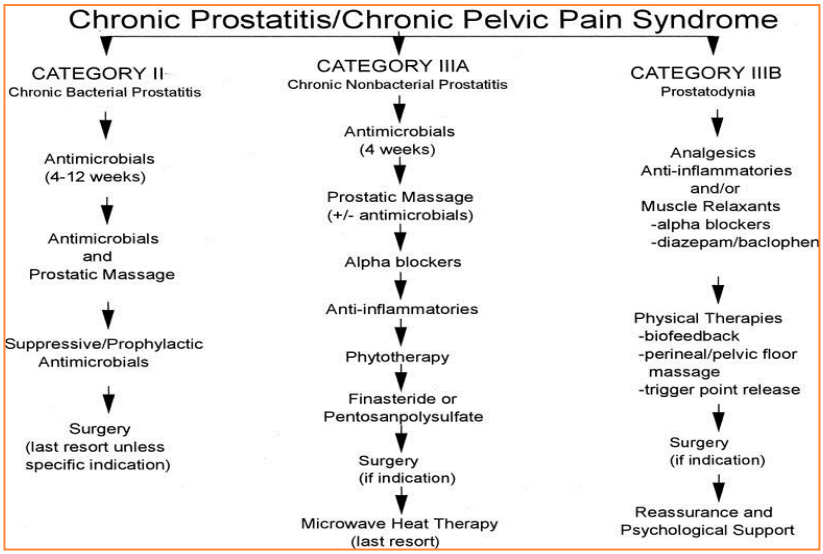
Traditional	National Institutes of Health	Description
Acute bacterial prostatitis	Category I	Acute infection of the prostate gland
Chronic bacterial prostatitis	Category II	Chronic infection of the prostate gland
N/A	Category III chronic pelvic pain syndrome (CPPS)	Chronic genitourinary pain in the absence of uropathogenic bacteria localized to the prostate gland with standard methodology
Nonbacterial prostatitis	Category IIIA (inflammatory CPPS)	Significant number of white blood cells in expressed prostatic secretions, postprostatic massage urine sediment (VB3), or semen
Prostatodynia	Category IIIB (noninflammatory CPPS)	Insignificant number of white blood cells in expressed prostatic secretions, postprostatic massage urine sediment (VB3), or semen
N/A	Category IV asymptomatic inflammatory prostatitis (AIP)	White blood cells (and/or bacteria) in expressed prostatic secretions, postprostatic massage urine sediment (VB3), semen, or histologic specimens of prostate gland

N/A, not applicable.

Figure 2: It's not required to know the categories of prostatitis. This table just shows that there's more than one type. Some are due to bacteria, and some are not due to bacteria. And we fail to isolate the bacteria when doing urine culture

1.4.1 ACUTE BACTERIAL PROSTATITIS

- Rare
- Acute pain
- Irritative and obstructive voiding symptoms
- Fever, chills, malaise, N/V
- Perineal and suprapubic pain
- Tender swollen hot prostate
- Rx: antibiotics and urinary drainage



Acute bacterial prostatitis: Patient should get admitted b/c it's a serious problem. He may get hypotension 90/40 (urosepsis – septic shock)

It's a rare clinical emergency condition that maybe life threating ⓘ BUT chronic prostatitis is more common.

1.5 CYSTITIS

- **Signs and symptoms**
  - Dysuria, frequency, urgency, voiding of small urine volumes
  - Suprapubic/lower abdominal pain
  - ± Hematuria
  - No fever even if it's severe
- **Diagnosis**
  - Dipstick: When nitrate is (+), it indicates an infection
  - Urinalysis
  - Urine culture; is the gold standard.ⓘ It takes 2 days. Start treatment before waiting for results b/c we know what are the commonest organisms.
- **Treatment**
  - Usually treatment of UTI in women is just for 3 days, to avoid any effect on normal bowel flora. In men, the treatment is usually for a week.

Cystitis is more common in women than men, why?

B\c women have shorter urethras (4.5 cm). Some of them are genetically predisposed to bacteria as the lining of the bladder is more susceptible to E.coli.

Table 14–10. TREATMENT REGIMENS FOR ACUTE CYSTITIS

Circumstances	Route	Drug	Dosage (mg)	Frequency per Dose	Duration (days)
Women					
Healthy	Oral	Ciprofloxacin	500	Every 12 hr	3
		Enoxacin	400	Every 12 hr	
		Levofloxacin	500	Every day	
		Lomefloxacin	400	Every day	
		TMP-SMX	160–800	Every 12 hr	
		TMP	100	Every 12 hr	
		Microcrystalline nitrofurantoin	100	Four times a day	
		Norfloxacin	400	Every 12 hr	
Symptoms for >7 days, recent urinary tract infection, age >65 yr, diabetes, diaphragm use		TMP–SMX or Fluoroquinolone	160–800 As above	Every 12 hr As above	7
Pregnancy	Oral	Amoxicillin	250	Every 8 hr	7
		Cephalexin	500	Four times a day	
		Microcrystalline nitrofurantoin	100	Four times a day	
		TMP-SMX	160–800	Every 12 hr	
Men					
Healthy and <50 years old	Oral	TMP-SMX or Fluoroquinolone	160–800 As above	Every 12 hr As above	7

Figure 3

1.6 PYELONEPHRITIS

- Definition: inflammation of the kidney and renal pelvis
- **Signs and symptoms:**
  - Chills
  - Fever
  - Costovertebral angle tenderness (flank pain)ⓘ
  - GI: Abdominal pain, N/V, and diarrhea
  - Gram -ve sepsis - mild flank pain
  - Dysuria, frequency
- **Investigations:**
  - Urine C&S (culture & sensitivity):+VE (80%)
  - Enterobacteriaceae (E. coli), Enterococcus
  - Urinalysis:↑ WBCs, RBCs,Bacteria
  - Blood test for renal function: (±) ↑serum Creatinine
  - CBC: Leukocytosisⓘ.
  - Urine dipstick, microscopy: To get rapid results
  - Imaging: To rule out any possible obstruction
    - IVP (Intravenous Pyelogram)
    - U/S
    - CT

When will the patient get fever? If the bacteria has ascended and reached the kidney (pyelonephritis)

-Some women will ignore their feeling of dysuria and they come to the ER with urosepsis.

ⓘ In culture, the most common organism we see in pyelonephritis is gram (-) rods E.Coli followed by enterococcus species.

Pyelonephritis complicated by obstruction:

- Renal stones complicated by ovary cancer that is blocking the kidney; in this case, we have to drain kidney. We don't only give antibiotics b/c there is a collection of pus by putting the tube in the kidney **"Nephrostomy Tube"**, under local anesthesia> used in obstructive infective kidney especially if patient is very sick.
- **In U/S**, we will see hydronephrosis; dilated kidney.
- Another option: If patient is better than the first example, we can do **"Double J"**, which is a tube placed inside the ureter during surgery to ensure drainage of urine from the kidney into the bladder. Stent is temporary treatment to bypass the blockage > b/c if we manipulate the stones, the patient may have bacteremia and die.

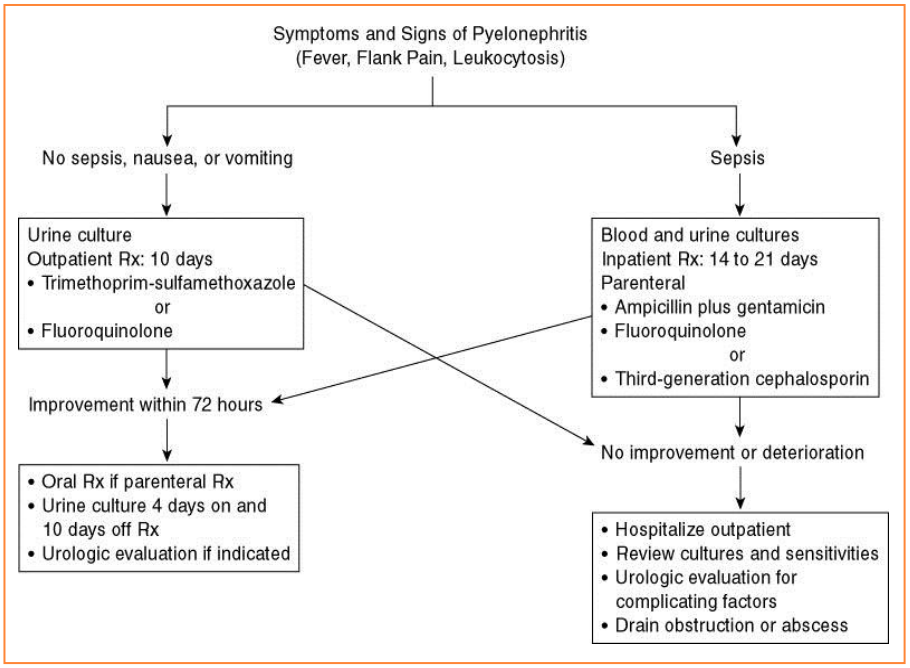


Figure 4: Just a guideline.

2 UROLITHIASIS

2.1 INTRODUCTION

- The condition associated with urinary calculi.
  - Common disease in Saudi Arabia
  - Egyptian mummies 4800 BC
  - Prevalence of 2% to 3%
  - Lifetime risk: Male : 20%, Female: 5-10%
  - Recurrence rate 50% at 10 years
- **Risk factors:**
  - Intrinsic Factors
    - Genetics
    - Age (2Os-4Os); young people.
    - Sex M>F
  - Extrinsic Factors
    - Geography (mountainous, desert, tropics)
    - Climate (July - October)
    - Water Intake
    - Diet (purines, oxalates, Na)
    - Occupation (sedentary occupations)
- **How do stones form?**
  - Supersaturated (patient doesn't drink water) → solute will concentrate→ Crystal Growth.
  - Aggregation of crystals →stone.
- **Most people have crystals in their urine, so why doesn't everyone get stones?**
  - Anatomic abnormalities.
  - Modifiers of crystal formation: Inhibitors/promoters

Cystinuria is an autosomal recessive disease: So it affects children (it's hard to tell if a child is drinking water). And, in general, if not treated, it can lead to death b\c of the complications like renal failure. When you do transplantation for them: new kidneys >the disease is gone

Anatomic abnormalities:

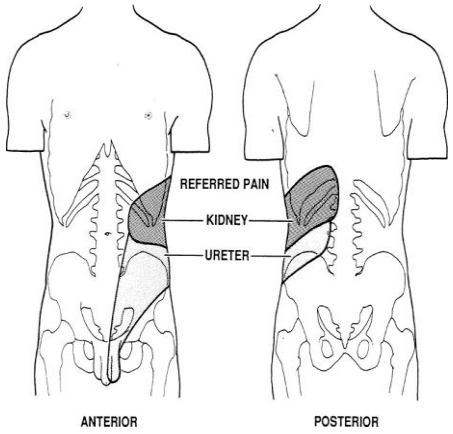
Presence of certain abnormalities of the urinary tract like hydronephrosis or obstruction in the urinary tract leads to stasis (stoppage) of the urine and then the super-saturation of minerals that eventually leads to formation of stones.

  - Citrate, Mg, urinary proteins (nephrocalcin); are inhibitors for stone formation.
  - Oxalate; is a promoter for stone formation such as: coffee, chocolate and soda drinks, except some of them which also contain citrate that will inhibit stone formation
- **Common stone types** ⓘ
  a) **Calcium** stones 75% (Ca Ox) calcium oxalate
  b) **Uric acid** stones; uric acid is found in animal protein & it's the commonest cause of radiolucent kidney stones.
  c) **Cystine** stones: Cystine is an amino acid. Remember them by COLA: cystine, oxaline, lysine and arginine; the proximal tubules are unable to reabsorb these amino acids. All of them are water soluble except Cystine, that's why it forms stones ⓘ.
  d) **Struvite** stones.

The renal angle is very tender in pyelonephritis, less tender in renal stones and not tender in appendicitis.

2.2 SIGNS AND SYMPTOMS

- Renal or ureteric colic
- Frequency, dysuria
- Hematuria
- GI symptoms: N/V, ileus, or diarrhea
- **DDx** :
  - Gastroenteritis
  - Acute appendicitis
  - Colitis
  - Salpingitis
- Restless ⓘ
  - ↑HR, ↑BP
  - Fever (If UTI)
  - Tender costovertebral angle



Taking History of renal colic:

You have to memorize the signs and symptoms.

Renal colic comes with flank pain. So you should ask about PAIN which has 8-10 questions that you should cover.

And when you take Hx of renal colic, you should form some differentials for flank pain such as:

-If pain is worse with bowing and improves by lying down – MSK pain

-If the pain radiates to right or left lower quadrant – Renal stone

-Radiates to labia in women and to scrotum in men – Renal stone

-Pain when coughing – Cholecystitis

-Pain with movement and goes to leg – Prolapsed disk.

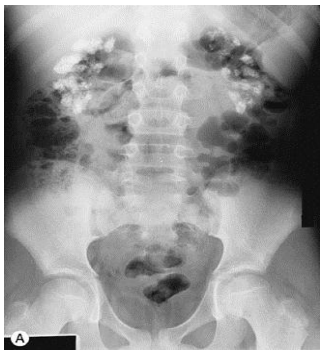
-If the pain comes after eating – Cholecystitis (and may also vomit)

-The pain is in the pre-umbilicus then goes to the right lower quadrant - Appendicitis

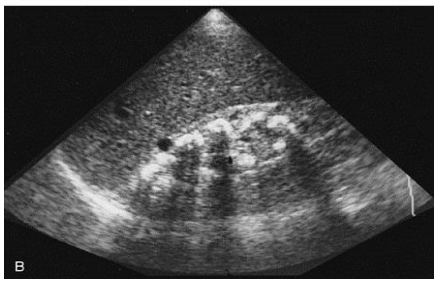
-Young married female with Hx of no period for 2 months – Ectopic pregnancy

2.3 INVESTIGATIONS

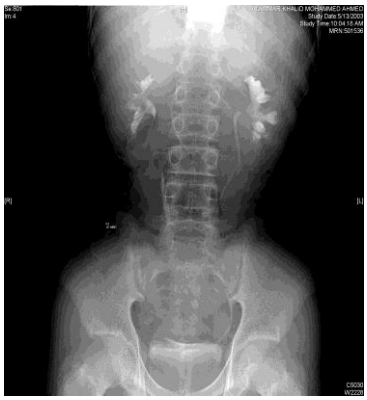
- Urinalysis :
  - RBCs
  - WBCs
  - Bacteria
  - Crystals
- Imaging (table 1)
  - Plain Abdominal Films (KUB); shows only radiopaque stones.
  - Intravenous Pyelogram (IVP); shows radiolucent (uric acid stone) & radiopaque stones (calcium stones).
  - Ultrasonography (U/S); shows hyperechoic stones + acoustic shadow.
  - Computed Tomography (CT): The gold standard; most sensitive and specific & shows the radiolucent stones. So it's the first step.

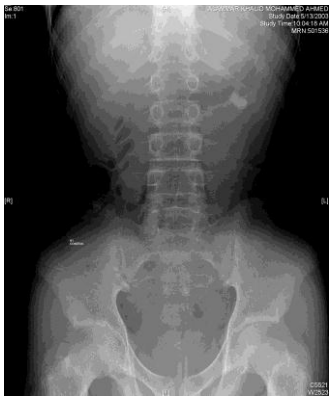


KUB

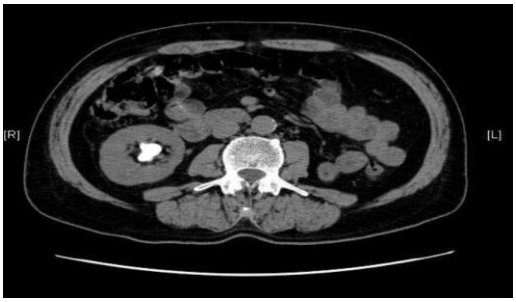


U/S

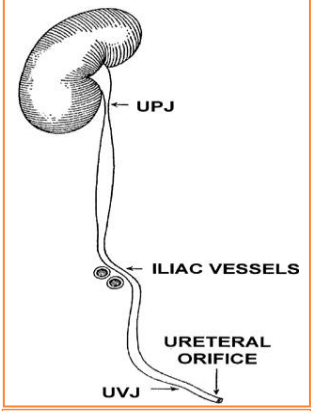




IVP: In case of high grade obstruction, you may stay 2 days without seeing any findings.



CT



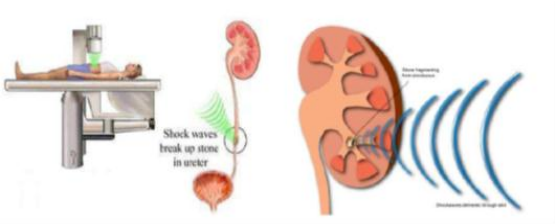
The 3 normal narrowings in the ureters

- Stenosis of Ureteropelvic Junction leads to stasis and hydronephrosis.
- UPJ: Ureteropelvic Junction
- UVJ: Ureterovesical Junction.

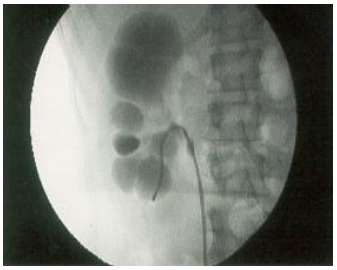
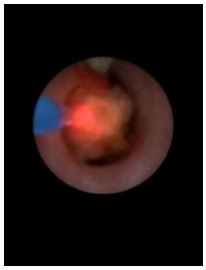
2.4 TREATMENT

1. Conservative:
    a. Hydration
    b. Analgesia
    c. Antiemetics
    d. Stones (<5mm) >90% undergo spontaneous passage.
2. **Indications for admission:**
    a. Renal Impairment
    b. Refractory Pain
    c. Pyelonephritis; patient has 3 mm stones with fever and chills> pyelonephritis.
    d. Intractable N/V; can't take oral analgesia.

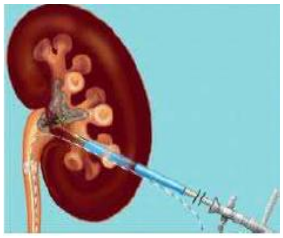
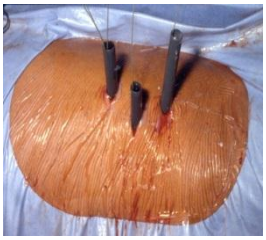
3. **Shock Wave lithotripsy (SWL):** Good for kidney stones and small stones; potential injury to ovary.



4. **Ureteroscopy:** Breaks up large stones by laser.

5. **Percutaneous Nephrolithotripsy (PNL)**: For huge stones

6. **Open surgery:** Not used anymore.

3 VOIDING DYSFUNCTION

Failure to store	Failure to Empty
– Bladder Problems – Overactivity: common in women or b/c of spinal cord injury, stroke > loss of control by causing damage to micturition inhibitory center. – Hypersensitivity	– Bladder Problems – Neurologic – Myogenic – Idiopathic
– Outlet Problem – Stress Incontinence: With pregnancies and deliveries, the pelvic wall muscles is gone (?), the support is gone so with a little increase in abdominal pressure> leakage – Sphincter Deficiency	– Outlet Problem – BPH: Benign Prostatic Hyperplasia – Urethral Stricture – Sphincter Dyssynergia
– Combination	– Combination

4 BENIGN PROSTATIC HYPERPLASIA

4.1 CLINICAL FEATURES:

- LUTS (Irritative/Obstructive)
- Poor bladder emptying
- Urinary retention
- Urinary tract infection

- Hematuria
- Renal insufficiency

4.2 PHYSICAL EXAMINATION:

1. DRE (Digital rectal Examination) ⓘIf it's hard to palpable the nodules, it means Cancer.
2. Focused neurologic exam
   - Prostate Ca
   - Rectal Ca
   - Anal tone
   - Neurologic problems
3. Abdomen: Distended bladder ⓘ

4.3 INVESTIGATIONS

1. Urinalysis, Culture
   a. UTI
   b. Hematuria
2. Serum Creatinine
3. Serum Prostate-Specific Antigen; it is elevated in prostatic cancer.
4. Flow rate
5. U/S (kidney, bladder and prostate).

4.4 MANAGEMENT

1. Medical therapy
   a. α-Adrenergic Blockers; selective α1 blocker that opens the prostate.
      i. Tamsulosin
      ii. Alfuzosin
      iii. Terazosin
   b. Androgen Suppression; 5α reductase inhibitor > shrinks prostate 60% in 6 months
      i. Finasteride
2. Surgical Rx
   a. Endoscopic (e.g. TURP, laser ablation, prostatic stent); Cut adenoma that blocks the passage.

   b. Open prostatectomy.

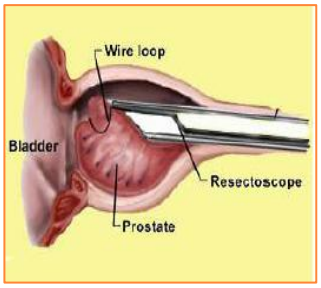


Figure 5: Endoscopy

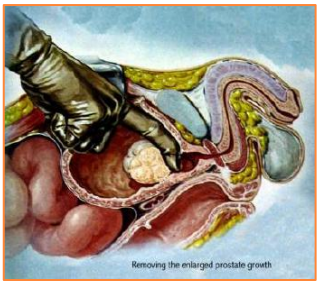


Figure 6: Prostatectomy

5 MCQS

1. A 13-year old boy presented to the Emergency Room with painful right scrotal swelling. It was gradual in onset over the last 5 days. He gave history of dysuria and suprapubic pain for the last 2 weeks. The most common cause of his symptoms is:
   a. Epididymitis
   b. Hydrocele
   c. Testicular Torsion
   d. Testicular Trauma

2. A 22-year old single male presented with dysuria and urethral discharge, 5 days after unprotected intercourse. On examination, there is erythema over his urethral meatus with yellowish discharge. The most likely causative organism for his presentation is:
   a. Chlamydia trachomatis
   b. Escherichia coli
   c. Herpes simplex virus
   d. Neisseria gonorrhea
3. A 65-year old diabetic woman presented with right flank pain and fever for 2 days. She has been complaining of dysuria and suprapubic pain for more than one week. She is nauseated and had 3 episodes of vomiting. The most likely diagnosis is:
   a. Acute cholecystitis
   b. Acute pyelonephritis
   c. Pancreatitis
   d. Renal colic
4. Irritative urinary tract symptoms include all of the following except:
   a. Dysuria
   b. Hesitancy
   c. Frequency
   d. Urgency
5. Main causative organism for UTI is:
   a. E. Coli
   b. Chlamydia
   c. Proteus
   d. Gonorrhea
6. The main symptoms of pyelonephritis are:
   a. Fever
   b. Flank pain
   c. Chills
   d. All of the above
7. The most common type of urinary tract stones is:
   a. Calcium stones
   b. Uric acid stones
   c. Cystine stones
   d. Struvite stones
8. All of the following are true about epididymitis except:
   a. It takes days or weeks to develop
   b. It can be diagnosed by US
   c. Dysuria and pain are the main complaints
   d. Testicular scan reveals ischemia of the testicles

🗝 Answers: 1:a, 2:d, 3:b, 4:b, 5:a, 6:d, 7:a, 8:d

EMERGENCIES IN UROLOGY

1 INTRODUCTION AND CLASSIFICATION

- Require rapid diagnosis and immediate treatment.
- Compared to other surgical fields there are relatively few urological emergencies
- Classification/topics
  - Non-traumatic:
    - Hematuria
    - Renal colic
    - Urinary retention
    - Acute scrotum
    - Priapism
  - Traumatic:
    - Renal trauma
    - Uretral injury
    - Bladder trauma
    - Urethral injury
    - External genital injury

2 NON-TRAUMATIC UROLOGICAL EMERGENCY

2.1 HEMATURIA

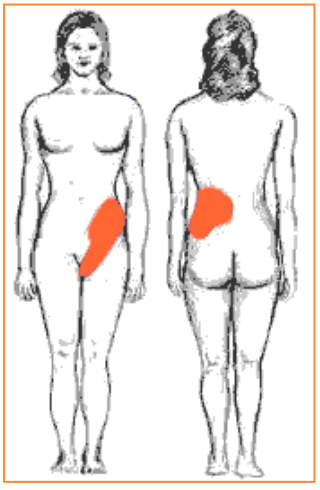
- Definition: blood in the urine
- Types:
  - Gross
    - Clinically visible/ emergency or urgency
    - Up to 40% is malignancy
    - 1 ml of blood in 1 liter of urine is usually visible
  - Microscopic
    - Not visible
    - Here the patient is told that he has Hematuria
    - Not an emergency or urgency
    - 3 or more RBC/high power field
- Causes:
  - They vary according to: patient age, presence of symptoms, presence of risk factors for malignancies and the type (gross/microscopic)
  - They could be:
    1. Pre renal: SLE, Sickle cell disease, hemophilia, anticoagulants.
    2. Renal: Tumor (benign or malignant), Renal stasis, Stone, TB, Glumerulonephritis.
    3. Post Renal: Tumor (bladder or ureter, Bilharzias, Prostate pathology, urethral stricture, urethral polyp/tumor.
- History: Very important to diagnose
  - Chief complaint:
    - Age: for example transitional cell carcinoma is not common in children

        - Residency: Bilharzias is common in Jizan
        - Duration
        - Occupation: Factories
        - Painless:
    - Usually transitional cell carcinoma (TCC) originating from the urothelium of the bladder.
    - Risk factors for TCC: Smoker, above 40, LUTS irritation, radiation to the pelvis, bilharzias
        - Painful: Stones, UTI, Trauma, Renal vein thrombosis
        - Timing: helps in recognizing the site of the bleeding:
    - Initial: urethra
    - Terminal: bladder neck or triagone
    - Total: rest of the bladder and upper tract
        - Amount of bleeding, Clots and shape, trauma and history of bleeding from other sites
  - Associated urinary and other systemic symptoms
  - History of bleeding disorders, infections, stones, TB, bilharzias.
  - Family history of malignancy or hematological disease
  - Drugs and colored foods and drinks
  - SMOKING:ⓘ asking about smoking is very crucial because it is known to be a risk factor for bladder transitional cell carcinoma and renal cancer
- Management:
  - Work up
    - Full work up is mandatory
    - History, examination (not much signs)
    - Investigations: Single most important imaging method is CTU (CT Urography) ⓘ
    - 3 way urethral catheter and wash out heavy bleeding
    - Treat the underlying cause

2.2 RENAL COLIC

- The commonest urological emergency (in Saudi Arabia cases are seen daily)
- One of the commonest differentials associated with acute abdomen
- Characteristically: Sudden onset of severe pain in the flank

2.2.1 HISTORY OF PAIN: ⚐ⓘ



- Sudden onset, intermittent, relieved by analgesia & nothing aggravates it
- Colicky in nature
- Radiation
  - The kidney and upper ureter are innervated from dermatomes T7-T9.
    - In men the pain will radiate to the testicle because it embryological originates from the same site and then the testicle descends
  - Mid ureter: dermatome T10 > radiate to the iliac fossa
    - If this happens in right side can be confused with appendicitis
  - Distal ureter: dermatome T12> triagone of the bladder, posterior urethra, scrotal skin, labia majora and lower abdomen
- Location may change from the flank to the groin SO the location of the pain is not a food indicator of the location of the stone
- Patient is not comfortable and might be rolling around

- Associated with nausea/vomiting
- Ureter stones:
  - Sudden severe pain
  - Urinary symptoms and suprapubic pain
- Remember that the pain of a renal colic is very painful, one of the worst a human can experience

2.2.2 DIFFERENTIAL DIAGNOSIS

Figure 1

- Radiculitis (figure 1)
  - Musculoskeletal pain that happens due to irritation of the nerve root in the intervertebral foramen
  - A common form of it is sciatica
  - Irritation of the intercostals nerves (T7,8,9) can give a similar picture
  - Usually aggravated by movement unlike stones that are relieved by movement
  - Radiates to lower limb if involving sciatic nerve roots
  - History: back pain and predisposed mobility (carrying something heavy)
- Chest: Pneumonia and Myocardial infarction
- Abdomen: ruptured abdominal aortic aneurism, bowel obstruction, appendicitis, IBD, burst peptic ulcer and diverticulitis.
- Pelvis: Ectopic pregnancy and ovarian pathology (twisted cyst)
- Testicular torsion

2.2.3 WORK UP:

- History
- Examination:
  - Patient wants to move around to find a comfortable position. This helps in differentiating from appendicitis.
  - Fever: indicates infection and needs extra hydration
- Investigations:
  - Pregnancy test
  - Mid stream urine: for Hematuria and urine analysis
  - Urea and electrolytes: asses renal function
- Radiological investigation
  - CT without contrast: (Figure 2)
    - **Imaging modality of choice** ⓘ
    - Greater specificity (95%) and sensitivity (97%) for diagnosing ureteric stones
    - Can identify other, non-stone causes of flank pain.
    - No need for contrast administration.
    - Faster, taking just a few minutes
    - the cost of CTU is equivalent to that of IVU
  - Intravenous urogram (IVU): X-Ray and contrast before and after injection
  - KUB: Plain X-ray of the kidney, ureter and bladder
  - Renal ultrasound (RUS): not good for investigation stones
  - MRI:
    - Very accurate way of determining whether or not a stone is present in the ureters
    - Time consuming and expensive (not available in all hospital)

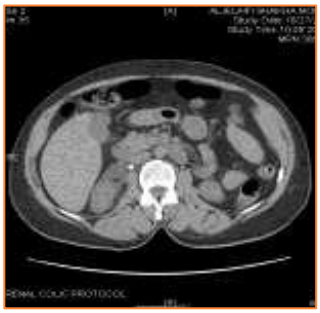


Figure 2

        - Used for pregnant ladies (no radiation)
    - In summary:ⓘ
        - CT without contrast is the module of choice in investigating a renal colic in an emergency setting
        - MRI is used for pregnant ladies only because it is time consuming

2.2.4 MANAGEMENT:

- Medical:
    - Pain relief
        - NSAID (IM, IV, Oral or suppository)
        - Opiates analgesics (Morphine)
    - Hyper hydration (IV-fluids and drinking water)
    - Watchful waiting: **95% of stones measuring 5 mm or less will pass on their own** ⓘ
- Surgical
    - Indications for surgery: ⓘ
        1. To relieve obstruction and/or remove the stone
        2. Pain that fails to respond to analgesia
        3. Associated fever: kidney must be drained to reduce risk of peylonephritis
        4. Impairment in renal function because of the stone (causing uremia)
        5. Obstruction is unrelieved for >4 weeks (Because after 4 weeks the obstruction will cause necrosis)
        6. Personal or occupational reasons: doctors or pilots
    - Types of surgical intervention:
        - Temporary relieve of obstruction:
        - JJ stent from renal pelvis to bladder
        - Percoetaneous nephrostomy tube
        - Definitive treatment:
        - Extracorporeal ShockWaves Lithotripsy (ESWL)
        - Percoetaneous Nephrolithotomy (PCNL)
        - Uretroscopy (commonly known as laser)
        - Laparoscopic extraction (rare)
        - Open surgery (rare)

2.3 URINARY RETENTION

2.3.1 ACUTE URINARY RETENTION

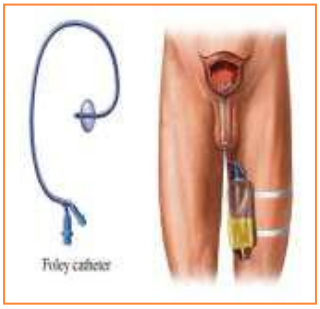


Figure 3

- **Painful** inability to void with relief of pain following drainage of the bladder by catheterization
- More in Men than in Women
- Causes:
    - Men:
        - Benign prostatic enlargement due to hyperplasia is the most common cause (usually in >40 years of age) ⓘ
        - Carcinoma of the prostate
        - Abscess in the prostate
        - Urethral stricture
    - Women:

    - Pelvic organ prolapsed (cystocele, rectocele, uterine prolapse)
    - Urethral stricture or diverticulum
    - Post surgery for stress incontinence
    - Pelvis masses (e.g. Ovarian mass)
- Management:
  - Initially: to relieve the pain!
    - Urethral catheterization:
      - Using a 3 way or Foley's catheter (figure 3)
      - Make sure to give adequate analgesia to prevent spasm
    - Suprapubic catheter: (figure 4)
      - Passed directly to the bladder through the skin
      - Used when urethra cannot be accessed (stricture)
  - Definitive treatment: treat the underlying cause

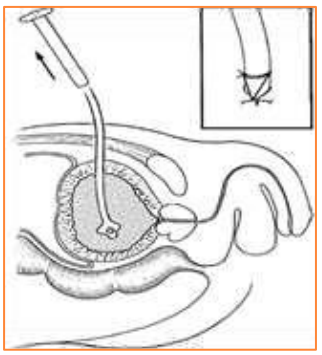


Figure 4

2.3.2 CHRONIC URINARY RETENTION:

- Obstruction here develops slowly and the bladder is distended (stretched) very gradually over weeks/months
- Pain is not a feature ⓘ
- Can be associated with:
  - Reduced renal function or renal failure
  - Upper tract dilation and hydronephrosis
- Presentation:
  - Urinary dribbling
  - Overflow incontinence (vesicle pressure exceed the urethral pressure
  - Palpable bladder with no pain
- Management:
  - In general it is more difficult that acute retention because the cause is usually neurological
  - Renal support and treat electrolyte imbalance
  - Bladder drainage in a slow rate to avoid sudden decompression (can cause Hematuria)
  - Treatment of the underlying cause

Epididymitis is inflammation of the epididymis

Orchitis is inflammation of the Testicle

Epididymo-orchitis is inflammation of both

2.4 ACUTE SCROTUM: ⓘⓘ

- Also known as scrotal pain or testicular pain
- Emergency situation requiring prompt evaluation, differential diagnosis, and potentially immediate surgical exploration
- Differential diagnosis (Box 1)
  - Epididymitis
    - Most common cause
    - Can also be Epididymo-orchitis
  - Torsion of the spermatic cord: the most serious complication!

Box 1
- Torsion of the spermatic cord
- Torsion of the appendix testis
- Epididymitis
- Epididymo-orchitis
- Inguinal hernia
- Communicating hydrocele
- Hydrocele
- Hydrocele of the cord
- Trauma/insect bite
- Dermatologic lesion
- Inflammatory vasculitis (Henoch-Schonlein purpura)
- Idiopathic scrotal edema
- Tumor
- Spermatocele
- Non-urogenital pathology e.g. adductor tendinitis

2.4.1 TORSION OF THE CORD ⓘ

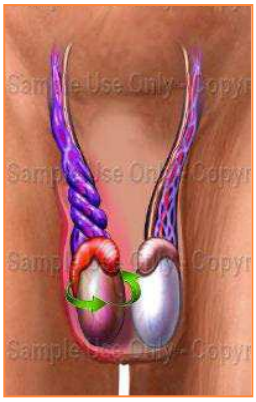
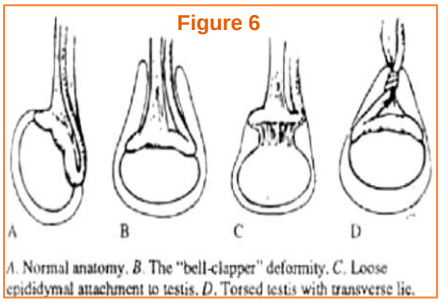


Figure 5

- **General consideration**:
  - Epidemiology
    - Common among teenagers 12-18
    - Possible in children and neonates
    - Unlikely to occur after the age 25 years
  - **True surgical emergency of the highest order**
  - The testicular parenchyma will develop irreversible ischemic injury as soon as 4 hours
    - The twisting will lead to occlusion of venous return→ swelling and blockage of arterial supply
    - The longer the time of torsion → more ischemia
  - As duration of torsion increases the possibility of testicular salvage decreases
- **Anatomical variations**: (figure 7)
  A. Normal.
  B. Bell clapper deformity. Tunica vaginalis surrounds the whole testicle so it is very loose
  C. Loose epididymal attachment to the testis
  D. Torsed testis with transverse or oblique lie

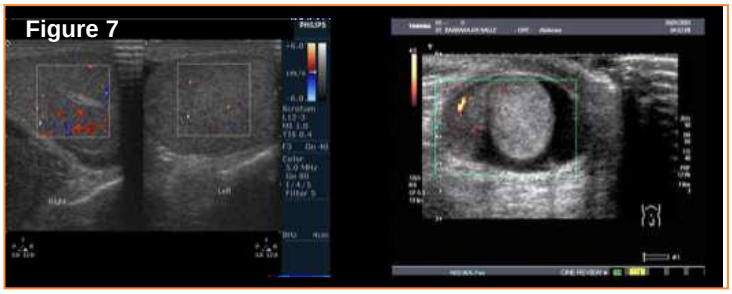
  Figure 6

  A. Normal anatomy. B. The "bell-clapper" deformity. C. Loose epididymal attachment to testis. D. Torsed testis with transverse lie.

  - Types: (not important)
    - Extra-vaginal
    - Intra-vaginal
- **Presentation:**ⓘ
  - Acute onset of scrotal pain
    - Sharp and severe
    - May be intermitting due to torsion then detorsion
  - Majority have a history of prior episodes of severe, self limited scrotal pain and swelling
  - Nausea and vomiting due to the pain
  - Referred to the Ipsilateral lower quadrant of the abdomen
  - Children may present with abdominal pain.
    - So any child that complains of severe abdominal pain may need to have a genital examination
    - Doctor mentioned a scenario: a mother brought her child to the clinic and said "my son went to school and ate bad food and now he has abdominal pain and nausea/vomiting" after further inspection the child had Torsion of the cord.
  - Dysuria and other bladder symptoms are usually absent (unlike Epididymitis)
- **Physical exam**:ⓘ
  - Affected testis is high and lying transverse
  - Acute swelling and scrotal edema or secondary hydrocele
  - Absent cermasteric reflex(because the nerve is within the spermatic cord)
  - Testis is tender and larger: the patient will not let you touch it
  - Elevation of the scrotum causes MORE pain (unlike Epididymitis)
- **Investigations**:

- Usually we do not need investigations because this is an emergency and a high degree of suspicion is enough to send the patient to the OR immediately ⓘ
- Adjunctive tests aid in the deferential diagnosis
- Confirm the ABSENCE of torsion
- Tests used:
  - **Sound Doppler** examination of the cord: high false positive and false negatives
  - **Color Doppler ultrasound**: (figure 8)
    - **<u>Investigation of choice</u>**
    - Done in the OR a lot of the time.
    - Assessment of anatomy and determining the presence or absence of blood flow.- to see the arterial blood supply of the testis
    - In the picture : in the left there is absence of blood supply , secondary hydrocele without arterial flow
    - Sensitivity: 88.9% specificity of 98.8%
    - Operator dependent.



  - **Radionuclide imaging**: (figure 9)
    - Assesses testicular blood flow
    - Shows a photopenic area in cases of torsion
    - False impression from hyperemia of scrotal wall
    - Sensitivity of 90% and specificity of 89%
    - Not helpful to determine a hydrocele or hematoma (does not assess anatomy)
  - **Surgical exploration**:
    - Diagnostic and therapeutic ⓘ
    - A scrotal incision is done and the affected site is examined first
      - a needle prick is done and if there is no blood coming out or black tissue it means it is dead
    - The cord should be detorsed.
    - Testes with marginal viability should be placed in warm and re-examined after several minutes.
    - A necrotic testis should be removed
    - If the testis is to be preserved, it should be fixed
    - The contra-lateral testis must be fixed to prevent subsequent torsion
- Remember when there is a high possibility of testicular torsion from history and examination → take the patient to the OR and do not wait ⓘ

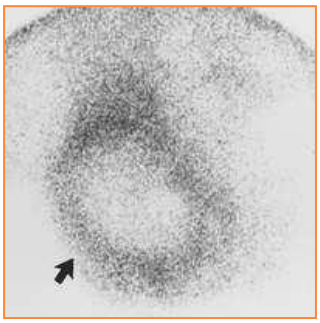


Figure 8

2.4.2 EPIDIDYMO-ORCHITIS

- **Presentation**:
  - Common in Saudi Arabia (can be a manifestation of Brucella)
  - Indolent process causing little or no pain
  - Usually gradual and not sudden and gets severe towards the end
  - Scrotal swelling, erythema and pain.
  - Dysuria and fever are common
  - Patients with history of STD like gonorrhea or UTI
- **Physical examination**
  - Localized epididymal tenderness
  - Swollen and tender epididymis. Or massively swollen hemi-scrotum
  - Cermasteric reflex is **present.** ⓘ
  - Patient feels less pain when the scrotum is raised
- Urine analysis might show bacteruria and/or positive culture and WBC
- **Management**:
  - Bed rest for 1-3 days
  - Scrotal elevation with athletic supporter
  - Parental or Oral antibiotics should be instituted when UTI is documented or suspected
  - AVOID urethral instrumentation to reduce risk of more infection. ⓘ

2.5 PRIAPISM

- **Defined** as a persistent erection of the penis for more than 4 hours that is not related or accompanied by sexual desire
- **Types of Priapism:**
  - Ischemic:
    - Painful type
    - Also called veno-occlusive or low flow
    - Most common type
    - Pathophysiology: thrombosis of the venous system causing congestion and engorgement which leads to the erection
    - Causes include:
      - Hematological disease: Sickle cell ⓘ
      - Malignancy that infiltrated the corpora cavernosa
      - Drugs like prostaglandin injection
  - Non-ischemic:
    - Painless type
    - Also called Arterial or high flow
    - Pathophysiology: perineal trauma will cause an atriovenous fistula which fills the corpora
- The persistence of Priapism will cause clotting which leads to healing by fibrosis in the corpora and this will damage it and the patient will lose the ability of erection
- **Causes**: ⓘ
  - Primary (idiopathic) in 30-50% of the cases
  - Secondary (as mentioned above): Drugs, trauma, pelvic malignancies, hematological disease, neurological.
- **Diagnosis**:

- Obvious from history!
  - Erection for more than 4 hours
  - Document if it is painful or not
  - Previous history of Priapism
  - Ask about predisposing factors and possible causes
- Examination:
  - Erect penis that can be tender (in low flow) or not
  - Characteristically the corpora cavernosa are rigid and the Glans is flaccid
  - Abdominal examination for evidence of malignancy
  - Digital rectal exam: to examine the prostate and check for anal tone (neurological assessment)

- **Investigations**:
  - CBC
  - Hemoglobin electrophoresis for SCD
  - Urinalysis for toxicology
  - Blood gases taken from either corpora

Variable	Low flow (ischemic/occlusive)	High-flow (non-ischemic/Fistula)
Blood color	Dark blood	Bright red blood (similar to arterial blood at room temperature)
pH	<7.25 (acidosis)	=7.4 (normal)
pO2	<30 mmHg (hypoxia)	>90 mmHg (normal)
pCo2	>60 mmHg (hypercapnia)	<40 mmHg (normal)

  - Color Doppler flow in cavernous arteries
    - Ischemic: in flow is low or nonexistent
    - Non-ischemic: inflow is normal to high
  - Penile pudendal arteriography in cases of trauma
- **Treatment**:
  - Depends on type of Priapism
  - Conservative treatment should be tried first
    - Ask the patient to climb the stairs to open venous channels
  - Medical treatment: bicarbonates, high o2 and cold enema
  - Surgical treatment: aspiration and saline wash of the corpora
  - Treat the underlying cause

3 TRAUMATIC UROLOGICAL EMERGENCIES

3.1 RENAL TRAUMA

- The kidneys are relatively protected from traumatic injuries so a considerable degree of force is usually required to injure a kidney.
- Mechanism and causes:
  - Blunt trauma:
    - Direct blow or acceleration/deceleration injuries
    - Road traffic accidents, falls from heights, falls on flanks
  - Penetrating trauma: knives, gunshots, iatrogenic (during operations)

3.1.1 RENAL IMAGING: ⓘ

- Modality of choice is contract enhanced CT. ⓘ

- **Indications for renal imaging:**
  1. Macroscopic Hematuria
  2. Penetrating chest, flank, and abdominal wounds
  3. Microscopic [>5 red blood cells (RBCs) per high powered field] or dipstick
  4. Hematuria in hypotensive patient (SBP <90mmHg)
  5. A history of a rapid acceleration or deceleration
  6. Any child with microscopic or dipstick Hematuria who has sustained trauma – even < 5 RBC.
- **Modalities available:**
  1. IVU:
     a. widely replaced by CT scan with contrast
     b. on table IVU: if patient is transferred immediately to the operating table without having had a CT scan and a retroperitoneal hematoma is found
     c. done to see if the other kidney is functioning and/or exists because the injured kidney might have to be removed
  2. CT scan
     a. Without contrast: does not allow accurate staging
     b. With contrast: imaging modality of choice + other abdominal injuries can be assessed ⓘ
  3. Renal ultrasound
     a. Advantages:
        i. can certainly establish the presence of two kidneys
        ii. the presence of a retroperitoneal hematoma
        iii. power Doppler can identify the presence of blood flow in the renal vessels
        iv. To follow up on a hematoma after CT is done
     b. Disadvantages:
        i. Cannot accurately identify parenchymal tears, collecting system injuries, or extravasations of urine until a later stage when a urine collection has had time to accumulate.

3.1.2 STAGING: ⓘ (FIGURE 10)

- Grade I: flank pain + Hematuria with or without pericapsular hematoma, but no evident kidney damage
- Grade II: injury to the cortex only of 1cm or less with hematoma
- Grade III: injury to the cortex and medulla without reaching the collecting system with hematoma (more than 1cm)
- Grade IV: injury reaching to the collecting system OR thrombosis to the renal vessels
  - On IVU there will be extravasations of contrast and decreased filling
- Grade V: shattered kidney completely

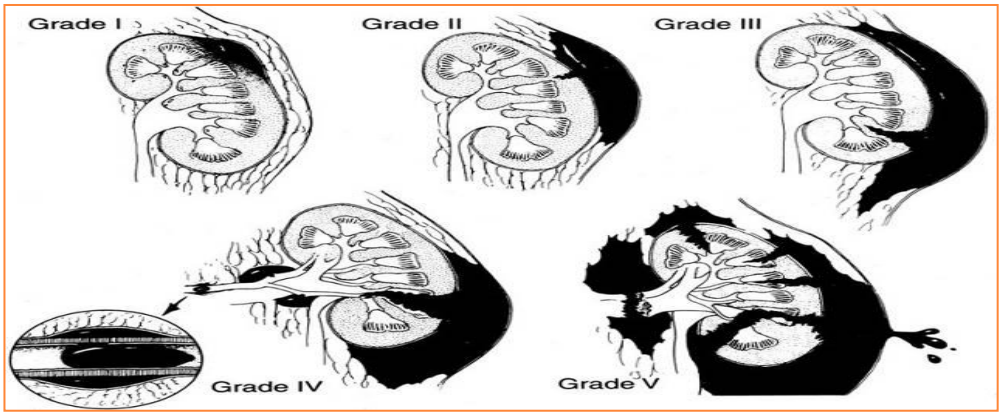


Figure 9

3.1.3 MANAGEMENT

A. Conservative
   - Over 95% of blunt injuries
   - 50% of renal stab injuries and 25% of gunshot wounds injury
   - Needs a specialized center
   - Includes:
     - Wide bore IV lines to transfuse fluids
     - IV antibiotics
     - Bed rest
     - Serial CBC and HCT
     - Follow up US and/or CT

B. Surgical exploration (indications for surgery):
   - Persistent bleeding: tachycardia and/or hypotension failing to respond to appropriate fluid and blood replacement
   - An expanding peri-renal hematoma after laparotomy
   - Pulsatile peri-renal hematoma after laparotomy

3.2 URETRAL INJURIES

- The ureters are protected from external trauma by surrounding bony structures, muscles and other organs therefore their injury is rare.
- **Mechanisms and causes:**
  1. External trauma
     - Rare because severe forced is required
     - Can be blunt or penetrating
     - Blunt external trauma severe enough to injure the ureters will usually is associated with multiple other injuries.
     - Penetrating knives or bullets to the abdomen may also damage the ureter
  2. Internal trauma
     - More common but it is still uncommon
     - Iatrogenic: causes by doctors during surgeries (hysterectomy, oopherectomy, sigmoidcolectomy, urertoscopy, cesarean section, laparoscopies and orthopedic operations
- **Diagnosis:**
  - Requires high index of suspicion

- Usually diagnosed **intra-operatively**
- Late diagnosis: (these are suggestive of ureter injuries):
  1. An ileus: presence of urine within the peritoneal cavity
  2. Prolonged postoperative fever or overt urinary sepsis
  3. Persistent drainage of fluid from abdominal or pelvic drains, from a wound or the vagina.
  4. Flank pain if the ureter has been ligated
  5. An abdominal mass representing a urinoma
  6. Vague abdominal pain

- **Treatment options:**
  - JJ Stenting
  - Primary closure of partial transaction of the ureter
  - Direct ureter to ureter anastomosis
  - Re-implantation of the ureter into the bladder using a psoas hitch or a Boari flap
  - Trans uretero-ureterostomy
  - Auto-transplantation of the kidney into the pelvis
  - Replacement of the ureter with ileum
  - Permanent cutaneous ureterostomy
  - Nephrectomy

3.3 BLADDER INJURIES

- Common in caesarean sections
- **Causes**:
  - Iatrogenic
    - Transurethral resection of bladder tumor (TURBT)
    - Cystoscopic bladder biopsy
    - Transurethral resection of prostate (TURP)
    - Cystolitholapaxy
    - Caesarean section, especially as an emergency
    - Total hip replacement (very rare)
  - Penetrating trauma to the lower abdomen or back
  - Blunt pelvic trauma—in association with pelvic fracture or ‘minor’ trauma in a drunkard patient
  - Rapid deceleration injury seat belt injury with full bladder in the absence of a pelvic fracture
  - Spontaneous rupture after bladder augmentation
- **Types of perforation:**
  - **Intra-peritoneal perforation**: the peritoneum overlying the bladder has been breached along with the wall of the bladder allowing urine to escape into the peritoneal cavity.
  - **Extra-peritoneal perforation**: the peritoneum is intact and urine escapes into the space around the bladder, but not into the peritoneal cavity.
- **Presentation:**
  - Recognized intra-operatively
    - The classic triad of symptoms and signs that are suggestive of a bladder rupture ⓘ :
      1. Suprapubic pain and tenderness
      2. Difficulty or inability in passing urine
      3. Hematuria

- **Management**:
  - Extra-peritoneal:
    - Bladder drainage +++++
    - Open repair +++
  - Intra peritoneal :
    - Open repair…why?
    - Unlikely to heal spontaneously.
    - Usually large
    - Leakage causes peritonitis
    - Other organs are usually injured

3.4 URETHRAL INJURIES

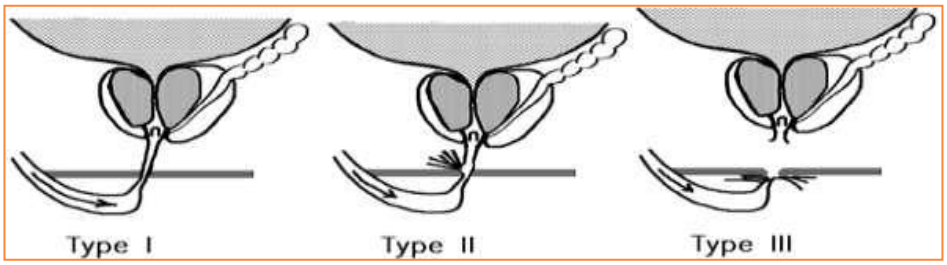
3.4.1 ANTERIOR URETHRAL INJURIES

- Rare
- **Mechanism**:
  - Majority are a result of straddle injuries in boys or men (jumping while your legs are open)
  - Direct injuries to the penis
  - Penile fractures
  - Inflating a catheter in the anterior urethra
  - Penetrating injuries by guns or knives
- **Symptoms and signs**
  - Blood at the end of the penis
  - Difficulty in passing urine
  - Frank Hematuria
  - Hematoma around the site of rupture
  - Swelling of the penis
- Diagnosis is by **Retrograde Urethrography:**
  - Less filling means greater damage
    - Contusion: no extravasations of fluid
    - Partial rupture: extravasations of contrast with contrast present in the bladder
    - Complete disruption: no filling of the posterior urethra or bladder
- **Management**:
  1. Contusion
     - Do nothing
     - Small gauge catheter for one week
  2. Partial Rupture of Anterior Urethra
     - **No urethral catheterization!!!!**
     - Majority can be managed by suprapubic urinary diversion for one week
     - Penetrating partial disruption (e.g., knife, gunshot wound), primary (immediate) repair
  3. Complete Rupture of Anterior Urethra.
     - Unstable patient: a suprapubic catheter.
     - Stable patient: the urethra may either be immediately repaired or a suprapubic catheter is placed
  - Penetrating Anterior Urethral Injuries are generally managed by surgical debridement and repair

Retrograde urethrogram: contrast is injected through the urethra using a catheter and images are taken.

3.4.2 POSTERIOR URETHRAL INJURIES:

- Great majority of posterior urethral injuries occur in association with pelvic fractures,
- 10% to 20% have an associated bladder rupture
- **Signs**:
  - Blood at the meatus, gross Hematuria, and perineal or scrotal bruising.
  - High-riding prostate when examining by Digital rectal exam
- **Classification of posterior urethral injuries**
  - type I:(rare) stretch injury with intact urethra
  - type II : (25%) partial tear but some continuity remains
  - type III:(75%) complete tear with no evidence of continuity
  - In women, partial rupture at the anterior position is the most common urethral injury associated with pelvic fracture



- **Management**:
  - Type 1 and type 2 are treated with Stenting with a urethral catheter
  - Type 3:
    - Patient is at varying risk of urethral stricture, urinary incontinence, and erectile dysfunction (ED)
    - Initial management with suprapubic cystotomy and attempting primary repair at 7 to 10 days after injury.

3.5 EXTERNAL GENITAL INJURIES

- Penile fractures: during sexual intercourse
- Glans injury during circumcision
- Penile amputation and injury
- Scrotal injury
- Female external genital injury: in sports, crime or during vaginal labour

4 MCQS

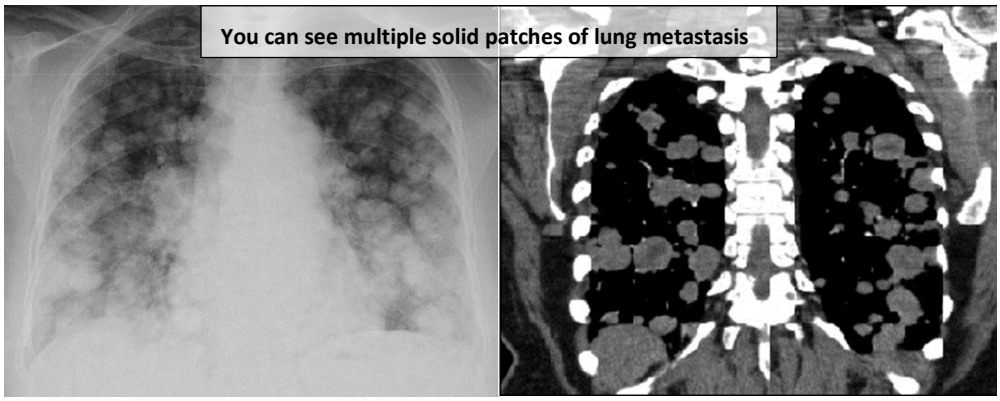
1. A 12 year old boy presented to the ER department with sudden onset of severe testicular pain with no history of trauma and no fever. What is the most likely diagnosis?
   A. Hydrocele
   B. Testicular torsion
   C. Tuberculosis Epididymitis
   D. Varicocele
2. If the diagnosis is testicular torsion how would you further proceed with your work up?
   A. Take the patient to do a CT scan
   B. Give the patient analgesia and ask him to return to you in 3 days
   C. Take the patient to the OR immediately for surgical exploration
   D. Administer antibiotics as testicular torsion is an infectious emergency
3. A 25 year-old male presented to the ER in a stable condition after a motor vehicle accident. He complains of left flank pain. You suspect renal injury. Which ONE of the following would be the best test to investigate renal injury?
   A. CT scan Urography
   B. Intravenous urography (IVU)
   C. MRI
   D. Renal ultrasound
4. Which ONE of the following is an indication for a surgical intervention in ureteric stones?
   A. Gross Hematuria
   B. If the stone is 6 millimetre in diameter
   C. Impaired renal function due to obstruction
   D. Stone in distal ureter

Answers: 1;B , 2;C , 3;A , 4;C

GENITOURINARY ONCOLOGY

1 RENAL TUMORS

- Benign tumors of the kidney are rare
- All renal neoplasms should be regarded as potentially malignant
- Renal cell carcinomas arise from the **proximal tubule cells** ⓘ
- Male: female ratio is approximately 2:1
- Increased incidence seen in von Hippel-Lindau syndrome
- Pathologically may extend into renal vein and inferior vena cava
  - *It could reach the heart*
  - *Tumor thrombus could obstruct IVC and causes bilateral DVT.*
- Blood born spread can result in 'cannon ball' pulmonary metastases
  - *The **lungs** are the commonest site for metastasis* ⓘ



"Cannon Ball" Metastases

1.1 CLINICAL FEATURES

- *The commonest presentation is: incidental finding* ⓘ
- 10% present with classic triad* of
  1) *Gross* hematuria
  2) Loin pain
  3) *Palpable* mass
     * *This is usually a sign of advanced disease*
- Other presentation include a pyrexia of unknown origin, hypertension
- Polycythemia due to erythropoietin production
- Hypercalcemia due to production of a PTH-like hormone

1.1.1 PARANEOPLASTIC SYNDROME

- A unique feature of renal cancer
- This is when the tumor starts secreting **hormones** e.g. ADH or EPO
- Treatment of this syndrome is by treating the underlying cause by surgical removal, not symptomatic treatment.
- Other systemic manifestations of paraneoplastic syndrome include:
  - Pyrexia of unknown origin (PUO)
  - Hypertension
  - Polycythemia (due to erythropoietin production)

Onchocytoma is the commonest **benign** tumor.

Most common kidney **cancer** is **renal cell carcinoma.**

The commonest renal cell carcinoma histological **subtype** is **clear cell** carcinoma

The renal cell carcinoma arising from the collecting duct cells is *collecting duct carcinoma* of the kidney.

Familial **papillary cell carcinoma is hereditary** and runs in families (all family members should be screened).

Von Hippel-Lindau Syndrome:

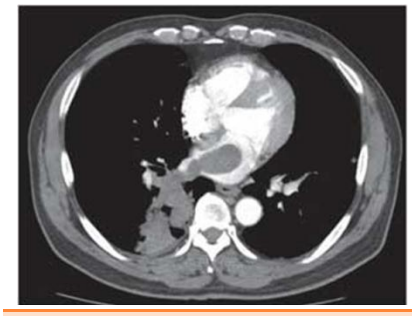
- Genetic disease
- Mutation: short arm of chromosome 3 ⓘ
- CNS hemangio-blastomas, pheo-chromocytomas, pancreas and kidney cysts, renal cell carcinoma
- Also associated with adrenal gland malignancies

- ADH: Anti-diuretic hormone
- EPO: Erythropoietin

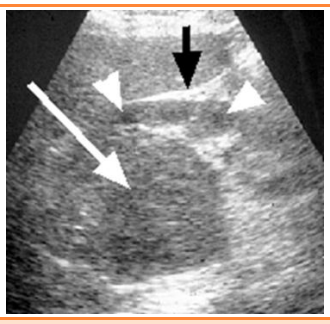
  - Hypercalcaemia due to production of a PTH-like hormone
  - Non-metastatic hepatic dysfunction called **Stauffer's syndrome**, characterized by elevated liver enzymes
    - ☝ Remember: no liver metastasis, no jaundice
- All treated by surgical removal of kidney tumor EXCEPT hypercalcaemia, which can be treated medically ⓘ

1.2 INVESTIGATIONS

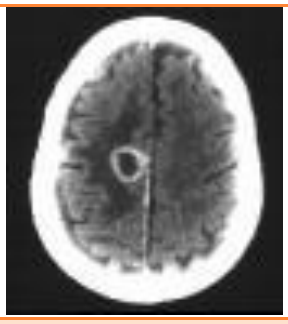
- Diagnosis can often be confirmed by renal ultrasound
- CT scanning allows assessment of renal vein and caval spread
  - *It is used for staging*
- Echocardiogram (TEE) should be considered if clot in IVC extends above diaphragm



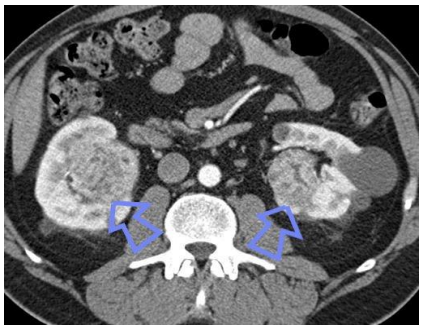
CT of the patient's chest when he was first diagnosed with intracardiac extension

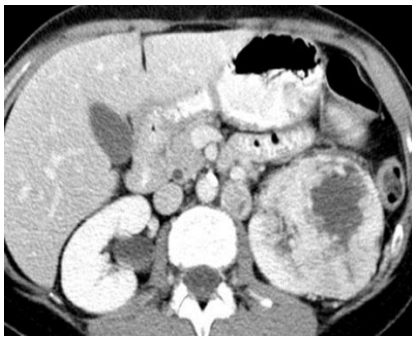


White arrow shows a mass in the right ventricle



Solitary mass in the brain due to metastasis. Solitary masses → surgical removed





When you find bilateral tumors, think of familial syndromes like VHL

If there is a **LOCALIZED** tumor in the heart, then we should treat it surgically.

** Only if localized*

When tumor thrombus extends above diaphragm, survival rate ~20%

1.3 TREATMENT

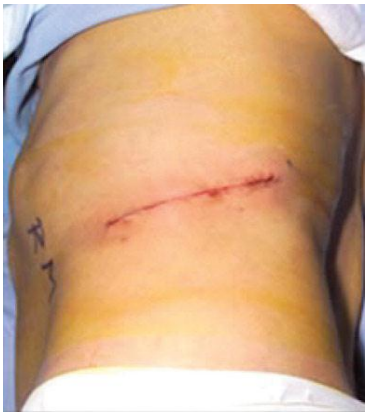
- Unless extensive metastatic disease, it invariably involves **surgery**
- Surgical options usually involve a **radical nephrectomy**
- Kidney approached through either a transabdominal or loin incision
- Renal vein ligated early to reduce tumor propagation
- Kidney and adjacent tissue (adrenal, perinephric fat) excised
- Lymph node dissection of no proven benefit
  - *Remove only for lab purposes and staging*
  - *Whether you remove them or not, patients will have recurrences*
- Solitary (e.g. lung metastases) can occasionally be resected

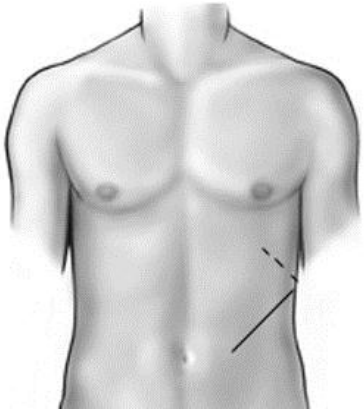
ⓘ **Remember**:

Kidney tumors are both radio-resistant and chemo-resistant, but radio/chemo Rx indicated in cases of **symptomatic bone metastasis**, in order to relieve pain

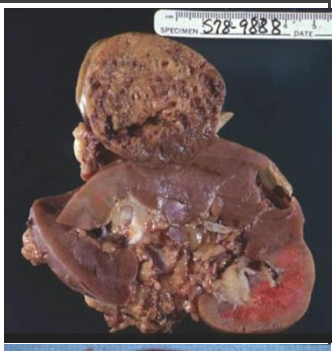
- Radiotherapy and chemotherapy have NO role
  - *Indicated in case of symptomatic bone metastasis to reduce pain*
- Immunotherapy can help (performance status)
  - *Monoclonal antibodies, interferon, cytokine inhibitors*
  - *Very cytotoxic*
  - *Given only to patients with good performance status*
  - *Not curable but it can prolong his life for 6-8 months*

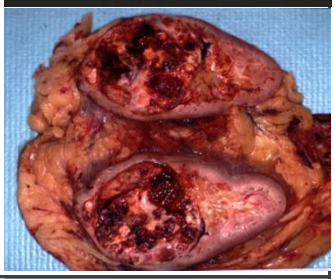
Open radical nephrectomy: This is a surgery that causes a big scar that has to cut the muscles so the patient will suffer and feel pain with respiration





Gross Appearance and Histopathology (Post-Nephrectomy)





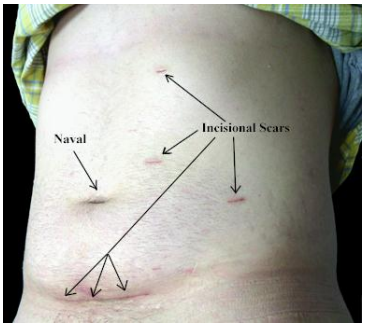
Laparoscopic nephrectomy: *(GOLD STANDARD)*

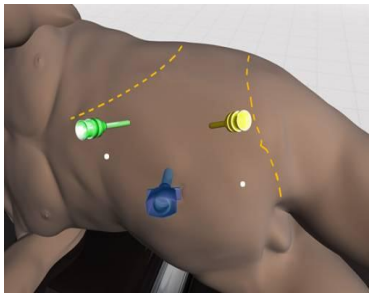
Advantages: 1. Shorter hospital stay 2. Less pain 3. Same results.

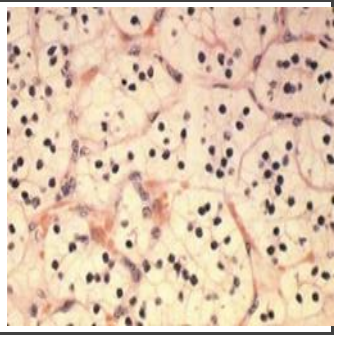
We use the groin incision to remove large tumors, because that way we do not need to cut muscle, it is a muscle splitting incision.

For post-menopausal women who have undergone hysterectomy > Transvaginal approach

Very clear, impacted cells with dark nuclei and clear cytoplasm. This is the commonest histo-pathological subtype ▼







Staging of kidney tumor includes:

1. Clinical staging by CT scan
2. Pathological staging

Grading system for kidney cancer is called:

Fuhrman system

1.4 PROGNOSIS

- Early stage: 5 year survival is 95%
- Metastatic disease: 3-6 months average survival

2 BLADDER TUMORS

2.1 PATHOLOGY

- Of all bladder carcinomas:
  - **90% are transitional cell carcinomas (TCC)**
  - 5% are squamous carcinoma
  - 2% are adenocarcinomas (*due to congenital fistulas; develops in the dome of the bladder*)
- TCCs should be regarded a 'field change' disease with a spectrum of aggression
- **80% of TCCs are superficial and well differentiated**
  - *Above the muscle layer (muscularis propria)*
  - Only 20% progress to muscle invasion
  - Associated with good prognosis, *but higher recurrence rate*
- **20% of TCCs are high-grade and muscle invasive**
  - 50% have muscle invasion at time of presentation
  - Associated with poor prognosis

Squamous carcinoma:

- Bad prognosis, in fact the worst
- High risk groups (chronic irritation):
  - Smokers*
  - Chronic UTI
  - Stones
  - Chronic indwelling catheter
  - Spinal cord injury
  - Schistosomiasis*

2.2 ETIOLOGY

1. Occupational exposure
   a. ~20% of transitional cell carcinomas are believed to result from occupational factors
   b. Chemical implicated - aniline dyes, chlorinated hydrocarbons
2. Cigarette **smoking***
3. Analgesic abuse e.g. phenacitin
4. Pelvic irradiation - for carcinoma of the cervix
5. Schistosoma haematobium* associated with increased risk of squamous carcinoma ⓘ

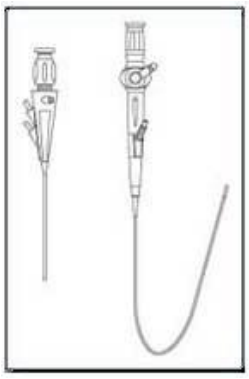
2.3 CLINICAL FEATURES

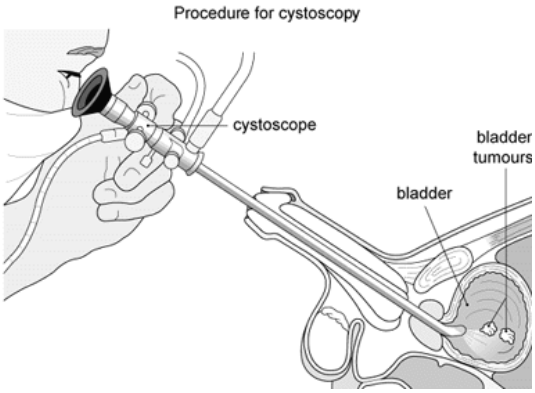
- 80% present with **painless hematuria**
  - Gross painless hematuria
  - Terminal hematuria
- Also present with treatment-resistant infection or bladder irritability and sterile pyuria *(DDx: TB)*

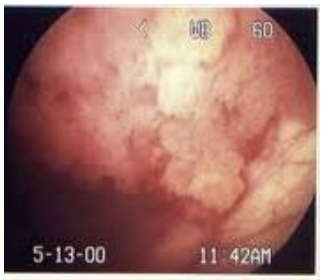
Painless gross hematuria is considered to be cancer until proven otherwise.

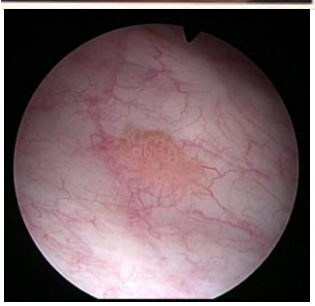
2.4 INVESTIGATIONS (OF PAINLESS HEMATURIA)

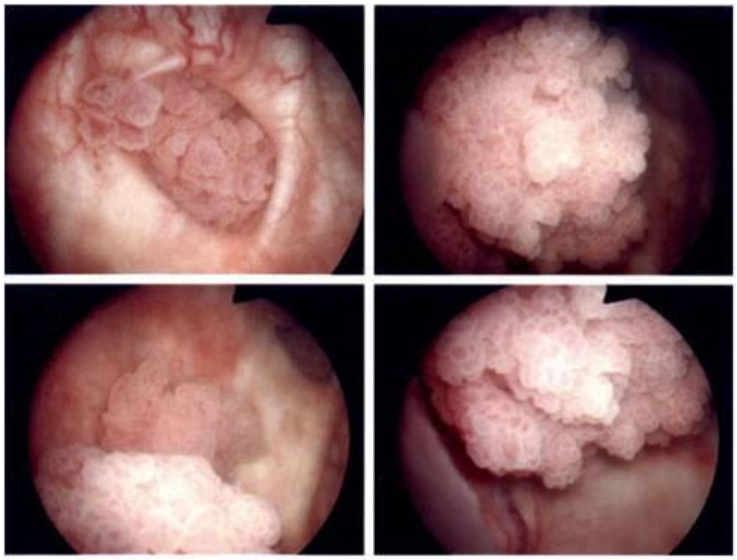
1. Urinalysis
2. Ultrasound - bladder and kidneys
3. KUB - to exclude urinary tract calcification
4. **Cystoscopy** (*a **MUST** in this case*)
5. Urine Cytology
6. Consider IVU if no pathology identified (*shows filling defect, or sometimes hydronephrosis due to obstruction of the ureters, which is a bad sign indicating progressive disease*)









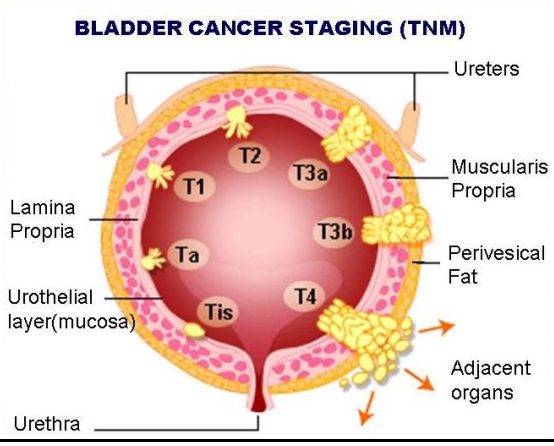


Bladder diverticulum causes stagnation of urine → chronic irritation. Diverticulum appears as a pouch.

2.5 PATHOLOGICAL STAGING

- Requires bladder muscle to be included in specimen
- Staged according to **depth** of tumor invasion

Superficial	Tis	In-situ disease
	Ta	Epithelium only
	T1	Lamina propria invasion
Invasive	T2	Superficial muscle invasion
	T3a	Deep muscle invasion
	T3b	Perivesical fat invasion
	T4	Prostate or contiguous muscle



T2 and above needs removing the whole bladder

Grading:

- *G1: Well differentiated*
- *G2: Moderately well differentiated*
- *G3: Poorly differentiated*

2.5.1 CARCINOMA IN-SITU

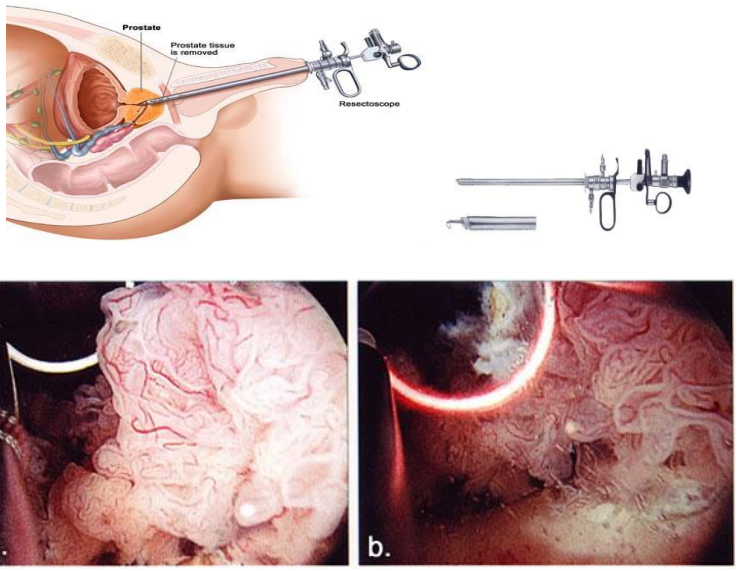
- Carcinoma-in-situ is an aggressive disease
- Often associated with positive cytology ⓘ
- 50% patients progress to muscle invasion ⓘ
- Consider immunotherapy
- If fails patient may need radical cystectomy

2.6 TREATMENT

2.6.1 SUPERFICIAL TCC

- Requires transurethral resection and regular cystoscopic follow-up
  - *To watch out for recurrence due to the high recurrence rate of superficial TCC*
- Consider prophylactic chemotherapy if risk factor for recurrence or invasion (e.g. high grade)
  - *High risk: 1.**Multiple** tumors 2. **Big** tumors 3.Carcinoma **in situ***
- Consider immunotherapy
  - BCG ⓘ = attenuated strain of Mycobacterium bovis
  - Reduces risk of recurrence and progression
  - 50-70% response rate recorded
  - Occasionally associated with development of systemic mycobacterial infection

Transurethral Resection of Bladder Tumor (TURBT)



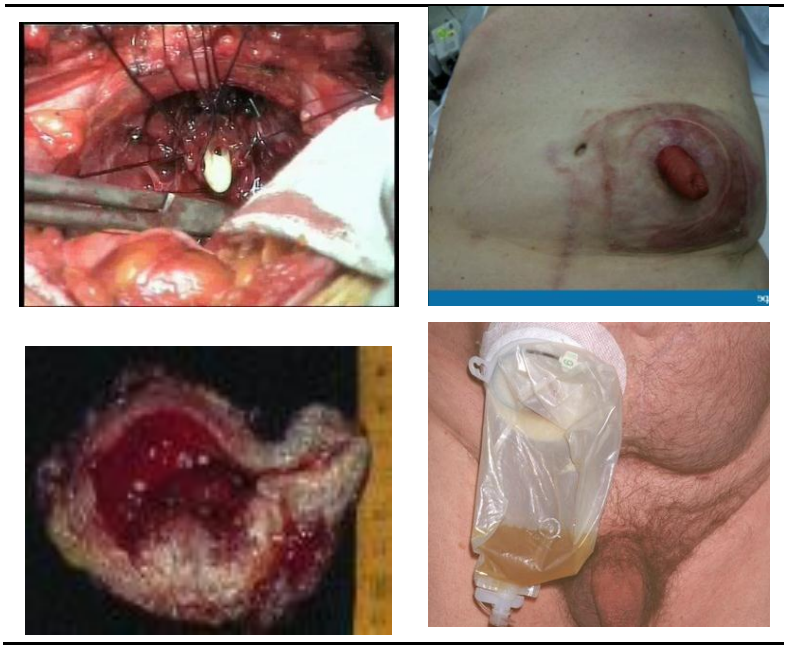
2.6.2 INVASIVE TCC

- Radical cystectomy has an operative mortality of about 5%
- Urinary diversion achieved by:
  - Ileal conduit
  - Neo-bladder

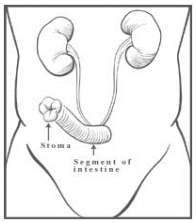
***Radical cystectomy**: removal of bladder, prostate, distal ureter and lymph nodes*

** In females: also the uterus, cervix and anterior vaginal wall*

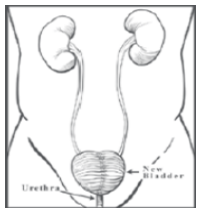
- Local recurrence rates after surgery are approximately 15% and after radiotherapy alone 50%
- Pre-operative radiotherapy is no better than surgery alone
- Adjuvant chemotherapy may have a role



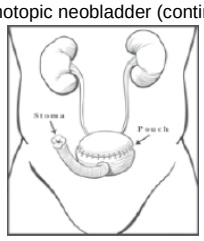
Ileal conduit (incontinent)



Continent cutaneous reservoir



Orthotopic neobladder (continent)



3 PROSTATE TUMORS

PROSTATE CANCER

- Commonest malignancy of male urogenital tract ⓘ
  - *8th most common tumor in in KSA*
- Rare before the age of 50 years
  - *Screening is recommended at age 40*
- Found at post-mortem in 50% of men older than 80 years
  - *The patient usually dies from other causes (it will not kill the patient)*
- 5-10% of operations for benign disease reveal unsuspected prostate cancer

Screening program in North America: for males above the age of 40 every year

- PSA Test
- Digital Rectal Exam

If any of them positive this is an indication to take a **biopsy.**

3.1 PATHOLOGY

- The tumors are **adenocarcinomas** ⓘ
- Arise in the **peripheral zone** of the gland ⓘ
- Spread through capsule into peri-neural spaces, bladder neck, pelvic wall and rectum
- Lymphatic spread is common
- Hematogenous spread occurs to axial skeleton
- Tumors are graded by Gleeson classification

Malignant prostate tumors usually arise in the peripheral zone, while benign prostate hyperplasia (BPH) arises in the transitional zone.

3.2 CLINICAL FEATURES

- Majority these days are picked up by screening

- 10% are incidental findings at TURP
- Remainder present with bone pain, cord compression or leuco-erythroblastic anemia
- Renal failure can occur due to bilateral ureteric obstruction

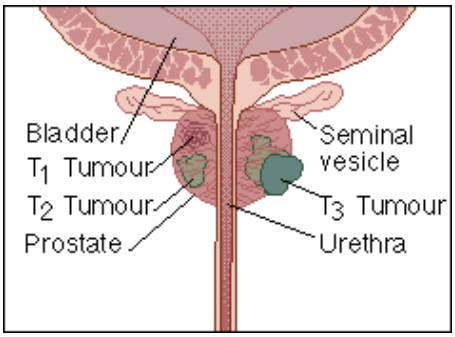
3.3 DIAGNOSIS

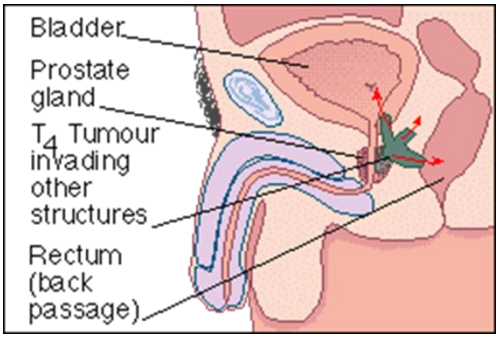
- With locally advanced tumors diagnosis can be confirmed by **rectal examination**
- Features include hard nodule or loss of central sulcus
- Transrectal biopsy should be performed
- Multiparametric MRI may be useful in the staging of the disease
- Bone scanning may detect the presence of metastases
- Unlikely to be abnormal if asymptomatic and PSA < 10 ng/ml

3.3.1 SERUM PROSTATE SPECIFIC ANTIGEN (PSA)

- Kallikrein-like protein produced by prostatic epithelial cells
- 4 ng/ml is the upper limit of normal
- >10 ng/ml is highly suggestive of prostatic carcinoma
- Can be significantly raised in BPH
- Useful marker for monitoring response to treatment

3.3.2 STAGING





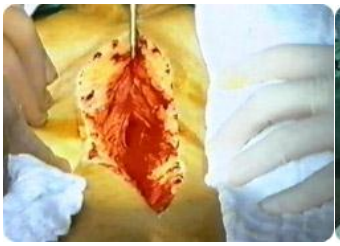
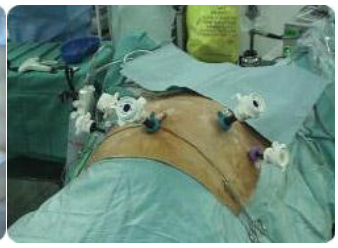
3.4 TREATMENT

- More men die **with** prostate cancer <u>than</u> **from** prostate cancer
- Treatment depends on stage of disease, patient's age and general fitness
- Treatment options are for:
  - Local disease
    - Observation *(old men ≥ 80 with localized disease)*
    - Radical radiotherapy *(prostate cancer is radiosensitive)*
    - Radical prostatectomy
  - Locally advanced disease
    - Radical radiotherapy
    - Hormonal therapy
  - **Metastatic disease**
    - **Hormonal therapy**

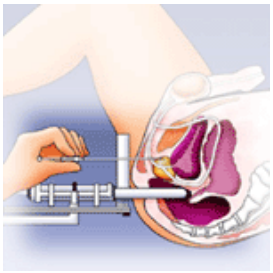
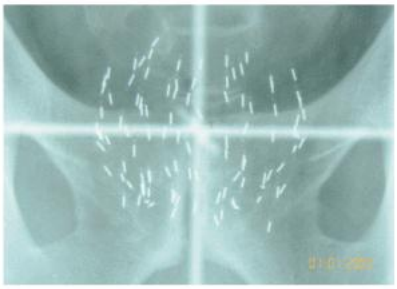
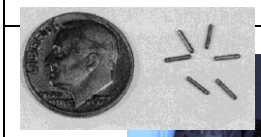
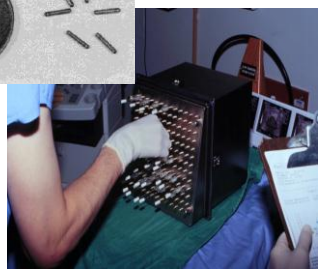
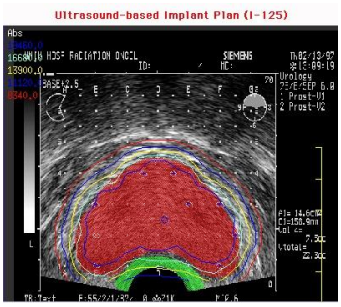
3.4.1 HORMONAL THERAPY

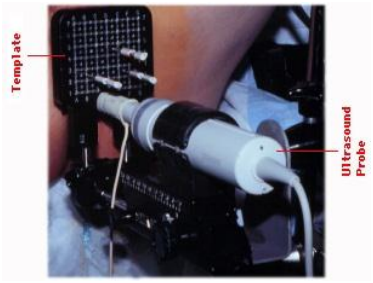
- 80-90% of prostate cancers are androgen dependent for their growth ⓘ
- Hormonal therapy involves androgen depletion
- Produces good palliation until tumors 'escape' from hormonal control
- Androgen depletion can be achieved by:
- Bilateral orchidectomy
- LHRH agonists (e.g. goseraline)
- Anti-androgens (e.g. cyproterone acetate, flutamide, biclutamide)
- Complete androgen blockade

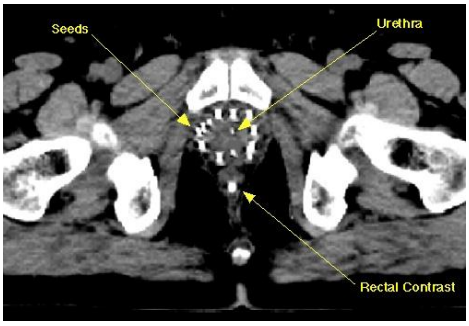
Open/Laparoscopic/Robotic

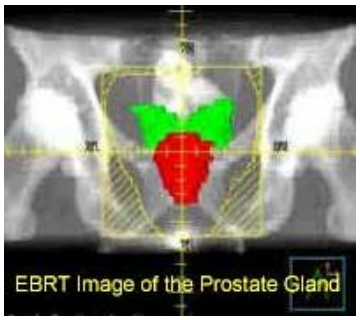

Brachytherapy: internal radiotherapy, in which the radiation source is inside the body

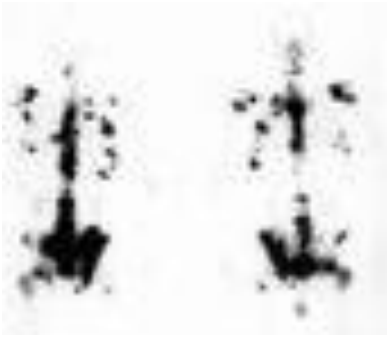






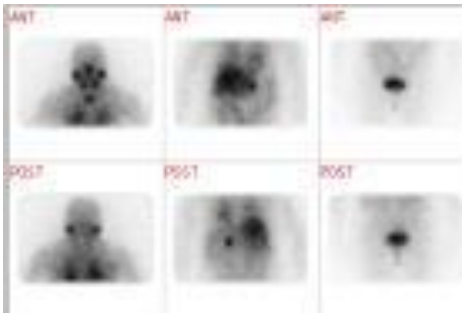




EBRT: External beam radiation therapy







4 TESTICULAR TUMORS

A disease of young men

- Commonest presentation: ipsilateral ***painless*** testicular swelling
- Commonest malignancy in young men ⓘ
- Highest incidence in Caucasians in northern Europe and USA
- Peak incidence for teratomas is 25 years and seminomas is 35 years ⓘ
- In those with disease localized to testis > 95% 5-year survival possible
- Risk factors include cryptorchidism, testicular maldescent, Klinefelter's syndrome, and *testicular torsion*

4.1 CLASSIFICATIONS

- Seminomas (~50%) ☛ Radiosensitive
- Non-Seminoma (~50%) ☛ Radio-resistant
  - Teratomas
  - Yolk sac tumors
  - Embryonal
  - Mixed Germ cell tumor

4.2 INVESTIGATIONS

- Diagnosis can often be confirmed by **testicular ultrasound**
- Pathological diagnosis made by performing an inguinal orchidectomy
- Disease can be staged by thoraco-abdominal CT scanning
- **Tumor markers** are useful in staging and assessing response to treatment
  - Alpha-fetoprotein (α-FP)
    - Produced by yolk sac elements

        - Not produced by seminomas
    - Beta-human chorionic gonadotropin (β-hCG)
        - Produced by trophoblastic elements
        - Elevated levels seen in both teratomas and seminoma
    - LDH

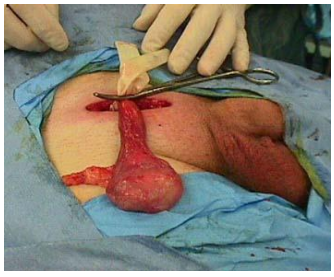
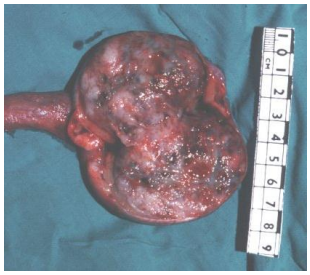
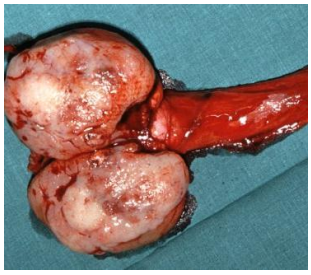
4.3 STAGE DEFINITION

Stage I **IM**	Disease confined to testis Rising post-orchidectomy tumor marker
Stage II **A** **B** **C**	Abdominal lymphadenopathy < 2 cm 2-5 cm > 5 cm
Stage III	Supra-diaphragmatic disease

4.4 TREATMENT

4.4.1 SEMINOMA

- Seminomas are radiosensitive
- The overall cure rate for all stages of seminoma is approximately 90%.
- Stage I and II disease treated by **inguinal** orchidectomy plus
    - Radiotherapy to ipsilateral abdominal and pelvic nodes ('Dog leg') or
    - Surveillance
- Stage IIC and above treated with chemotherapy

? True or False:

Radical orchiectomy is done within scrotum.

False. Done through the groin.

4.4.2 NON-SEMINOMA

- Non-Seminoma are not radiosensitive
- Stage I disease treated by orchidectomy and surveillance vs. RPLVD vs. chemo
- Chemotherapy (BEP = Bleomycin, Etopiside, Cisplatin) given to:
    - Stage I patients who relapse
    - Metastatic disease at presentation

5 MCQS

1) The most common presentation of renal tumors is:

a. Fever of unknown origin
b. Hypertension
c. Incidental finding
d. Hematuria

2) **Which of the following is the commonest malignancy in young men?**
   a. Lung
   b. Testicular
   c. Colon
   d. Bone
3) **Nephrouretroectomy is the treatment of choice in :**
   a. Transitional cell carcinoma of the renal pelvis
   b. Renal cell carcinoma
   c. Non-functioning pyelonephrotic disease
   d. Non-functioning tuberculosis
   e. Angiomyolipoma
4) **Regarding cancer prostate all true except:**
   a. It's a very common disease in the kingdom
   b. The growth of the tumor can be affected by steroids
   c. Usually treated by testosterone
   d. Can be treated by estrogens
   e. Can present with back pain
5) **Benign prostatic hyperplasia all true except:**
   a. Is a disease of the young
   b. Usually presents with hematuria
   c. Can present with renal failure
   d. Usually present with hydronephrosis
   e. Can cause bladder stones

🗝 **Answers**: 1 = c, 2 = b, 3 = a, 4 = c, 5 = a

ACUTE ABDOMINAL PAIN IN CHILDREN

1 OBJECTIVES

- Realize the impact of age
  - Where/who are the history sources
- Recognize and interpret the
  - important symptoms
  - Important signs

2 HISTORY [THE IMPACT OF AGE]

- Less than 3-4 year
  - Verbal expression
  - Difficult to communicate
  - Fear of strangers
- **History sources**
  - **Mother is the best source**
  - Social barrier less than what we expect
  - **Father is not very reliable**
  - Nurses are reliable
    - Not always possible / available
    - Important in ICU
- Other doctors

3 SYMPTOMS OF SURGICAL ABDOMEN

- **Feeding**
  - Feeding well ➔ healthy baby
  - Poor feeding
    - Sick baby → from any GI or systemic cause (ear infection)
    - GI obstructed
    - Pain
- **Vomiting** → sick baby
  - Regurgitation is frequent in babies
    - considered Pathological if associated with failure to gain weight, respiratory infection
  - Frequency
  - Color
  - Force
    - Projectile → proximal obstruction
    - Small amount after each feeds → regurgitation
- **Bowel movement (BM)**
  - Frequency
    - What is the normal for infant? 4 per day to once in 2-3 days
    - Constipated, obstructed

        - Failure to pass meconium in the first 24-48hrs newborns, Meconium is 80% passed in the first 24 hrs, 95% in the 48hr. Greenish, sticky, dark
    - Consistency
        - Loose / watery → diarrhea
        - Firm & dry → constipation
    - Color
        - Very pale → ?
        - Black → Melena
        - Bright red
- Upper & Lower GI Bleed is very rare in children. On the other hand anal fissures are common.
- **Crying baby**
    - Babies communicate their needs by crying
        - Hungry
        - Wet (Urinated)
    - At >6 month ➔ emotions →they learn to cry for other reasons
        - Want to be carried
        - Want to play
    - Baby who continue to cry, refuse feeding and dry ➔ "irritable baby"
    - pain
        - Abdominal pain
        - Other causes → Ear ache
    - Non-crying baby with reasons can be worrisome → very sick
- **Development**
    - Growth (height and weight)
        - Chronic problems (Metabolic, **Nutrition => gut health**)
    - Psychological
        - Mental problem, chromosomal abnormalities
    - Motor
        - Syndrome
        - Metabolic

Meconium is the early feces (stool) passed by a newborn soon after birth, before the baby has started to digest breast milk (or formula).

4 RELAYED SYMPTOMS (BY PARENTS)

- **External abnormality**
    - Anything that is not normal
        - Swelling
            - Abscess (swelled, red and tender abdomen)
            - Mass (swelling and non-tender)
            - Hernia (swelling that comes and goes in the inguinal region)
        - Color changes
            - Inflammation
            - Rash
            - Vascular malformation
    - Mental changes
        - ↓Responsiveness
            - Sleepy
            - Not interested in feeding

- Indicates; sepsis, shock, CNS trauma, metabolic (O2, Glucose, urea)

5 ABDOMINAL PROBLEMS

1. Vomiting
2. Constipated / diarrhea
3. Poor feeding
4. Abdominal distension
5. Palpable mass
6. Very dark or very pale colored stool
7. Jaundice

6 PHYSICAL EXAM

- Vital signs
  - Fever
  - RR, BP, HR, O2 Sat
  - Babies usually have higher HR, RR. Lower BP. The younger the child, higher the values
- Consciousness (crying)
  - Crying baby →not very sick (not critical)
  - Unusually calm baby who doesn't respond normally → sick
- Exam while crying
  - Can't hear the chest well
    - Focus on inhalation
  - Can't examine abdomen well
    - Examine while taking breath
    - Keep hand on abdomen
  - Can't concentrate
    - Parent are stressed → less time
- **Never do a rectal examination on babies. It's not helpful, it causes anal fissures and it's very painful**.
- Otherwise it's similar to adults
- A good history = a good logical story, Known major Predisposing factors → → Describe the current problem → other risk factors → Symptoms of other possible complications

7 INVISTGATIONS

- Due to the relative difficulties in taking a reliable history and performing an accurate physical exam
- We tend to depend more on investigations in diagnosing the underlying problems in infants

8 QUESTIONS

1. **5 weeks old boy brought to you by his parents because of recurrent vomiting. Parents indicated that the baby vomits with significant force all the milk he had ate completing the feed. Where do you think is the level of obstruction?**

a) Esophagus
b) Middle ileum
c) Proximal colon
d) Pylorus

2. The child who is most likely to need a surgical consultation?

a) 1 month-old breast fed baby didn't pass stool for 4 days
b) 5 day-old baby with fever , passing soft light yellow stool
c) 3 day-old baby didn't pass meconium during the first 48 hours of life
d) 12 month-old baby didn't pass frequent liquid stool for one day

3. 6 months old baby boy presented to emergency department with history of possible swallowing of metallic object. The father said he was not sure if the baby swallowed the object. The next most appropriate is:

a) Perform an upper GI endoscopy
b) Perform a chest X-ray of the chest and upper abdomen
c) Perform a chest X-ray to the neck and chest, AP and lateral
d) Ask the mother about the incidence

🗝 Answers: 1;D, 2;C , 3;D

COMMON INGUINOSCROTAL CONDITIONS IN CHILDREN

1 INGUINAL HERNIA

1.1 INTRODUCTION

- **Hernia** is the protrusion of an organ or the fascia of an organ through the wall of the cavity that normally contains it
- **Inguinal Hernia:** Extension of the perineum (and usually its contents – small intestines) through the inguinal canal
- It has two subtypes: indirect (more common) and direct
    - An indirect inguinal hernia follows the tract through the inguinal canal
    - A direct inguinal hernia usually occurs due to a defect or weakness in the transversalis fascia area of the Hesselbach triangle
- 99% of groin hernias are indirect inguinal hernia

Umbilical hernia: Intersection between cranial fold, abdominal wall, lateral fold. They have to meet in the center – most of the times they do not meet 100% resulting in a defect in the umbilicus > umbilical hernia

An **incisional hernia** occurs when the defect is the result of an incompletely healed surgical wound

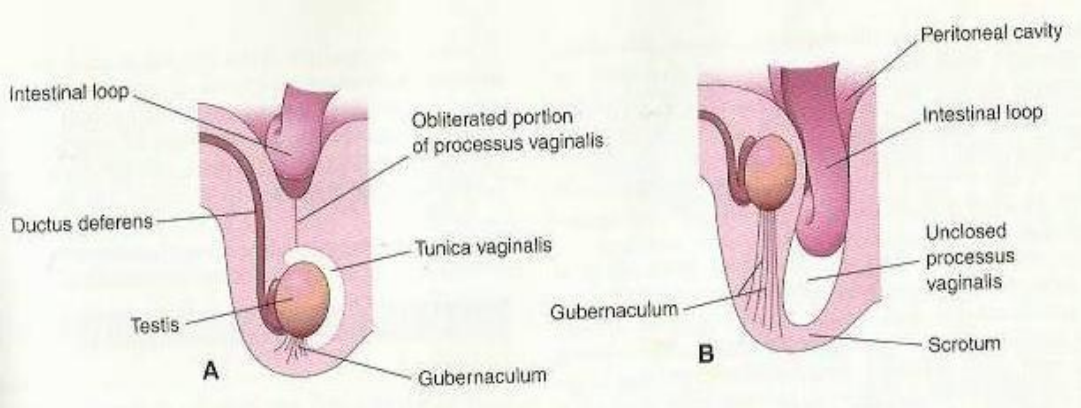
1.2 ANATOMY OF INGUINAL CANAL

- It extends from the deep inguinal ring which is the connection between peritoneal cavity and the groin to the external ring.
- Boundaries:
    - Anterior: External oblique muscle
    - Posterior: Transversalis fascia
    - Inferior wall (floor): Inguinal ligament
    - Superior wall (roof): Internal oblique and transversus abdominis
- The deep ring is lateral to the inferior epigastric vessels.
    - It is the LANDMARK to differentiate between direct and indirect inguinal hernia
    - This indicates an Indirect Inguinal Hernia
- If it's plugged medial to the inferior epigastric vessels then it's a direct inguinal hernia
- It is difficult to differentiate between direct and indirect inguinal hernia clinically

1.3 ETIOLOGY

- Extension of the prenium (and usually its contents) through the inguinal canal because of:
    - Patent processes virginals: The embryological canal that the testes descend through to the scrotum
    - Congenital inguinal hernia: The processes virginals remains in open communication with the peritoneal cavity.
    - A loop of intestine may herniated through it into the scrotum.
    - The opening may be:
        - Incomplete

- Complete



1.4 PRESENTATION

- The most common presentation is swelling " plugging " in the groin area
- Painless Inguinal swelling :
  - Intermittent (appears and disappears) – the mother will tell you the swelling comes and go
  - The right side is affected more than the left side- more in males ⓘ
  - The swelling disappear when lying down and appear when standing up due to the effect of the gravity ⓘ
- In the hernia, swelling starts in the groin then descends to the scrotum (opposite to hydrocele)
- There is thickness of the spermatic cord (felt in the groin area)
- Reducibility of the swelling ⓘ

1.5 TYPES

1. **Simple:** The swelling is reduced spontaneously without mechanical enhancement
2. **Complicated**: The swelling is reduced by an expert hand only and not spontaneously – also called Incarcerated hernia
3. **Strangulated**: The swelling does not get reduced + there is a decrease of the blood supply to the herniated sac thus resulting in ischemia (this type is painful)

1.6 COMPLICATIONS

1. Incarceration
2. Strangulation
3. Obstruction of the bowel
4. Testicular atrophy: Due to compression of the blood vessels

1.7 MANAGEMENT

- Simple non-complicated hernia

- If it was not complicated, we do herniotomy as soon as it is feasible.

- Incarcerated hernia
  - +/- Sedation and analgesia
  - Check to reduce it
  - Urgent herniotomy
- Strangulated hernia (Emergent herniotomy in 24-48 hours)
  - Irreducible hernia – urgent surgery" in a day or 2" to avoid complications ".
  - It's irreducible because the hernia contains dead tissue, thus stimulating inflammatory reaction around it.(So, you cannot push it in).
  - Compression in the testicular vessels decreases the blood flow to the testis > atrophy " ⓘ
  - What t is the danger of leaving hernias in females? ovary – not fixed will be necrotic
- Obstructive type inguinal hernia: it irreducible, content of the hernia is bowel (mainly small bowel), it cause obstruction of the bowel
  - How do patient with obstructive inguinal hernia will present?
    - Abdominal distention
    - Vomiting: (greenish. Why? Because the obstruction is distal to the ampulla of vater (2nd part of duodenum), so the bile will go to the bowel where there is obstruction which prevents it from going down. Since it has to go somewhere, the only way is up.
    - Constipation – and maybe obstipation (complete obstruction of the bowel, no pass of stool and gas) ⓘ
  - When you see patient with abdominal distention and growing bulge, patient with obstructive inguinal hernia what will be the next step? EMERGENCY surgery, why? To avoid bowel ischemia
  - General rule for future doctors, never leave a patient with obstructive bowel without intervention.
  - How do they present?
    - SEVERE pain
    - Swelling
    - Redness of the Over lying skin (change the color of the over lying skin is a bad sign for any pts with hernia), why? It main the hernia id strangulated subtype and need emergent surgery (herniotomy).

It's different in children than in adults.

- In children, the inguinal hernia is indirect (so go from the deep ring through the canal to the external ring)

- It's fixed by separating the hernia sac from the other content of the inguinal hernia which differs between males and females.

- So you have to separate the sac from the adjacent structure.

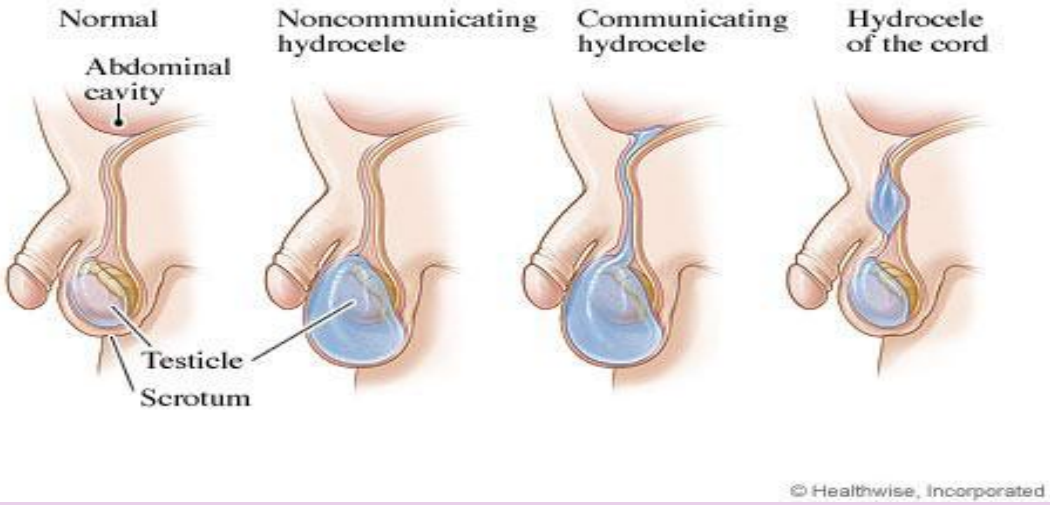
- In children, you have to do simple high ligation at the level of the deep ring and that is herniotomy.

-If any content is present in the hernia, you have to get it back to its normal location.

2 HYDROCELE

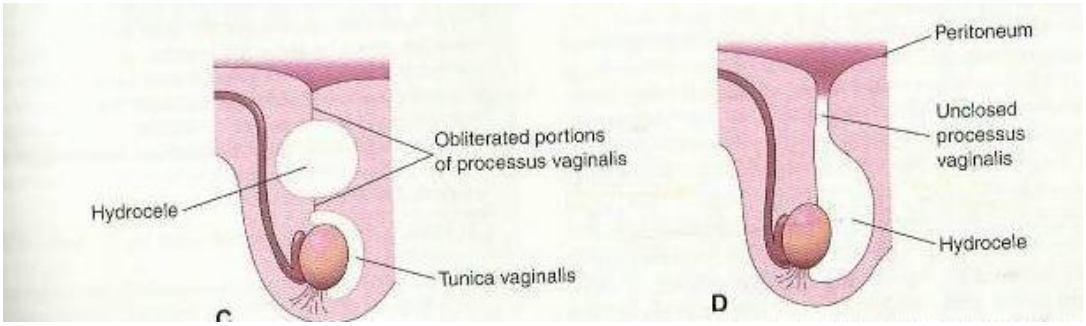
- **Hydrocele:** Accumulation of fluid in the testes ((so it is a fluid filled sac around the testis))
- **Types:**
  a. Incysted hydrocele: The fluid around the testicles is absorbed
  b. Non-communicated hydrocele: The fluid stays around the testicles and is not absorbed.(there was a tunnel then it was obliterated
  c. Communicated hydrocele: The fluid flows back and forth between the scrotum and the abdomen.(communication between abdominal and scrotum, so you can squeeze the fluid back to the peritoneum cavity)

d. Hydrocele of the cord: The fluid is located in the spermatic cord, between the scrotum and the abdomen.



2.1 ETIOLOGY

- Same as inguinal hernia: P atent processus vaginalis
- The opening is smaller than in inguinal hernia so only the fluid only comes through
- Fluid may accumulate forming middle part of the processus vaginalis. (C)
- If the abdominal end of the processus vaginalis remains open but is too small to permit herniation of intestine peritoneal fluid passes into the patent processus vaginalis forming a hydrocele of the testis. (D)



2.2 PRESENTATION ⓘ

- Non reducible swelling
- Painless (asymptomatic), swollen testicle, which feels like a water balloon. A hydrocele may occur on one or both sides.
- During a physical exam, the doctor usually finds an swollen scrotum that is not tender. Often, the testicle cannot be felt because of the surrounding fluid. The size of the fluid-filled sack can sometimes be increased and decreased by pressure to the abdomen or the scrotum.
- If the size of the fluid collection varies, it is more likely to be associated with an inguinal hernia.

- The groin is not swelled as IH (get above the swelling) ⓘ

2.3 MANAGEMENT

- Hydroceles are usually not dangerous, and they are usually only treated when they cause discomfort or embarrassment, or if they are large enough to threaten the testicle's blood supply.
- The treatment is not urgent so we can wait until the 2nd year of age because it may spontaneously get resolved. If not, we do surgery. ⓘ
- What the different between IH and hydrocele (both are common & in children)?
  - Hydrocele is a fluid full sac in the scrotum
  - Etiology:
    - It`s the same as of IH (persistent of patent processes vaginalis), ppv: is the extension of the peritoneal out of the abdominal cavity, enter through the deep ring, IC, and the external ring.
    - The open of the ppv is small in hyrocele, but in hernia it big so allow abdominal content to go through it .
  - If you can feel the testis it`s hernia, if u can`t feel it cuz of all fluid it hydrocele.
- Why to different between them? Cuz if it is hydrocele you don`t need to fix it right away, majority will disappear by itself, wait for 2 years if didn't disappear enter to fix it.

Hydrocele	Inguinal Hernia
Scrotal swelling	Inguino-scrotal swelling
Not reducible	Check reducibility ⓘ
+ve transillumination (not specific)	May have +ve transillumination
The pt is fine & not irritable	The pt is irritable

3 UNDESCENDED TESTES

3.1 INTRODUCTION

- Types:
  a. **True undescended testes:** Normally, testes descend from the genital ridge to the scrotum. If it stopped anywhere in the normal pathway above the scrotum, it is called true undescended testes “Retained testis”.
  b. **Ectopic:** It stops anywhere rather than the normal pathway, most commonly in the superficial inguinal pouch.
  c. **Retractile:** The testis descends normally at birth, but goes up again due to hyperactivity of the cremasteric muscle (cremasteric reflex). It may be milked again though. It can also ascend in the inguinal canal spontaneously.
- Normal phenomenon in children; the majority of them resolves.
- Incidence:
  - At birth: 3-4%

Testis descend from the abdominal at the kidney level in the retroperitoneum, descend to the inguinal canal to the scrotum any rest in this processes we call it true Undesigning testis.

  - At one year: 1%
  - Pre-term: 30%
- It's important to know the different types because each has a different management.

3.2 PRESENTATION

- Empty scrotum
- The testis could be :
  - Palpable : you can feel it in the groin area
  - Not palpable (it usually in the abdominal cavity)
  - Non palpable Undesigning testis if u can't feel the testis in groin what will be the next step?! ⓘ
- We expect the testis in abdomen > so to visualize the abdominal activity we will do laparoscopy trying to search for testis.
- Laparoscopy can be diagnostic and therapeutic to bring the testis down to scrotum.

3.3 DIAGNOSIS

- Imaging has no role unless the testis was not palpable
  - In this case, we use MRA, MRI and US to determine the site of the testis.
  - The best imaging modality for Dx is MRA.
  - The gold standard tool for Dx and Rx is Laporoscopy. ⓘ

The most common Dx method is the clinical picture and the mother's fear.

3.4 MANAGEMENT

- The retractile type does not need medical intervention. It usually returns to its normal position at puberty because of the increased weight of the testes and well development of the muscles.
- But the other types need surgical intervention:
  - The treatment should be done at the age of 6-12 to give a chance for spontaneous testicular descent after birth.
  - The reason we don't wait 2,3 or 4 years is because fixation of the testis will be affected by then.
  - If it's palpable
    - Open orchiopexy: Small incision, same as hernia, in which open groin and search for testis.
  - If it's non-palpable:
    - Laparoscopy-assisted orchiopexy
    - Two stages Fowler-Stephens orchiopexy
- Other indications of surgery: (also considered possible complications)
  - Abnormal fertility
  - Testicular tumor
  - Cosmetic/Social
  - Trauma/Torsion
- The higher the testes the worst the prognosis.
- Also, if it was bilateral the worst the prognosis.
- The most feared outcomes are infertility and malignancy (high risk at ages 20,30,40).

Orchiopexy: Fixation of testis in scrotum, we place testis back to normal position to minimize cancer risk and to enhance the fertility!

If the testis is higher in the abdomen, we need to do a second surgery. The procedure is called the "Two Stages Fowler-Stephens Orchiopexy".

4 ACUTE SCROTUM

- Acute onset of pain in the scrotum
- Pediatric surgical emergency: It might lead to testicular loss

4.1 PRESENTATION

- Pain is the major feature; do not wait for swelling and redness.
- It may be associated with lower abdominal pain.
- It may also have an atypical presentation such as right flank pain
- They present with painful scrotum +/- swelling +/- redness.
- They present with sudden onset of scrotal pain that can progress to swelling and redness which means the testis is necrotic. Pt can have abdominal pain and N/V.
- Signs:
    - Tenderness of testis
    - High lying testis
    - Maybe lying in horizontal plane
    - Absent Cremasteric reflex (very specific)
- When the Hx and Ex suggest testicular torsion, the next step is emergent scrotal exploration. Imp!!
- That's because if we wait to do a Doppler ultrasound or nuclear scan we will waste valuable time. Instead, we should take the boy to the OR and do emergent scrotal exploration and untwist the testis.
- If it's the left testis > untwist clockwise (fix contralateral testis)
- If it's the the right testis > untwist counterclockwise

4.2 CAUSES

- Causes include:
    - Torsion of appendages (commonest)
    - Testicular Torsion
    - Idiopathic scrotal edema
    - Epididymo-orchitis
    - Other conditions e.g. Incarcerated hernia, acute hydrocele, HSP, trauma

4.2.1 TORSION OF APPENDAGES

- Embryological remnants of the mesonephric and mullerian duct system occur as tiny (2-10 mm long) appendages of testis
- Appendix testes (hydatid of Morgagni), appendix epididymitis, etc..
- Peak age: 10-12 years
- Presentation:
    - Pain at the upper part of the testis (more gradual onset), the rest of the testis is not tender
    - Blue dot sign (the most specific sign) and usually at the top of the testis
    - Swollen & red hemiscrotum appears on the 2nd day of onset of pain. So, in this case, they are an early presentation whereas in acute scrotum they present late.

- Management:
  - Conservative
  - Operative: If torsion cannot be excluded

4.2.2 TESTICULAR TORSION

- Incidence: 1:4000
- Two peaks: Perinatal and peripibertal
- Symptoms:
  - Lower abdominal pain and vomiting
  - Hemiscrotal pain
  - Swollen & red hemiscrotum
- Signs:
  - Tender
  - Absent Cremasteric reflex (most specific – 98%)
  - Lies higher than contralateral testis
  - Horizontal in position
- Investigations:
  - Color Doppler US
  - Radionuclide scan
  - High clinical suspicion of torsion needs no investigation but needs immediate intervention
- Management:
  - Timing is critical 4-6 hours (risk of ischemia)
  - Exploration if in doubt
  - Untwist and assess viability
  - Fix the other side
  - If more than 12 hours it is likely to be non-viable (gangrenous) and may need orchiectomy

4.2.3 IDIOPATHIC SCROTAL EDEMA

- Peak age: 4-5 years
- Presentation:
  - Swelling & redness in scrotum
  - Minimal pain
  - Usually bilateral
  - Samoan color is very pathognomonic
- Management
  - Conservative: Self-limiting within 1-2 days

5 MCQS

1. Regarding the scrotal swellings:
   a. Haemetocele is very common
   b. Hydrocele could be inguinoscrotal
   c. Solid epididymal swelling is usually tumor
   d. Transluminant testicular mass is a tumor

e. Usually examined with the patient lying down

2. The first symptoms of strangulated Inguinal Hernia is:
   a. Vomiting
   b. Fever
   c. Septic shock
   d. Constipation
   e. Pain
3. The following are important steps in the management of strangulated hernia except:
   a. Nasogastric tube
   b. Antibiotics
   c. Conservative treatment until obstruction is relieved
   d. Intravenous fluids
   e. Consent for possible bowel resection

🔑 Answers: 1:B, 2:E, 3:C

ACUTE ABDOMEN

1 INRODUCTION

1.1 DEFINITION

- Acute abdomen denotes any sudden onset, spontaneous non-traumatic disorder in the abdominal area that requires urgent surgery in some cases (most of them).

1.2 GENERAL APPROACH TO ACUTE ABDOMEN

- The standardized approach for all acute abdominal disorders is the (SOAP) approach:
  - **S**ubjective– History Taking
  - **O**bjective - Physical Examination
  - **A**ssessment – Investigations
  - **P**lan – Treatment (based on the final diagnosis)
- The approach is not that different from an elective case, except in patients who are hemodynamically unstable and will go into shock, resuscitation should be initiated first.
- Analgesia or painkillers are not preferable to be given until a diagnosis is made.

2 HISTORY AND EXAMINATION

2.1 HISTORY

2.1.1 AGE

- Newborn child presents with acute abdominal pain; most likely, it is a digestive disease (bowel atresia - congenital anomaly in which there is incomplete development of the intestinal tract, typically with closures and “dead ends” that block flow through the intestines. or meconium ileus - Obstruction of the intestine (ileus) due to overly thick meconium).
- Child who present with an acute abdominal pain, mesenteric adenitis is suspected.
- 12-year-old boy who present with an acute abdominal pain, appendicitis is suspected.
- Elderly patient with acute abdominal pain, obstruction due to cancer or acute diverticulitis is highly suspected.

***Mesenteric* adenitis** is general term for an inflammation of a gland or lymph node

2.1.2 PAIN

- **Site**
  - Site will give an idea about what is the organ involved:
  - Right upper quadrant → think about gall bladder or liver.
  - Right lower quadrant most likely it is appendicitis.
  - Left lower quadrant think about diverticulitis.
- **Onset**: Sudden or gradual
- **Character**

  - Dull “mild pain”
  - Trooping “in wounds”
  - Stabbing “ something in closed space like gallbladder and renal colic”
  - Compression ”MI”
  - Burning “gastritis”
  - Colicky in nature “bowel obstruction”
- **Radiation**
  - Cholecystitis to the tip of the right shoulder.
  - Pancreatitis to the back.
- **Timing** important to decide management
  - Examples:
    - Patient with pain in the right lower quadrant, most likely it is appendicitis, if the patient reported that the pain started last night, surgery is the likely choice of management
    - If the same patient reported that he/she had this pain 4-5 days ago and the pain is getting worse then you diagnose him/her with appendicular mass, the approach will be conservative rather than surgical.
- **Severity**
  - Pain scale from 1 to 10, 0 no pain \ 10 worst pain.
  - Mild pain (0-4), moderate (5-7), severe (8-10).
  - Acute abdomen is in the severe category.
- **Relieving** and **aggravating** factors
  - Fatty food elicits biliary colic.
  - Antacid for burning pain in the epigastrium, milk will temporarily relieve the pain but after an hour, pain will become worse (milk contain protein --> protein increase gastric acid secretions). Milk is a temporal buffer.
- **Progression**.
- **Associated symptoms**: Nausea and vomiting with severe pain.

2.1.3 VOMITING

- Hematemesis
- Volume : small or large amount
- Projectile "force" In children usually due to pyloric stenosis In newborn due to congenital hypertrophy of pylorus. In adults, gastric outlet obstruction
  - Causes of gastric outlet obstruction :
    - Scarring due to chronic peptic ulcer
    - Gastric cancer obstructs the pylorus
    - Superior mesenteric artery syndrome
    - In bezoar psychiatric patient who eats foreign bodies e.g. Hair forming a ball that obstructs the gastric
- Frequent or occasional
- Does vomiting relieve the pain or not:
  - Most of abdominal colic’s relieved by vomiting
- Content:
  - Undigested food
  - Digested food: greenish

Superior mesenteric artery syndrome is characterized by compression of the third or transverse portion of the duodenum between the aorta and the superior mesenteric artery. This results in chronic, intermittent, or acute complete or partial duodenal obstruction

2.1.4 DEFECATION:

- It is important to ask about the bowel habits.
- **Constipation** for 2 days with acute abdominal pain means there's an obstruction
    - Ask them can they pass gases or not, if not it's called **Obstipation** "complete bowel obstruction".
- Diarrhea with acute abdomen usually means infection; gastroenteritis usually does not cause acute abdominal pain unless bowel perforation happens.
    - Salmonella lead to typhoid fever and typhoid fever can cause gastroenteritis that lead to bowel perforation and acute abdominal pain.
- Acute abdominal pain with severe diarrhea "mixed with blood"
    - Ulcerative colitis
    - Bowel ischemia
    - Crohn's disease

2.1.5 FEVER

- Rigors with acute abdominal pain means Sepsis due to cholangitis

2.1.6 PAST HISTORY

- Similar episodes of UC or Crohn's disease but in less degree
- Past abdominal surgery adhesion, bowel obstruction, bowel strangulation or ischemia
- Bowel obstruction due to hernia
- Peptic ulcer perforation
- Gall stones Obstruction
    - Acute cholecystitis (is a sudden inflammation of the gallbladder that causes severe abdominal pain)
    - Pancreatitis
    - Ascending cholangitis

Peptic ulcer perforation is a hole in the wall often leads to catastrophic consequences. Erosion of the gastro-intestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain

2.2 EXAMINATION

1. **General look:**
    a. Lying on bed and they look ill and in pain, uncomfortable moving, because they want to obtain a position that relieves them from peritoneal irritation, sometimes they roll in bed in renal colic or sometimes in acute cholecystitis when gallbladder get contracted with stones
        i. Anything related to stone make patient roll in bed
        ii. Appendicitis dull aching pain that does not make patients roll in bed
2. **Vital signs:** Important to see the hemodynamic state of the patient wither if the patient is tachycardic, tachypenic or hypotensive, they must be treated immediately or they will go into shock.
3. **Head and neck**
    a. Check the eyes for jaundice. "jaundice+ fever+ abdominal pain to diagnose **cholangitis**"

b. JVP: in acute abdomen, patient will be hypovolemic hence the JVP will disappear
c. Mucus membrane: sings of dryness
d. Lymph node may present with lymphadenopathy

4. **Chest**
   a. Pleural effusion caused by pneumonia. In lower pneumonia or lobar pneumonia you'll hear crackles and bronchial breathing
5. **Abdomen**
   a. Inspection: distended, does not move with respiration because the peritoneum contracting the muscles of the abdomen, might see other signs (ex. In chronic liver disease...etc)
   b. Palpation: start superficial away from the site of pain.
   c. Percussion
      i. Dullness fluid ascites
      ii. Tympanic or tympanitic, drum-like sounds heard over air filled structures during the abdominal examination which suggest bowel obstruction
   d. Auscultation:
      i. Paralytic ileus because of infection, absence of bowel sounds.
      ii. Mechanical obstruction (bowel obstruction, UC, strangulation, condition in which circulation of blood to a part of the body is cut off by constriction, Enteritis) will lead to hyperactive bowel sounds.
6. **Rectal Examination**
   a. Trickling of exudates in the Douglas pouch
   b. Between the rectum & uterus in female
   c. Rectum & bladder in male
   d. Pressing interiorly to see if there is tenderness
   e. Look for blood & malena.
   f. Any mass specially in elderly
7. **Vaginal Examination**
   a. Ectopic pregnancy by moving the uterus “put your finger till you reach cervix then you move the cervix” but more commonly you inspect with speculum to check for pelvic inflammatory disease, it manifests by exudates\ pus ”vaginal discharge”
      i. Rule out salpingitis (infection and inflammation in the fallopian tubes).

3 INVESTIGATIONS

1. **Complete Blood Count:**
   a. High WBC "Leukocytosis" more than 40,000 is a suggestive of appendicitis
   b. Low hemoglobin indicates hemorrhage, UC, Ischemia, Ulcer, anemia.
   c. Platelet count, if the patient is thrombocytopenic because sometimes thrombocytopenia can happen due to severe sepsis also it is
   d. An indication of a problem that might prevent you from doing surgery or in splenomegaly.

Normal spleen stores red blood cells and platelets, the cells that help your blood clot, an enlarged spleen it begins to filter normal red blood cells as well as abnormal ones, reducing the number of healthy cells in your bloodstream. It also traps too many platelets. Eventually, excess red blood cells and platelets can clog your spleen, interfering with its normal functioning.

2. **Electrolytes, BUN, Creatinine**
    a. In acute abdomen, there will be loss of fluid in and electrolytes will decrease
    b. Hypokalemia from upper GI cause (In vomiting you expect low potassium)
    c. Hyponatremia from lower GI cause (diarrhea)
    d. BUN & Creatinine if elevated? In acute abdomen, hypovolemic pre-renal azotemia, insufficient profusion to the kidney that will lead to renal failure.
3. **Liver Function Tests**
    a. If you suspect jaundice, biliary disease and cholangitis.
    b. High bilirubin and high alkaline phosphatase are suggestive of cholangitis.
    c. High ALT and AST are suggestive of Hepatitis.
4. **Serum Amylase**
    a. It will be high in pancreatitis but it will go down after 2-3 days, so check lipase because it will persist high in pancreatitis.
5. **Lactate**:
    a. (Product of anaerobic metabolism): if there is bowel ischemia.
6. **Arterial blood gases [ABGs]**
    a. Reflex the respiratory and metabolic states.
    b. Do it if ischemia is suspected, severe sepsis, metabolic acidosis and before anesthesia.
7. **Chest x-ray**
    a. Perforation of hollow viscous (commonly duodenal ulcer perforation), see air under the diaphragm. Ask for upright chest x ray
8. **Abdominal X-Ray – KUB:**
    a. In bowel obstruction the abdomen will look distended in supine position.
    b. Other AXR is erect “upright” position to look for air fluid level, if more than 3 it mean there's significant obstruction
    c. In gastroenteritis you can see dilated loops of small or large bowel but not necessary to have obstruction.
    d. KUB- for renal stones.
9. **Abdominal Ultrasound**
    a. Mainly used to rule out stones (gall bladder or renal), ascites, pyelonephritis, polycystic ovarian disease.
10. **Abdominal CT**
    a. To diagnose difficult echo vocal appendicitis (diagnosis of appendicitis is commonly clinical), rule out pancreatitis, tumors and bowel ischemia.
    b. Angiography / Duplex Scanning:
    c. If we suspect mesenteric ischemia so we can see the blood vessels causes of ischemia (thrombus, embolus)
    d. We usually do CT and angiography
    e. CT to see the bowel
11. **Angiography** to see blood vessels
    a. If they match no blood in the vessel and bowel is edematous this is gangrene.

b. Duplex: for peripheral Blood vessels.

4 DIAGNOSIS

- Acute Abdomen + Shock – Acute Pancreatitis/ Ruptured AAA (abdominal aortic aneurysm) resuscitate & immediate surgery otherwise patient may die in minutes.
- Generalized Peritonitis – Ruptured Viscus.
- Localized Peritonitis,
  - Example: RLQ rebound tenderness means Acute Appendicitis.
- Bowel Obstruction (distention of the abdomen no movement during respiration)
- Medical Causes [Lobar Pneumonia, Acute Inferior MI "if the patient have epigastric pain and you think of MI you can rule it out by doing ECG or Cardiac enzyme (troponin)"]

5 MANAGEMENT

- **Immediate operation** – Ruptured AAA
  - (Amount of bleeding is huge so if you don't stop it immediately patient will die, do surgery immediately and stop it)
- **Pre-operative** preparation and urgent operation within 6 hours
  - Because the condition can get worse if you operate immediately (ruptured Viscus but preoperatively is hypotensive dehydrated, has electrolyte abnormalities , quite septic , if you take him immediately to operation he might die, to prevent mortality in such condition resuscitate the patient and prepare them for surgery by giving fluids, antibiotics (they do it in ICU usually).
- **Urgent operation** within 24 hours
  - Especially in case of acute appendicitis
- **Conservative treatment**
  - In acute (pancreatitis operation will worsen the condition - except when there is pancreatic abscess or necrosis we operate on them)
  - IBD
  - Cholecystitis
- **Observation**
  - Patients with sudden onset acute abdominal pain, tender on examination but the diagnosis was not established yet. You should observe them (check on them every 2-4 hours tell next day if they have a disease it will manifest).
  - E.g. early appendicitis, after 24 hours will be obvious
  - If there is a follicle somewhere or ruptured Graafian follicle in the ovary, next day they feel better then you can discharge the patient at this step.
- **Discharge**

6 SCENARIOS &SUMMARY

6.1 SCENARIOS: (USE THE MNEMONIC SOAP)

Case 1:

A 35 year-old male presented to the ER with 2 days history of abdominal pain. He took antacids but did not help him at all!

Case 2:

A 55 year-old businessman presented to the ER with severe abdominal pain since 6 hours when he felt something like a burst in his abdomen. He is known with PUD and H-pylori but he was not taking his medications regularly

Case 3:

A 73 year-old male developed atrial fibrillation while recovering from an acute MI in the medical ward. The surgery team was consulted to evaluate a new onset of severe mid-abdominal pain

Case 4:

A 54 year-old lady presented to the ER complaining of generalized abdominal pain associated with vomiting, constipation for 2 days, and abdominal distention. She had an emergency Cesarean Section for her 5th baby 5 years back

6.2 SUMMARY

- Acute abdomen is a sudden abdominal disorder that requires an urgent operative intervention in some cases.
- Almost all acute abdominal events have a common general surgical approach based on the mnemonic SOAP.
- We have applied this general approach to some case scenarios such as acute appendicitis, perforated DU, acute mesenteric ischemia, and small bowel obstruction.

ESOPHAGEAL DISEASES

1 ACHALASIA

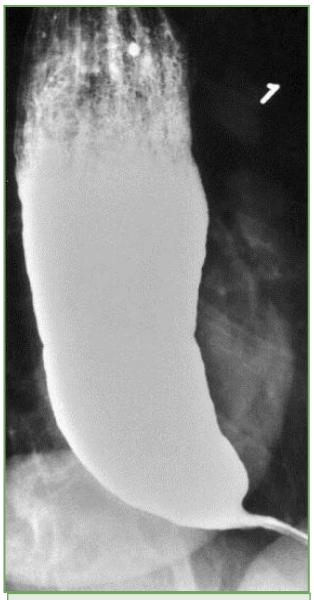
- Uncommon disease of esophageal motility disorder.
- Characterized by degeneration of the myenteric neurons that innervate LES and esophageal body.
- Pathogenesis: autoimmune, familial, viral.

1.1 PRESENTATION

- Most commonly presents in patients between the ages of 25 and 60 years.
- Equal male-to-female gender distribution.
- Symptoms:
  a. **Dysphagia** to solids and liquids is the (most common; 90% of patients).
  b. **Regurgitation** (2nd most common; 60% of patients).
     - Nocturnal regurgitation of esophageal contents can lead to night-time cough and aspiration.
  c. ***Weight loss*** *occurs in end-stage disease.*
  d. ***Chest pain*** *(20% to 60% of patients).*
  e. ***Heartburn*** *(30% of achalasia patients).*
     - *May be related to direct irritation of the esophageal lining by retained food, pills, or acidic by-products of bacterial metabolism of retained food.*

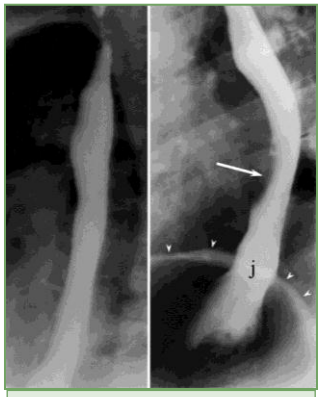
1.2 DIAGNOSIS

- CXR: may show air-fluid level.
- Barium study: quite dilated, and an air-fluid level may be secondary to retained secretions.
  - The classic finding is a gradual tapering at the end of the esophagus, similar to a bird's beak (rat tail).
- Upper endoscopy is the next diagnostic test in a patient with dysphagia or suspected achalasia (to rule out tumors).
  - Findings can include:
    - Dilated esophagus with retained food or secretions.
    - Normal in as many as 44% of patients with achalasia.
  - Difficulty traversing the GEJ should raise suspicion for pseudoachalasia due to neoplastic infiltration of the distal esophagus.
- Esophageal manometry (highest sensitivity for the diagnosis of achalasia):
  - Aperistalsis of the distal esophageal body.
  - Incomplete or absent LES relaxation (Hypertensive LES).
  - Manometric variants of achalasia exist.



"Bird's beak" or "rat's tail" on barium study

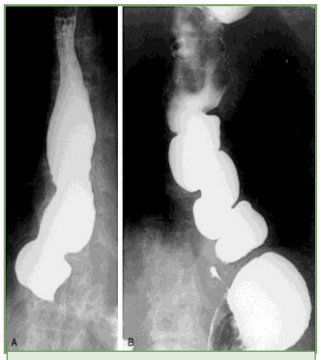
- The best known is vigorous achalasia –may represent an early stage-, defined by the presence of normal to high amplitude esophageal body contractions in the presence of a non-relaxing LES.



Barium study

1.2.1 SECONDARY ACHALASIA

- Chagas' disease is a parasitic infection caused by *Trypanosoma cruzi which can cause secondary achalasia.*
- The most concerning secondary etiology is cancer, which can present as achalasia through mechanical obstruction of the GEJ.
- Post fundoplication (surgery done for treatment of GERD patients): achalasia caused by *mechanical obstruction* of the GEJ by the fundoplication or diaphragmatic crural closure.
- Bariatric surgery using a gastric band device which constricts the proximal stomach a few centimeters below the LES.
- Some types of surgery could cause achalasia by reducing the LES diameter.



Dilation and tapering on barium study

1.3 TREATMENT

- The primary therapeutic goal in achalasia is to reduce the LES basal pressure.
- Treatment options include
  - Medical therapy: Nifedipine, Isosorbide dinitrate.
  - Botulinum toxin injection: block the release of acetylcholine; limited value.
  - Pneumatic dilation: blindly rupture the muscle fibers while leaving the mucosa intact.
  - Surgical myotomy (+ partial fundoplication).
- Symptomatic relief, particularly relief of dysphagia, is accepted as the primary desired outcome.

1.3.1 MEDICAL THERAPY

Inconvenient, only modestly effective, and frequently associated with side effects.

It is reserved for patients who are awaiting or unable to tolerate more invasive treatment modalities.

Pharmacologic therapies attempt to decrease the LES pressure by causing smooth muscle relaxation.

- Nitrates were first recognized as an effective treatment of achalasia.
  - Systemic vasodilatory effects and headaches limit their tolerability by patients.

- Calcium channel antagonists have a better side-effect profile when compared with nitrates.
    - 30% of patients report adverse side effects including peripheral edema, hypotension, and headache.

1.3.2 BOTULINUM INJECTION

- Injected into the LES targets the excitatory, acetylcholine-releasing neurons that generate LES basal muscle tone.
- Easy to administer and associated with relatively few side effects.

It is apparent that, with repeated injections, the response rates reported are similar or lower to that achieved with the initial injection; not very effective.

- Response rates at 1 month following administration average 78%, By 6 months, the clinical response rate drops to 58% and by 12 months to 49%

Given the limitations of the efficacy and durability of response, botulinum toxin is generally reserved for use in patients who are not candidates for more invasive treatments.

1.3.3 PNEUMATIC DILATATION

- Pneumatic dilation remains one of the most effective first-line therapies for achalasia.
- Long-term follow-up studies reported **significant symptom relapse of 50% at 10 years.**
- Complications of pneumatic dilation:
    - Gastroesophageal reflux 25-35%.
    - Esophageal perforation 3 %.

1.3.4 SURGERY

- **Standard management**
- Success rates >90% - with hospital stays averaging only a few days.
- Acid exposure is a known complication of surgical intervention for achalasia (reflux esophagitis).
- Even with a successful myotomy, it is expected that patients will have some degree of dysphagia as a consequence of esophageal peristaltic dysfunction.
- **Delayed recurrence** of postoperative dysphagia is most commonly caused by development of a recurrent high pressure zone at the LES or a peptic stricture complicating acid reflux.
- Laparoscopic Heller myotomy demonstrated excellent results, with 98% of patients reporting symptomatic improvement at 5.3 years.
- Several retrospective and prospective studies have reported superior success rates for surgery when compared with pneumatic dilation

1.3.5 REFRACTORY ACHALASIA

- In patients with achalasia that is refractory to therapy with Heller myotomy, options are limited.
- Although esophagectomy is considered in patients with marked dilation and sigmoid deformity, such patients may respond to Heller myotomy.

1.4 COMPLICATIONS

- The primary complications of achalasia are related to the functional obstruction rendered by the non-relaxing LES and include **progressive malnutrition and aspiration.**
- Uncommon but important secondary complications of achalasia include the formation of **epiphrenic diverticula and esophageal cancer.**
- There is an established link between achalasia and esophageal cancer, most commonly **SQUAMOUS CELL CARCINOMA.**
  - The overall prevalence of esophageal cancer in achalasia is approximately 3% with an incidence of approximately 197 cases per 100,000 persons per year.

2 ESOPHAGEAL DIVERTICULA

- **Causes:** Most diverticula are a result of a primary motor disturbance or an abnormality of the UES (upper esophageal sphincter) or LES (lower esophageal sphincter).
- **Site:** Can occur in several places along the esophagus. The three most common sites of occurrence are:
  - Pharyngoesophageal (Zenker's).
  - Parabronchial (mid-esophageal) .
  - Epiphrenic.
- **True vs. False:**
  - True diverticula involve all layers of the esophageal wall, including mucosa, submucosa, and muscularis.
    - Traction, or true, diverticula result from external inflammatory mediastinal lymph nodes adhering to the esophagus.
  - A false diverticulum consists of mucosa and submucosa only.
    - Pulsion diverticula are false diverticula that occur because of elevated intraluminal pressures generated from abnormal motility disorders.
    - Zenker's diverticulum and an epiphrenic diverticulum fall under the category of false, pulsion diverticula.

2.1 PHARYNGOESOPHAGEAL (ZENKER'S) DIVERTICULUM

- Most common esophageal diverticulum found today.
- It usually presents in older patients in the 7th decade of life.
- Found herniating into Killian's triangle, between the oblique fibers of the thyropharyngeus muscle and the horizontal fibers of the cricopharyngeus muscle.

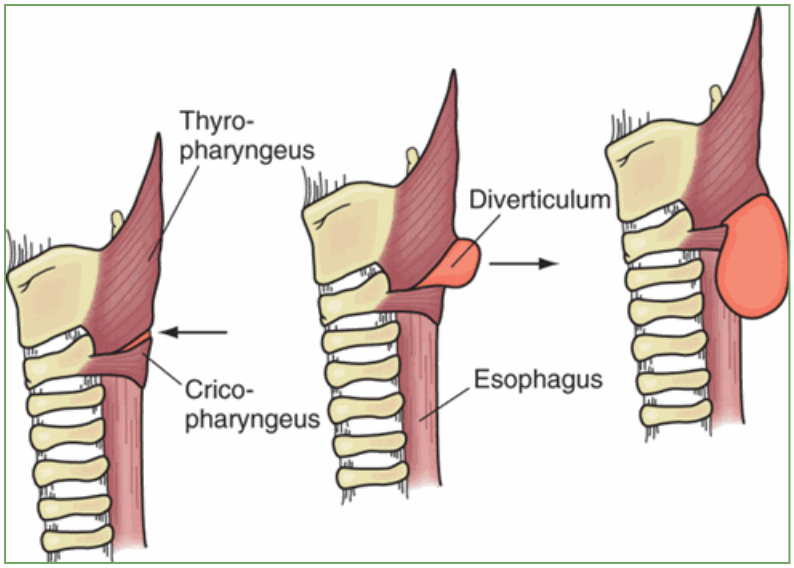
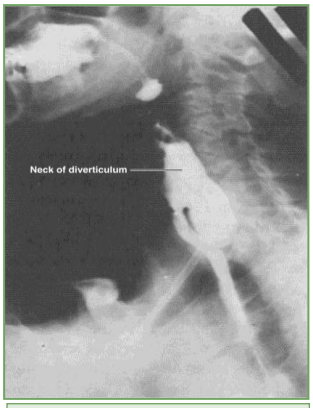


Figure 1: Killian's Triangle



Barium esophagaram

- **Symptoms**:
  - Sticking in the throat (common).
  - Nagging cough .
  - Excessive salivation.
  - Intermittent dysphagia .

  } *Signs of progressive disease*

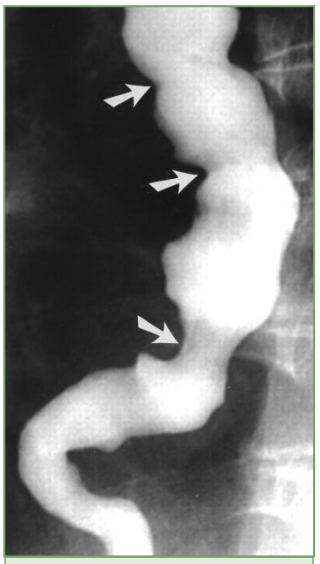
  - Regurgitation of foul-smelling, undigested material (common as the sac increases in size, because of fermentation of food).
  - Halitosis (bad mouth smell).
  - Voice changes.
  - Retrosternal pain.
  - Respiratory infections .

  } *Especially common in elderly*
- **Complications:** the most serious complication from an untreated Zenker's diverticulum is aspiration pneumonia or lung abscess.
- **Diagnosis:** is made by barium esophagram **ONLY**
  - Neither esophageal manometry nor endoscopy is needed to make a diagnosis of Zenker's diverticulum.
- **Treatment:**
  - Surgical or endoscopic repair of a Zenker's diverticulum is the gold standard of treatment.
  - Open repair involve:
    - Myotomy of the proximal and distal thyropharyngeus and cricopharyngeus muscles.
    - Diverticulectomy or diverticulopexy are performed through an incision in the left neck.
  - An alternative to open surgical repair is the endoscopic Dohlman procedure.
  - Endoscopic division of the common wall between the esophagus and the diverticulum using a laser or stapler has also been successful.

3 DIFFUSE ESOPHAGEAL SPASM

- **Pathology**:

- The esophageal contractions are repetitive, simultaneous, and of high amplitude.
- DES is a hypermotility disorder of the esophagus (non-peristalsis disorder).
- The basic pathology is related to a motor abnormality of the esophageal body that is most notable in the lower two thirds of the esophagus.



Corkscrew appearance

- **Clinical presentation:**
  - Is seen most often in women and is often found in patients with multiple complaints.
  - **Typically**: chest pain and dysphagia
    - Patients will complain of a squeezing pressure in the chest that may radiate to the jaw, arms, and upper back (could be misdiagnosed as angina pectoris).
    - May be related to: eating or exertion.
    - Aggravating: heightened emotional stress, acid reflux, cold liquids.
  - **Regurgitation** of esophageal contents and saliva is common (but NOT acid reflux).
  - **Associated with:** Irritable bowel syndrome (IBS) and pyloric spasm.
  - **Triggers:** GI problems e.g. gallstones, peptic ulcer disease (PUD), and pancreatitis.
- **Diagnosis:** esophagram and manometric studies.
- **Treatment:**
  - The mainstay of treatment for DES is **NON-SURGICAL**, and pharmacologic or endoscopic intervention is preferred.
  - Surgery is reserved for patients with recurrent incapacitating episodes of dysphagia and chest pain who do not respond to medical treatment.

4 CAUSTIC INJURY

The best cure for this condition is an ounce of prevention

- In children: ingestion is accidental and in small quantities.
- In teenagers and adults: ingestion usually is deliberate during suicide attempts, and in much larger quantities.
- Alkali ingestion is more common than acid ingestion because of its lack of immediate symptoms.
- Alkali ingestion is more devastating and almost always lead to significant destruction of the esophagus.

Three Phases Of Tissue Injury From Alkali Ingestion				
Phase Of Tissue Injury		**Onset**	**Duration**	**Inflammatory Response**
1	Acute necrosis	1-4 days	1-4 days	Coagulation of intracellular proteins
				Inflammation
2	Ulceration and granulation	3-5 days	3-12 days	Tissue sloughing
				Granulation of ulcerated tissue bed
3	Cicatrization and scarring	3 weeks	1-6 mo	Adhesion formation
				Scarring

- **Symptoms:**

Phase I	Patients may complain of oral and substernal pain, hypersalivation, odynophagia and dysphagia, hematemesis, and vomiting.
Phase II	These symptoms may disappear.
Phase III	Dysphagia reappears as fibrosis and scarring begin to narrow the esophagus.

  - Symptoms of respiratory distress, such as hoarseness, stridor, and dyspnea, suggest upper airway edema and are usually worse with acid ingestion.
  - Pain in the back and chest may indicate a perforation of the mediastinal esophagus, whereas abdominal pain may indicate abdominal visceral perforation.
- **Diagnosis:**
  1) Physical exam:
     a. Specifically evaluating the mouth, airway, chest, and abdomen.
     b. Careful inspection of the lips, palate, pharynx, and larynx is done.
     c. The abdomen is examined for signs of perforation.
  2) Early endoscopy is recommended 12 to 24 hours after ingestion to identify the grade of the burn.
  3) Serial chest and abdominal radiographs are indicated to follow patients with questionable chest and abdominal exams.

Degree of Burn	Endoscopic Evaluation	Treatment
First Degree	Mucosal hyperemia	48-hr observation
	Edema	Acid suppression
Second Degree	Limited hemorrhage	Aggressive IV resuscitation
	Exudates	IV antibiotics
	Ulceration	Acid suppression
	Pseudomembrane formation	
Third Degree	Mucosal sloughing	Inhaled steroids
	Deep ulceration	Fiberoptic intubation (if needed)
	Massive hemorrhage	
	Complete luminal obstruction	
	Charring	
	Perforation	

- **Management** (of the acute phase):
  - Goal: limiting and identifying the extent of the injury.
  - It begins with neutralization of the ingested substance.
  - Alkalis (including lye) are neutralized with half-strength vinegar or citrus juice (we give them an acids to compensate alkaline solution)
  - Acids are neutralized with milk, egg whites, or antacids.

- Emetics and sodium bicarbonate need to be avoided because they can increase the chance of perforation.

First degree burns	Second and third degree burns
• 48 hours of observation is indicated. • Oral nutrition can be resumed when a patient can painlessly swallow saliva. • A repeat endoscopy and barium esophagram are done in follow-up at intervals of 1, 2, and 8 months.	• Resuscitation is aggressively pursued. • The patient is monitored in the intensive care unit. • Kept (NPO) with IV fluids. IV antibiotics and a proton pump inhibitor are started. • Fiberoptic intubation may be needed and must be available.

5 ESOPHAGEAL PERFORATION

- Perforation of the esophagus is a surgical emergency.
- Early detection and surgical repair within the first 24 hours results in 80% to 90% survival.
- After 24 hours, survival decreases to less than 50%.
- **Causes:**
  - Forceful vomiting “Boerhaave's syndrome” (15%) .
  - Foreign body ingestion (14%) .
  - Trauma (10%).
  - Endoscopic instrumentation for a diagnostic or therapeutic procedure (Majority).

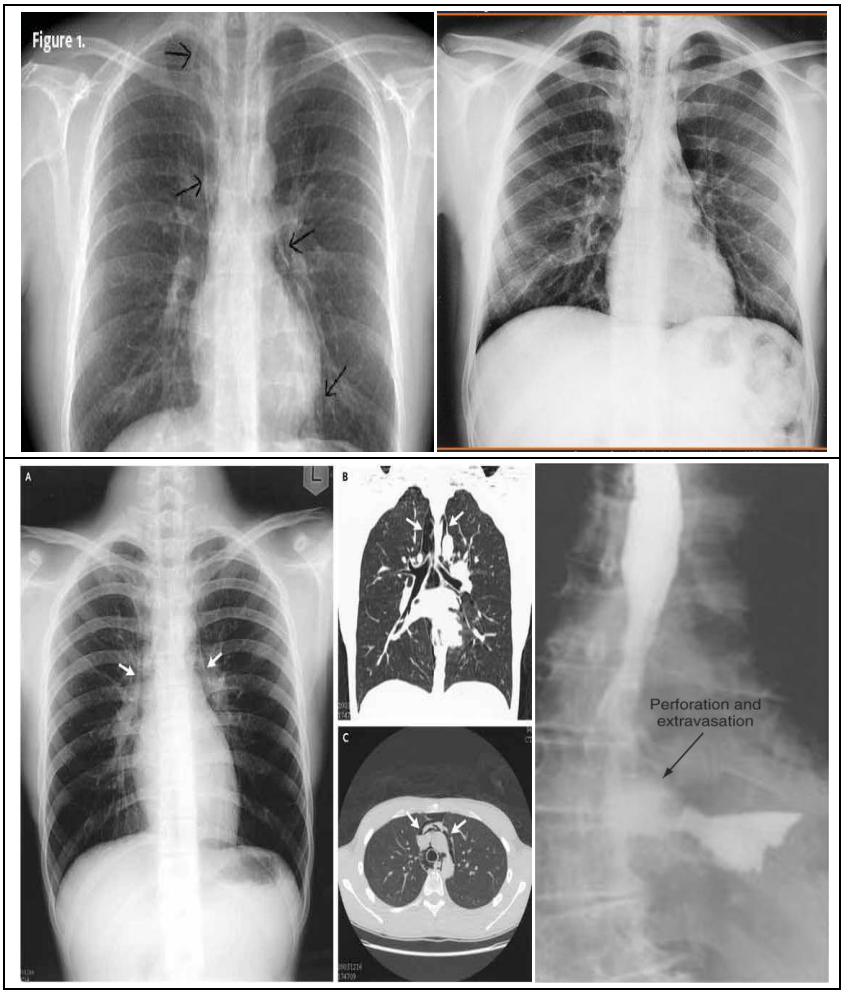
5.1 SIGNS AND SYMPTOMS

- Patients with esophageal perforation mainly come with **fever** & **dysphagia.**
  - Severe dysphagia, the patient is unable to swallow his saliva.
- Pain: neck, substernal, or epigastriac.
- +/- Vomiting or hematemesis.
- **Cervical perforations**:
  - May present with neck ache and stiffness due to contamination of the prevertebral space.
  - Could cause **subcutaneous emphysema** .
- **Thoracic perforations:**
  - Present with shortness of breath and retrosternal chest pain lateralizing to the side of perforation.
  - Could cause **pneumothorax.**
- **Abdominal perforations:** present with epigastric pain that radiates to the back if the perforation is posterior .
- **History**: trauma, advanced esophageal cancer, violent wretching (as seen in Boerhaave's syndrome), swallowing of a foreign body, or recent instrumentation.
- **On examination:**
  - Patient may present with tachypnea, tachycardia, and a low-grade fever but have no other overt signs of perforation.
  - Subcutaneous air in the neck or chest, shallow decreased breath sounds, or a tender abdomen are all suggestive of perforation.

- With increased mediastinal and pleural contamination, patients progress toward hemodynamic instability (shock).

5.2 DIAGNOSIS

- Lab: ↑ **WBC** count and ↑ **salivary amylase** in the blood or pleural fluid.
- Chest x-ray: may demonstrate a hydropneumothorax.
- Contrast esophagram: done using **barium** for a suspected thoracic perforation and **Gastrografin** is used for an abdominal perforation.
  - Most perforations are found above the GEJ on the left lateral wall of the esophagus which results in a 10% false-negative rate in the contrast esophagram if the patient is not placed in the lateral decubitus position.
- Chest CT: shows mediastinal air and fluid at the site of perforation.



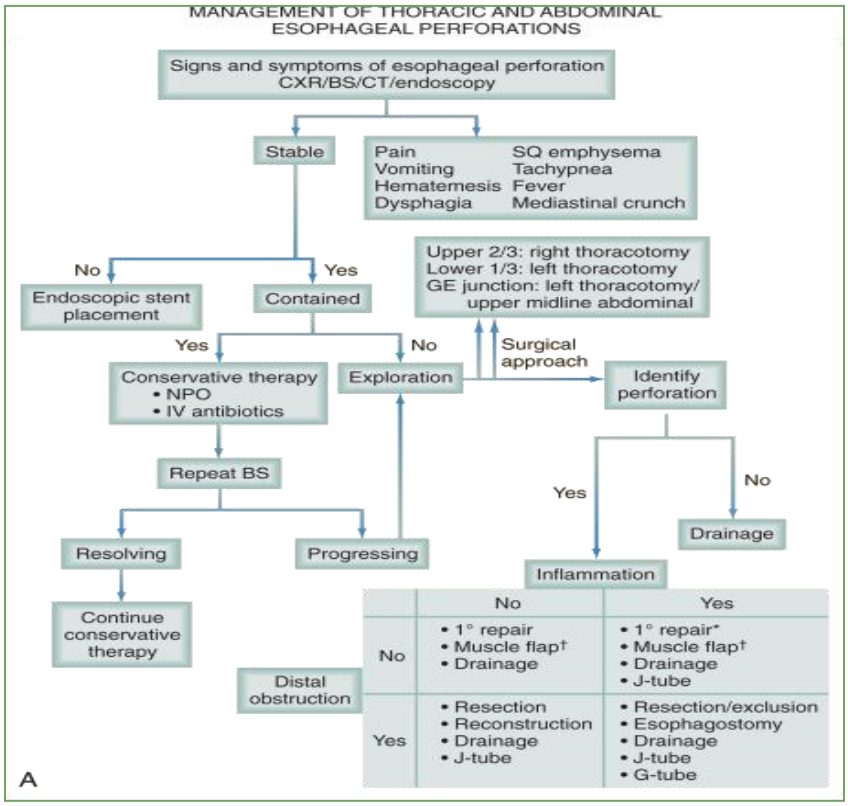
5.3 TREATMENT

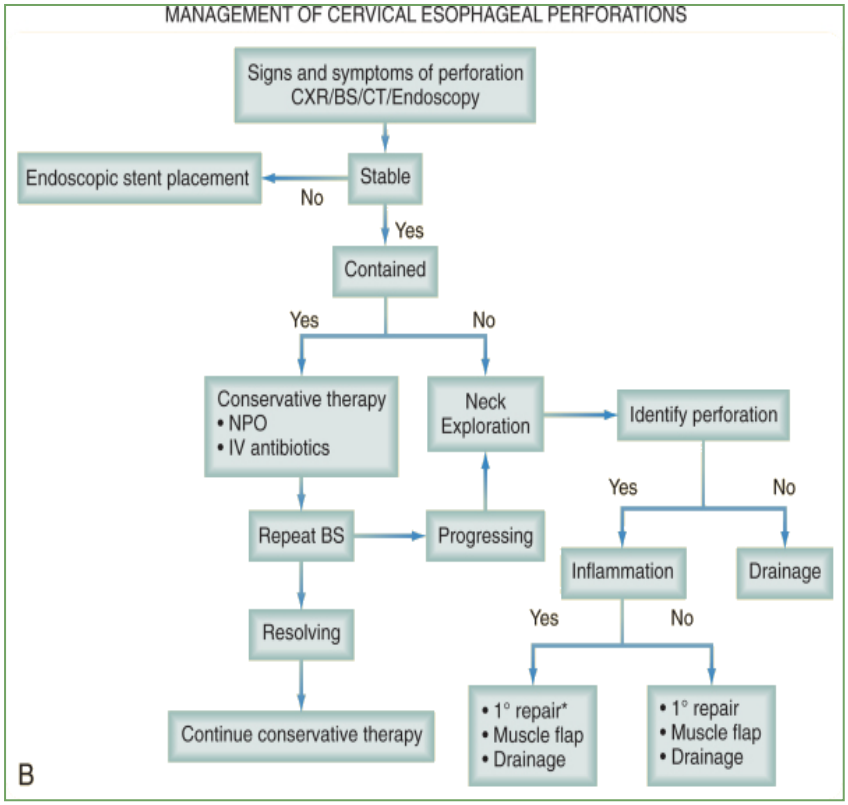
- A surgical endoscopy needs to be performed if the esophagram is negative or if operative intervention is planned.
  - Mucosal injury is suggested if blood, mucosal hematoma, or a flap is seen or if the esophagus is difficult to insufflate.

- Patients with an esophageal perforation can progress rapidly to hemodynamic instability and shock.
- If perforation is suspected:
  - Resuscitation with placement of large-bore peripheral IV catheters, a urinary catheter, and a secure airway, before the patient is sent for diagnostic testing.
  - IV fluids and broad-spectrum antibiotics are started immediately, and the patient is monitored in an ICU.
  - The patient is kept NPO, and nutritional access needs are assessed.

Surgery is not indicated for every patient with a perforation of the esophagus

- Management is dependent on several variables: stability of the patient, extent of contamination, degree of inflammation, underlying esophageal disease, and location of perforation.
- **The most critical variable** that determines the surgical management of an esophageal perforation is the **degree of inflammation** surrounding the perforation.
  - When patients present within 24 hours of perforation, inflammation is generally minimal, and primary surgical repair is recommended.
  - With time, inflammation progresses, and tissues become friable and may not be amenable to primary repair.
- The final variable to consider in the surgical management of esophageal perforations is the **location** of the perforation.

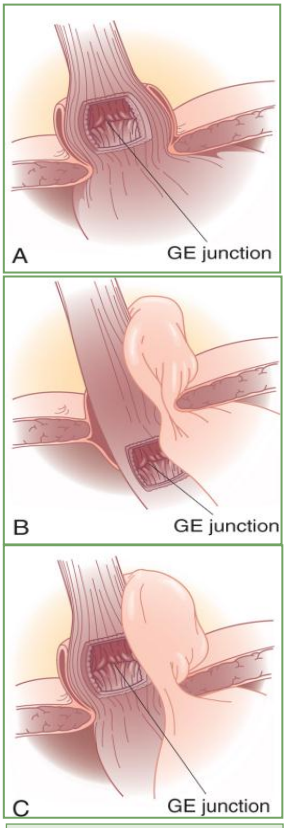




6 GASTROESOPHAGEAL REFLUX DISEASE

- **Definition:**
  - Symptoms OR mucosal damage produced by the abnormal reflux of gastric contents into the esophagus.
  - Often chronic and relapsing.
  - May see complications of GERD in patients who lack typical symptoms.
- **Pathology:**
  - LES has the primary role of preventing reflux of the gastric contents into the esophagus.
  - GERD may occur when the pressure of the high-pressure zone in the distal esophagus is too low to prevent gastric contents from entering the esophagus (when the LES is NOT contracting well).
  - GERD is often associated with a hiatal hernia:

Type I	The most common. Also called "sliding" hernia. Gastroesophageal junction is above the diaphragm.
Type II	Referred to as paraesophageal hernias. May be associated with GERD. GE junction is normal in position BUT part of the stomach herniated above the diaphragm.
Type III	Referred to as paraesophageal hernias. May be associated with GERD. GE junction is above the diaphragm and part of stomach too.
Type IV	Another organ is herniated into the chest e.g. spleen, colon.



Types of hiatus hernia

- **Epidemiology:**
  - About 44% of the US adult population have heartburn at least once a month.
  - 14% of Americans have symptoms weekly.
  - 7% have symptoms daily.

6.1 SYMPTOMS

Classic GERD	Complicated GERD
Substernal burning and or regurgitation	Dysphagia
Postprandial	Odynophagia: Retrosternal pain w/swallowing
Aggravated by change of position	Bleeding
Prompt relief by antacid	
Extra-esophageal	
Pulmonary	**ENT**
Asthma	Hoarseness (dysphonia)
Aspiration pneumonia	Laryngitis
Chronic bronchitis	Pharyngitis
Pulmonary fibrosis	Chronic cough
Other	Sinusitis
Chest pain	Subglottic stenosis
Dental erosion	Laryngeal cancer

6.2 DIAGNOSIS & TREATMENT

6.2.1 DIAGNOSIS

- Barium swallow (to confirm the diagnosis).
- Endoscopy (important to see the complication of GERD).
- Ambulatory pH monitoring (the gold standard and most accurate).
- Esophageal manometry.
- Bravo capsule is a capsule that receive the PH massages for 24 hours.

6.2.2 TREATMENT

- **Lifestyle Modifications (most important)**
  - Elevate head of bed 4-6 inches.
  - Avoid eating within 2-3 hours of bedtime.
  - Lose weight if overweight.
  - Stop smoking.
  - Modify diet:
    - Eat more frequent but smaller meals.
    - Avoid fatty/fried food, peppermint, chocolate, alcohol, carbonated beverages, coffee and tea.
  - OTC medications prn (as needed).
- **Acid Suppression Therapy**
  - H2-Receptor Antagonists (H2RAs)
    - Cimetidine (Tagamet®), Ranitidine (Zantac®), Famotidine (Pepcid®), Nizatidine (Axid®).
  - Proton Pump Inhibitors (PPIs)

        - Omeprazole (Prilosec®), Lansoprazole (Prevacid®), Rabeprazole (Aciphex®), Pantoprazole (Protonix®), Esomeprazole (Nexium ®)
- **Anti-Reflux Surgery:** Indications:
    - Failed medical management.
    - Patient's choice (opt for surgery despite successful medical management, due to life style considerations including age, time or expense of medications, etc).
    - Complications of GERD (e.g. Barrett's esophagus; grade III or IV esophagitis).
    - Medical complications attributable to a large hiatal hernia (e.g. bleeding, dysphagia).
    - "Atypical" symptoms (asthma, hoarseness, cough, chest pain, aspiration) and reflux documented on 24 hour pH monitoring.
- **Endoscopic Antireflux Therapies:**
    - Radiofrequency energy delivered to the LES
        - Stretta procedure.
    - Suture ligation of the cardia
        - Endoscopic plication.
    - Submucosal implantation of inert material in the region of the lower esophageal sphincter
        - Enteryx.

Summary of GERD:

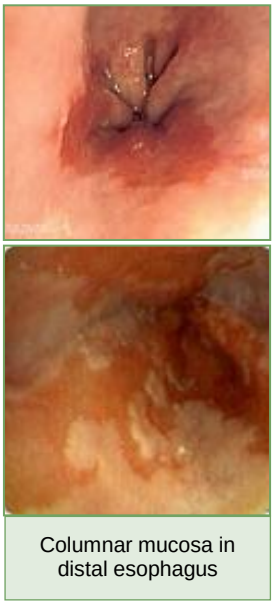
- It is due to low LES pressure that allows the reflux of gastric acids into the esophagus.
- Symptoms of GERD: 1) sore throat (2) epigastric pain (3) sub-sternal burning (4) hoarseness
    - Mainly occur post-prandial and with change of position.
    - Relieved by anti-acids.
- Diagnosis:
    - Barium swallow to confirm the diagnosis.
    - Endoscopy for complications.
    - PH monitor is the most accurate.
- Chronic GERD mainly followed by Barrett's esophagus which is a pre-malignant sign.

7 BARRETT'S ESOPHAGUS

- **Definition & pathology**:
    - Barrett's esophagus is a condition in which intestinal, columnar epithelium replaces the stratified squamous epithelium that normally lines the distal esophagus.
    - Chronic gastroesophageal reflux is the factor that both injures the squamous epithelium and promotes repair through **columnar metaplasia.**
    - Ten percent **(10%) of patients** with GERD develop Barrett's esophagus.
    - Although these metaplastic cells may be more resistant to injury from reflux, they also are more prone to **malignancy** (Barrett's esophagus is pre-malignant sign ⓘ) .
    - With continued exposure to the reflux disease, metaplastic cells undergo cellular transformation to low- and high-grade dysplasia
    - Dysplastic cells may evolve to cancer:

ⓘ Barrett's esophagus predisposes to **adenocarcinoma** NOT squamous cell carcinoma of the esophagus.

        - Low grade dysplasia only affecting mucosa and has a risk of cancer.
        - High grade dysplasia the patient for sure has carcinoma in situ.
    - **40-fold** increase in risk for developing esophageal carcinoma in patients with Barrett's esophagus.
- **Epidemiology**:
    - 70% of patients are **men** aged 55 to 63 years.
    - Men have a 15-fold increased incidence over women of adenocarcinoma of the esophagus, but women with Barrett's esophagus are increasing in number as the differences in the Western lifestyle between men and women diminish.
- **Clinical presentation:**
    - Many are asymptomatic.
    - Most patients present with symptoms of GERD: heartburn, regurgitation, acid or bitter taste in the mouth, excessive belching, and indigestion.
    - Recurrent respiratory infections, adult asthma, and infections in the head and neck also are common complaints.
- **Diagnosis**:
    - The diagnosis of BE is made by endoscopy and pathology. ⓘ
    - The presence of any endoscopically visible segment of columnar mucosa within the esophagus that on pathology identifies intestinal metaplasia defines BE.
- **Treatment**:
    - Surveillance:
        - **Yearly** surveillance endoscopy is recommended in all patients with a diagnosis of Barrett's esophagus.
        - For patients with low-grade dysplasia, surveillance endoscopy is performed at 6-month intervals for the first year and then yearly thereafter if there has been no change.
        - Patients undergoing surveillance are placed on acid suppression medication and monitored for changes in their reflux symptoms.
    - Anti-reflux surgery (controversial):
        - Those in favor of surgery argue that medical therapy and endoscopic surveillance may treat the symptoms but fail to address the problem.
        - The problem is the functional impairment of the LES that leads to chronic reflux and metaplastic transformation of the lower esophageal mucosa.
        - Surgery renders the LES competent and restores the barrier to reflux.
        - Studies have demonstrated regression of metaplasia to normal mucosa up to 57% of the time in patients who have undergone anti-reflux surgery.
    - Photodynamic therapy (PDT) is the **most common** ablative method used to treat BE.
    - Endoscopic mucosal resection (EMR) is gaining favor for the treatment of Barrett's esophagus with low-grade dysplasia.
    - Esophageal resection for Barrett's esophagus is recommended **only** for patients in whom high-grade dysplasia is found.

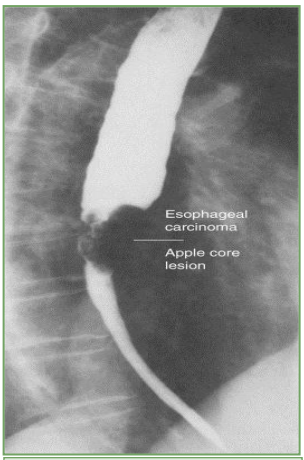


Columnar mucosa in distal esophagus

Pathologic data on surgical specimens demonstrate a 40% risk for adenocarcinoma within a focus of high-grade dysplasia.

8 CARCINOMA OF THE ESOPHAGUS

- Esophageal cancer is the fastest growing cancer in the western countries.
- **Squamous cell carcinoma** still accounts for most esophageal cancers diagnosed.
  - Arises from the squamous mucosa (native to esophagus), found in upper and middle third 70% of the time.
- In the US: **adenocarcinoma** in 70% of patients presenting with esophageal cancer.
  - Esophageal adenocarcinoma now 70% of all esophageal carcinomas diagnosed in Western countries. There are a number of factors that are responsible for this shift in cell type:
    - Increasing incidence of GERD.
    - Western diet.
    - Increased use of acid-suppression medications.
    - Intake of caffeine, fats, and acidic and spicy foods all lead to decreased tone in the LES and an increase in reflux.
- The **5-year survival rate**: 70% with polypoid lesions and 15% with advanced tumors.
- **Risk factors:**
  - Smoking and alcohol both increase the risk for foregut cancers by 5-fold (combined).
  - Food additives: nitrosamines (pickled and smoked foods).
  - Long-term ingestion of hot liquids.
  - Caustic ingestion, achalasia, bulimia, tylosis (an inherited autosomal dominant trait), Plummer-Vinson syndrome, external-beam radiation, and esophageal diverticula all have known associations with **squamous cell cancer**.
- **Symptoms**:
  - Early-stage cancers: asymptomatic or mimic symptoms of GERD.
  - Most patients with esophageal cancer present with **dysphagia and weight loss.**
    - Because of the distensibility of the esophagus, a mass can obstruct two thirds of the lumen before symptoms of dysphagia are noted.
  - Choking, coughing, and aspiration from a tracheoesophageal fistula (signs of advanced disease).
  - Hoarseness and vocal cord paralysis from direct invasion into the recurrent laryngeal nerve (signs of advanced disease).
  - Systemic metastases to liver, bone, and lung can present with jaundice, excessive pain, and respiratory symptoms.
- **Diagnosis**:
  1. Esophagram:
     - A barium esophagram is recommended for any patient presenting with dysphagia.
     - differentiate intraluminal from intramural lesions.



Barium esophagram

- discriminate between intrinsic (from a mass protruding into the lumen) and extrinsic (from compression of a structures outside the esophagus).
- The classic finding of an apple-core lesion.
- Not specific for cancer, but good first test to perform in patients presenting with dysphagia and a suspicion of esophageal cancer.

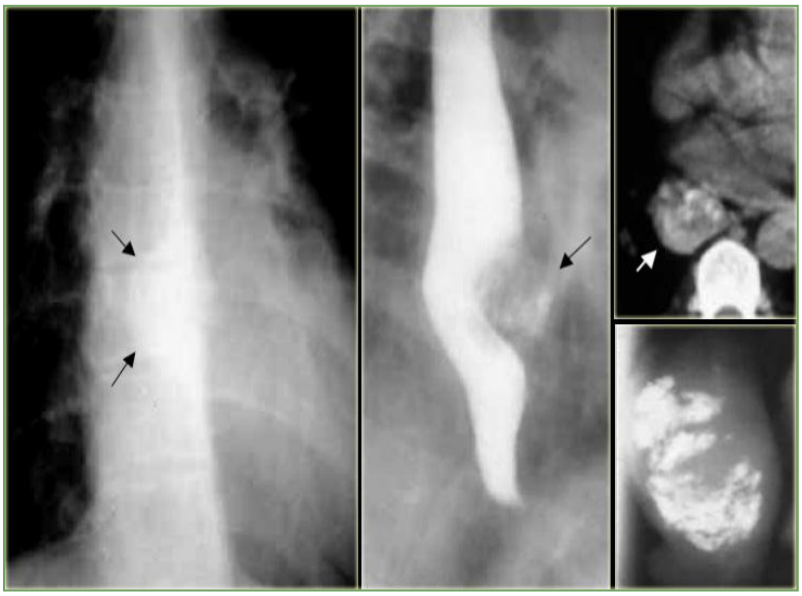
2. Endoscopy:
   - The diagnosis of esophageal cancer is made best from an endoscopic biopsy.
   - Any patient undergoing surgery for esophageal cancer must have an endoscopy before entering the operating room for a definitive resection.
3. CT scan: of the chest and abdomen is important to assess the length of the tumor, thickness of the esophagus and stomach, regional lymph node status and distant disease to the liver and lungs.
4. PET scan:
   - Evaluates the primary mass, regional lymph nodes, and distant disease.
   - Its sensitivity and specificity slightly exceed those of CT; however, they remain low for definitive staging.
5. Endoscopic ultrasound (EUS) is the most critical component of esophageal cancer staging ⓘ.
   - The information obtained from EUS will help guide both medical and surgical therapy.
   - Biopsy samples can be obtained of the mass and lymph nodes in the paratracheal, subcarinal, paraesophageal, celiac region.

- Treatment:
  1. Chemotherpay.
  2. Radiation therapy.
  3. Chemo-radiotherapy.
  4. Surgical resection.

9 LEIOMYOMA

- Leiomyomas constitute **60% of all benign** esophageal tumors.
- Found in men ≥ women and tend to present in the 4th & 5th decades.
- 80% are found in the distal 2/3 of the esophagus.
- Usually **solitary** and remain intramural, causing symptoms as they enlarge
- Recently, they have been classified as a gastrointestinal stromal tumor (GIST).
  - GIST tumors are the most common mesenchymal tumors of the gastrointestinal tract and can be benign or malignant.
  - Nearly all GIST tumors occur from mutations of the c-KIT oncogene, which codes for the expression of c-KIT (CD117).
- All leiomyomas are benign. Malignant transformation is rare ⓘ.
- **Clinical presentation:**
  - Many leiomyomas are <u>asymptomatic.</u>
  - Dysphagia and pain are the most common symptoms and can result from even the smallest tumors.

- **Diagnosis:**
  - CXR: not helpful.
  - Barium esophagram: leiomyoma has a characteristic appearance (smooth filling defect).
  - Endoscopy: extrinsic compression is seen, and the overlying mucosa is noted to be intact.
  - Endoscopic ultrasound (EUS): hypoechoic mass in the submucosa or muscularis propria.



- **Treatment:**
  - Leiomyomas are slow-growing tumors with rare malignant potential that will continue to grow and become progressively symptomatic with time.
  - Although observation is acceptable in patients with small (<2 cm) asymptomatic tumors or other significant co-morbid conditions, in most patients, surgical resection is advocated.
  - Surgical enucleation of the tumor remains the standard of care (thoracotomy or with video or robotic assistance).
  - The mortality rate is less than 2%, and success in relieving dysphagia approaches 100%.

10 MCQS

1. The gold standard investigation for GERD is:
   A) Ambulatory pH monitoring
   B) Barium swallow
   C) Endoscopy
   D) Clinical picture

2. Hiatus hernia:
   A) Reflux is not seen in paraesophageal type
   B) Dysphagia is the commonest symptom of sliding type
   C) Paraesophageal type is treated medically
   D) The gastroesophageal junction is intraabdominal in sliding type

3. Achalasia's usual investigating tools & equipment ... all except:
    A) Radionucleotide
    B) MRI
    C) Endoscopy
    D) Barium swallow

4. Most common site for squamous cell carcinoma of the esophagus is:
    A) Upper 1/3
    B) Middle 1/3
    C) Lower 1/3
    D) Site of esophageal reflux

5. Barrett's esophagus:
    A) Transforms into adenocarcinoma in 10% of cases
6. What is not true about leiomyoma of the esophagus?
    A) 10% are multiple
    B) They are due to mutation in c kit oncogene
    C) They arise from the mucosa of esophagus
    D) Most common site of origin is the middle 1/3 of esophagus

7. 10. Weight loss comes earlier with:
    A) Colon cancer
    B) Prostatic cancer
    C) Esophageal cancer
    D) Hepatoma

8. 70 year old male newly diagnosed with pharyngoesophageal (zenker's) diverticulum, which one of the following most likely the presenting symptoms?
    A) Abdominal pain
    B) Haematemesis
    C) Halitosis
    D) Vomiting

9. 55 year old newly diagnosed with diffuse esophageal spasm, his presenting history may include one of the following?
    A) Abdominal pain
    B) Chest pain and dysphagia
    C) Haematemesis
    D) Recurrent vomiting

🗝 Answers: 1;A, 2;A, 3;B (Source: bestpractice.bmj.com), 4;A (Source: Wikipedia), 5;A, 6;D, 7;C, 8;C, 9;B

ESOPHAGEAL DISEASES: CASES

1 OBJECTIVES

- Understand the history related to common esophageal diseases such as GERD.
- Understand the symptoms and signs of esophageal perforation.
- Understand the symptoms and signs of esophageal motility disorder.

2 CASE 1

Part 1:
A 50 year old male presented to you in the clinic with history of heartburn and hoarseness.
He is obese and a smoker.
Examination was unremarkable.

- *What else would you like to ask about in the history?*
- What are the symptoms of:
  - Classic GERD?
  - Complicated GERD?
- What are the extra-esophageal manifestations of GERD?
- What is your next step in management?

Part 2:
Barium-swallow report:
- No stricture or tumor.
- Small hiatus hernia.
- Evidence of reflux of the contrast.

- What are the types of hiatus hernia?
- What other tests & procedures can aid in the diagnosis? And what is their diagnostic value?

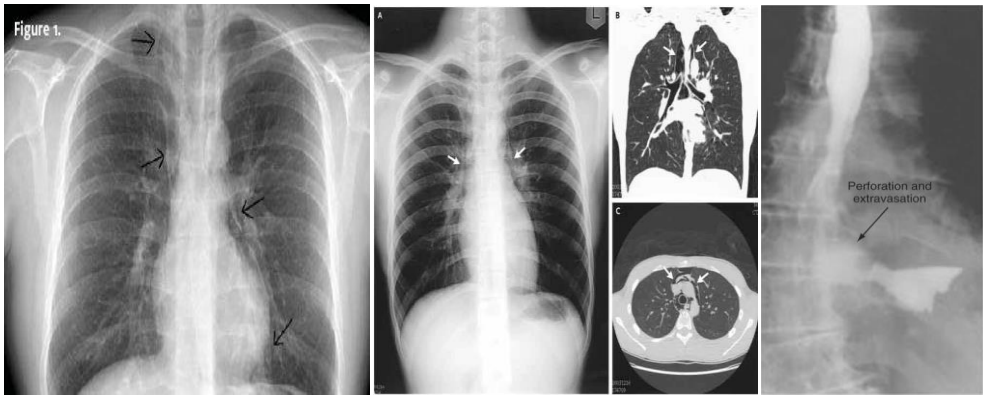
Part 3:
Biopsy was done.
Pathology report: esophagitis with intestinal, columnar epithelium replaces the stratified squamous epithelium (metaplasia) consistent with Barrett's Esophagus. No evidence of dysplasia.

- What is the next step in management?
- What are the indications of surgery?

Part 4:
You advise the patient to: Reduce weight and quit smoking, and schedule follow up endoscopy.
Started the patient on: Nexium 40 mg OD

3 months later, you did endoscopy for the patient. 6 hour post endoscopy patient started to complain of: chest pain & fever.

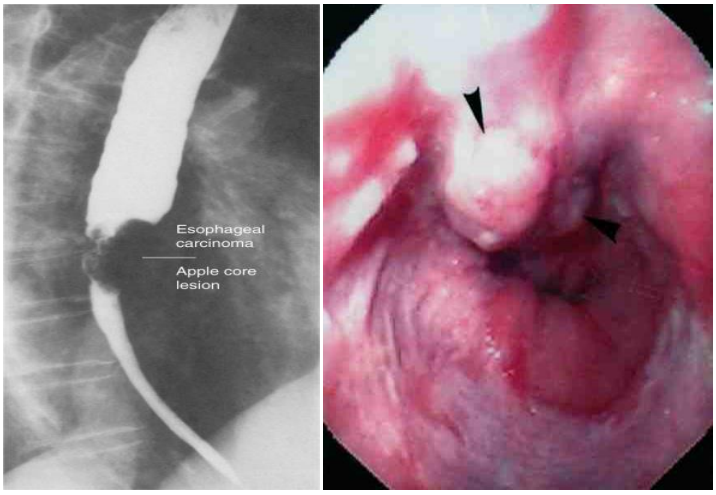
- What else would you like to ask about in the history?
- What is the next step in management?



- What is the treatment?

Part 5:
6 years later, he presented to your clinic complaining of: Dysphagia & weight loss.

- What else would you like to ask about in the history?
- Make a list of differential diagnoses.
- How are you going to manage this patient?



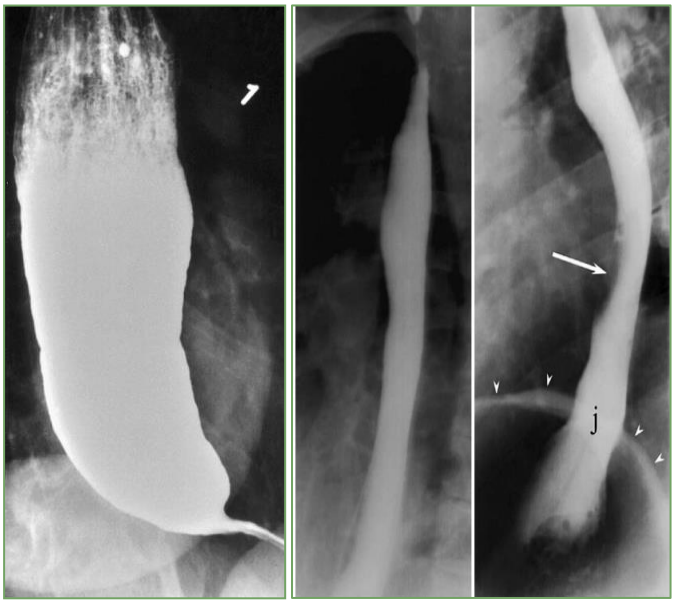
Part 6:
The biopsy from the endoscopy revealed: adenocarcinoma.

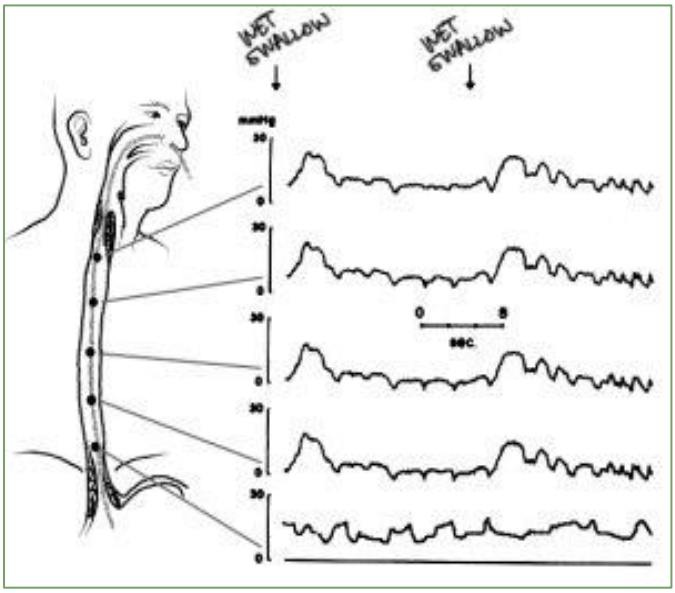
- What are the treatment options?

3 CASE 2

Part 1:
24 years old, healthy, presented to your clinic complaining of: Dysphagia.

- How will you manage this patient?





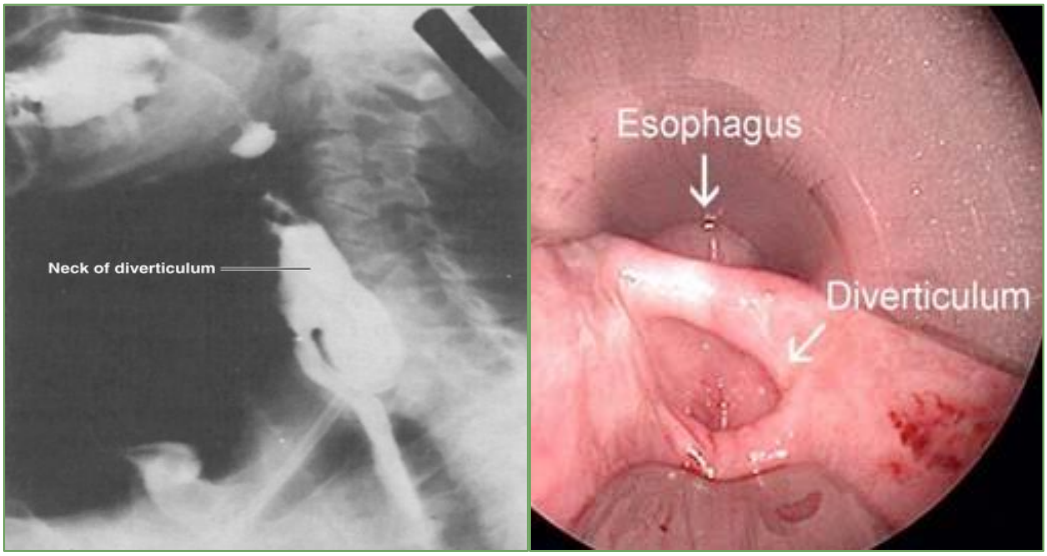
Part 2:
His manometry consistent with Achalasia.
Endoscopy showed:
- Dilated esophagus.
- Retained food particles.

- How will you treat this patient?

4 CASE 3

70 years old male, his wife bring him to your clinic because of:
- Bad breath.
- Chronic cough especially after eating.

- How will you manage this patient?



- What is the cause of esophageal diverticula?
- What are the different types of esophageal diverticula?
- What are the most common sites?

GASTRIC AND DUODENAL DISEASES

1 INTRODUCTION

- The duodenum is divided into 4 parts, which are closely applied to the head of the pancreas.
- The 1st part of the Duodenum:
  - 5 cm in length
  - Most common site for peptic ulceration to occur
  - Begins at the pylorus
  - Runs upward and backward on the transpyloric plane at the level of the first lumbar vertebra
  - The relations of this part are as follows:
    - Anteriorly: The quadrate lobe of the liver and the gallbladder
    - Posteriorly: The lesser sac (first inch only), the gastroduodenal artery (that's why posterior ulcers bleed ⓘ), the bile duct and portal vein, and the inferior vena
    - Superiorly: The entrance into the lesser sac (the epiploic foramen)
    - Inferiorly: The head of the pancreas

Superior Mesenteric Artery Syndrome: The obstruction of the 3rd part of the duodenum by the superior mesenteric artery

2 DISEASES OF THE STOMACH AND DUODENUM

2.1 PEPTIC ULCER

- Most common cause of abdominal pain related to the stomach and the duodenum ⓘ
- **Sites**:
  - Esophagus
  - Stomach
  - Duodenum
  - Jejunum (following a gastrojejunostomy)
  - Ileum (in relation to ectopic gastric mucosa in Meckel's diverticulum)
- Men are affected three times as often as women ⓘ
- Duodenal ulcers are ten times more common than gastric ulcers in young patients ⓘ
- In the older age groups the frequency is about equal ⓘ
- **Clinical presentation:**
  - Pain
  - Bleeding
  - Perforation
  - Obstruction

2.1.1 DUODENAL ULCER

- 95% occur in the duodenal bulb (2 cm), the first part of the duodenum ⓘ
- They may be acute (ulcers with a history of less than 3 months with no evidence of fibrosis) or chronic
- Common in young and middle-aged males

- Normal or increased acid secretion
- 90% caused by Helicobacter Pylori (GNCB aerophilic)
- **Clinical Features:**
  - Well-localized epigastric pain (mid-day, noon and night)
  - Pain when hungry, and is relieved by food ⓘ
- **Diagnosis:**
  1. EGD (Esophageogastroduodenoscopy)
  2. Gastric analysis:
     - Basal vs. Maximal (not practical and isn't used nowadays)
  3. Gastrin serum levels:
     - Severe or Refractory (Done if Zollinger-Ellison Syndrome is suspected or the treatment was not effective) ⓘ
  4. Contrast meal
     - Used when either endoscopy is contraindicated or complications of the ulcer have occurred ⓘ
- Before doing all the tests, you must first treat the patient if you suspect duodenal ulcer for at least 6 weeks ⓘ
- **Treatment**:
  1. Medical Treatment (80% in 6 weeks)
     - H2 antagonist (e.g. Zantac) – control acid secretion
     - Proton pump inhibitors (e.g. Omeprazol)
     - Antibiotics (e.g. Amoxicillin): For H. Pylori eradication
  2. Surgical Treatment [It has been limited to patients in whom complications have occurred or to block hormonal stimulation]
     - Vagotomy
     - Antrectomy and vagotomy
     - Subtotal gastrectomy

2.1.2 GASTRIC ULCER

- 95% occur along the lesser curvature in the distal half of the stomach ⓘ
- Gastric ulcers generally run a chronic course
- Common in 40-60 year old males (Gastric ulcer is more prevalent with older age)
- Gastric ulcers may develop into malignancy much more often than duodenal ulcers
- **Types:**
  1. In Incisura Angularis with normal acid
  2. Prepyloric and DU with high acid - Most common type
  3. In the Antrum due to NSAIDs
  4. At the Gastroesophageal Junction (GEJ)
- **Clinical Features:**
  1. Epigastric pain
  2. The pain occurs during eating and is relieved by vomiting (Patient might lose weight) (very ⓘ to help differentiate from duodenal)
- **Diagnosis:**
  - EGD with biopsy (Biopsy is important here to exclude malignancy)
  - Contrast swallow (Filling defect)
- **Treatment:**
  - Medical Treatment:

        - Not common
        - Eradication of H. Pylori
    - Surgical Treatment: Usually done to make sure that the ulcer does not develop into cancer ⓘ
- **Complications of surgical treatment for peptic ulcers:**
    1. Early complications: Leakage, bleeding and retention
    2. Late complications:
        A. Recurrent ulcer (marginal, stomal or anastomotic ulcers)
        B. Gastrojejunocolic and gastrocolic fistula
        C. Dumping syndrome ⓘ
            - There is no pylorus due to surgery, so the food will go to the small bowel directly due to eating food with osmotic potential
            - Patient will suffer from fainting and sweating
            - Early dumping
            - Late dumping is caused by hypoglycemia
            - Late dumping occurs 1-3 hours after a meal. The pathogenesis is thought to be related to the early development of hyperinsulinemic (reactive) hypoglycaemia.
            - Advise the patient to eat less sugar or give him acarbose
        D. Alkaline gastritis
        E. Anemia ⓘ
            - Iron deficiency
            - Vit. B12 deficiency (Pernicious anemia)
        F. Postvagotomy diarrhea
        G. Chronic gastroparesis
        H. Pylroic obstruction/ stenosis
- **Complications of Peptics ulcers:**
    - Pyloric obstruction:
        - Dull epigastric pain & projectile vomiting of large volumes of undigested food matter
        - Could be due to stricture formation
        - Medical treatment (must make sure pt is taking their medication even if the pain stops)
        - Surgical treatment
            1. Remove and anastomose
            2. Bypass
    - Perforation:
        - Occurs in acute ulcers (duodenal mostly)
        - On the anterior wall of the duodenum (duodenal ulcer)
        - Anterior ulcers cause perforation ⓘ**,** whereas posterior ulcers cause bleeding ⓘ
        - High risk: Female, old age, gastric ulcer
        - Acute onset of severe unremitting epigastric pain
        - **Diagnosis:** X-ray will demonstrate free air under the diaphragm [which means air in the peritoneum indicating that there is perforation of the viscus] (85%) and fill 400 cc of air by the Nasogastric tube (NGT) [Never do gastroscopy]
        - **Treatment:** NGT, ABS, Surgery

ⓘ Dumping Syndrome:

A condition where the ingested food bypasses the stomach too rapidly and enters the small intestine largely undigested. It happens when the upper end of the small intestine, the duodenum, expands too quickly due to the presence of hyperosmolar food from the stomach.

Clinical presentation:

- Tachycardia

- Flushing

- Sweating

- Colicky pain

- Hypoglycemia and may lead to fainting (seen more in late dumping)

ⓘ Beeding site in duodenal ulcers:

When bleeding (upper GI, presents with vomiting blood) is seen, we suspect the ulcer to be in the posterior wall of the 1st part of the duodenum. Perforation occurs in the anterior wall's ulcer, bleeding more commonly occurs in the posterior ulcer mainly due to the Gastroduodenal artery that lies behind the 1st part of the duodenum.

2.2 ZOLLINGER-ELLISON SYNDROME (GASTRINOMA)

- Peptic ulcer disease (often severe) in 95%
- Gastric hypersecretion + very high no. of ulcers + gastroma
- Elevated serum gastrin
- Single one is usually malignant
- Multiple is benign (MEN 1)
- **Diagnosis:**
  - Gastrin levels more than 500 pg/ml ⓘ
  - CT Scan, somatostatin scan
  - Portal vein blood sample
- **Presentation:** Diarrhea (steatorrhea due to the inactivation of the pancreatic lipase) and severe persistent epigastric pain
- **Treatment:**
  1. Medical treatment: Acid control (massive dose of PPI) ⓘ
  2. Surgical treatment: Distal hemi-gastrectomy and ulcer excision

2.3 UPPER GASTROINTESTINAL BLEEDING

- **Presentation:**
  - Hematemesis
  - Melena
  - Hematochezia [Occurs very rarely]
- **Causes of massive upper gastrointestinal hemorrhage:**

Common causes	Uncommon causes 5%
Peptic ulcer ⓘ **45%**	Gastric carcinoma
Duodenal ulcer **25%**	Esophagitis
Gastric ulcer **20%**	Pancreatitis
Esophageal varices **20%**	Hemobilia
Gastritis **20%**	Duodenal diverticulum
Mallory-Weiss syndrome **10%**	

Management:

1. Resuscitation
2. Detection and endoscopic treatment (If the cause is an ulcer we can either put a clip on it, burn it, use a rubber band or injection of a sclerosing agent to form a clot and stop the bleeding)
3. Surgical management

2.4 MALLORY-WEISS SYNDROME

- Usually caused by severe retching, coughing, or forceful vomiting ⓘ
- 10% of Upper Gastrointestinal Bleeding (UGIB) cases
- 1-4cm longitudinal tear in gastric mucosa at esophageal-gastric junction (most common site) ⓘ
- EGD is done to confirm diagnosis
- 90% of bleeding stops spontaneously:
  - By cold gastric wash (To induce vasospasm to stop the bleeding)
  - If it doesn't stop, we perform EGD
  - If the tear is small, we can burn it (cautery). If not, it will need surgical intervention.

2.5 STRESS GASTRODUODENITIS, STRESS ULCER & ACUTE HEMORRHAEGIC GASTRITIS

- Stress ulcer: Ulcer due to shock or sepsis

- Curling's ulcer: Ulcer due to burns
- Cushing's ulcer: Ulcer due to the presence of a CNS tumor or injury (more to perforate, high acid production)
- Acute Hemorrhagic Gastritis

2.6 GASTRIC POLYPS

- Incidental finding
- **Type of Gastric polyps:**
  1. Hyperplastic – treat with Omeprazole
  2. Adenomatous (Premalignant) – most serious
  3. Inflammatory
- Affects distal part of the stomach
- **Presentation:** Anemia
- EGD to R/O malignancy
- You have to resect the adenomatous type due to its malignant potential

If found through endoscopy, remove the polyp. If the polyp is found to be adenomatous, we do further investigations.

2.7 GASTRIC LEIOMYOMAS

- Incidental finding
- Benign smooth muscle tumor
- Common submucosal growth
- 90% asymptomatic, less than 1% present with massive bleeding
- **Diagnosis:** EGD and CT scan (bulging mass in the mucosa on endoscopy)
- Never take biopsy (the capsule will break) ⓘ
- Surgical wide excision

2.8 MENETRIER'S DISEASE

- Giant hypertrophy of the gastric rugae (thick rugae)
- Presents with hypoproteinemia
- Diarrhea, edema and weight loss
- **Treatment:**
  - Atropine (to reduce the secretion)
  - Omeprazole
  - H. Pylori eradication
  - If the patient still has symptoms we perform a gastrectomy (rarely so)

Mucosal hypertrophy may lead to abnormally large secretion of protein-rich mucus and acid (This over-secretion contributes to symptoms of epigastric pain and hypoproteinaemia).

2.9 PROLAPSE OF THE GASTRIC MUCOSA

- Occasionally accompanies small gastric ulcer
- **Presentation:** Vomiting and abdominal pain
- **X-Ray:** Antral folds into duodenum (Double ring on X-ray) [not well defined]
- **Treatment:** Antrectomy with Billroth 1

2.10 GASTRIC VOLVULUS ⓘ

- Benign disease, but lethal (can lead to death)
- **Types:**
  1. Its longitudinal axis (Organoaxial volvulus):
     - More common

    - Associated with HH (hiatal hernia)
  2. Transverse (Mesenterioaxial volvulus):
    - Line drawn from the mid lesser curvature to the mid greater curvature - Associated with vomiting (obstruction)
- **Presentation:** Severe abdominal (epigastric) pain and Brochardt's triad

Brochardt's Triad ⓘ

- Vomiting followed by retching and then inability to vomit
- Epigastric distention
- Inability to pass a nasogastric tube
- **Diagnosis**: Confirmed by a Ground Glass appearance on X-Ray
- If diagnosed, we should immediately take him to the OR

2.11 GASTRIC DIVERTICULA

- Uncommon
- Asymptomatic
- Weight loss, diarrhea
- It causes anemia
- **Diagnosis:** EGD, X-Ray
- Surgery

2.12 DUODENAL DIVERTICULA

- Affects 20% of the population
- Asymptomatic – incidental finding
- 90% in the medial aspect of the duodenum
- Rare before 40 years of age
- Most are solitary and 2.5 cm peri-ampullary of vater
- It can cause obstruction, bleeding and inflammation
- If it's asymptomatic, we leave it. If there is superficial cancer, we excise it.

2nd part of the duodenum is the most common site for diverticulum formation in the GI tract.

2.13 BEZOAR

- Retained concretions of indigestible foreign material in the stomach (foreign body in the stomach)
- **Types:**
  1. Trichobezoars: Formed from hair
  2. Phytobezoars: Indigestible plant material
- **Presentation:** Obstruction
- **Diagnosis:** EGD, X-Ray
- **Treatment:** Surgical removal

2.14 BENIGN DUODENAL TUMORS

- Brunner's gland adenomas
- Carcinoid tumors
- Heterotopic gastric mucosa
- Villous adenomas

2.15 SUPERIOR MESENTERIC ARTERY OBSTRUCTION OF THE DUODENUM

- Obstruction of the third portion of the duodenum leads to compression of the superior mesenteric artery (SMA) and Aorta
- Appears after rapid weight loss following injury
- Distance between two vessels is 10-20 mm
- Proximal bowel obstruction symptoms and signs (Vomiting)
- **Diagnosis:** CT Scan
- **Treatment:** Bypass surgery

Fat is the only thing that lies between the duodenum and the SMA. So when a person is cachexic and chronically ill, the fat will diminish and this will bring the duodenum and SMA closer to each other, leading to the obstruction.

2.16 REGIONAL ENTERITIS OF THE STOMACH & DUODENUM

- Food poisoning
- **Presentation:** Pain and diarrhea
- Clinical diagnosis
- Observation of the patient

Three emergencies that need immediate intervention:
1. Gastric volvulus
2. Superior mesenteric artery syndrome
3. Mesenteric thrombosis
- E.g. history of Atrial fibrillation; will cause embolization
- Severe pain, do CT

3 MCQS

1. Features of Dumping syndrome include all of the following except:
a. Tachycardia
b. Sweating
c. Palpitations
d. Constipation
e. Diarrhea

2. Regarding the treatment of duodenal ulcers:
a. Most duodenal ulcers are treated medically with no need for surgical intervention
b. Arteriography in bleeding ulcers is a useful diagnostic modality but has no place in therapy
c. Endoscopy in bleeding ulcers is a useful diagnostic modality but has no place in therapy
d. When a vagotomy is performed only one vagus should be divided in order to preserve the pyloric function
e. A Billroth 2 gastrectomy is more physiological and anatomical than highly selective vagotomy

3. Which one of the following statements is true about Mallory-Weiss syndrome:
a. It is caused by H. Pylori organism infection
b. It is a 1-4 cm longitudinal tear in gastric mucosa at EGJ
c. It causes 80% of upper GI bleeding
d. 5% of the bleeding stops spontaneously

Answer key: 1;D , 2;A , 3;B

INFLAMMATORY BOWEL DISEASE

1 OUTLINE

- What is the Disease?
- Epidemiology
- Pathophysiology
- Ulcerative Colitis
- Crohn's Disease

2 INFLAMMATORY BOWEL DISEASE

2.1 TWO CHRONIC DISEASES THAT CAUSE ULCERATION & INFLAMMATION OF THE INTESTINES

- **Ulcerative Colitis**
- **Crohn's Disease.**
- They have some features in common but there are some important differences
- 20% of patients have clinical pictures that fall in between (indeterminate colitis)

Some patients have (undetermined colitis): if the inflammation is in the large bowel and we do not know whether it is UC or CD

2.2 EPIDEMIOLOGY

- Most numbers are North American
- Increasingly diagnosed in Saudi Arabia
- It increased in the last decade due to improvement of the diagnostic methods and changing lifestyle

Epidemiology of inflammatory bowel disease

Incidence, per 100,000 (North America)	3-14 (CD)
	2-14 (UC)
Prevalence, per 100,000 (North America)	26-199 (CD)
	27-246 (UC)
Geography	Northern Countries > Southern Countries
Age of onset	Peak: 15-30
	Second Peak 50-80 (CD)
Sex	M = F
Race	Whites > Blacks
Ethnic	Jewish > Non-Jewish
Smoking	Associated with CD: protective in UC
Appendectomy	May be protective in UC
Possible genetic associations	Chromosome 16 (CD)
	Chromosome 3, 5, 7, 12, 19 (UC and CD), TNF-(CD); IL-1A (CD), IL-23 receptor (CD and UC), ATG I6L1 (CD), HLA-A2; HLA-DR1; DQw5 (CD), HLA-DR2 (UC)

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2.3 PATHOPHYSIOLOGY

- Unclear
- A number of factors may be involved: ⓘ
  - Host Factors
  - Environmental Factors

2.3.1 HOST FACTORS

- Genetics (Twins, Relatives, & children) → not very clear but we believe that the risk increase for relatives, if a family member has it.
- Obesity (increases the risk of IBD)
- Appendectomy (decreases the risk of IBD)

2.3.2 ENVIRONMENTAL FACTORS

- Smoking (protective against UC but it increases the incidence of CD)
- Infection
- Oral Contraception

2.3.3 CURRENT THEORY: ⓘ

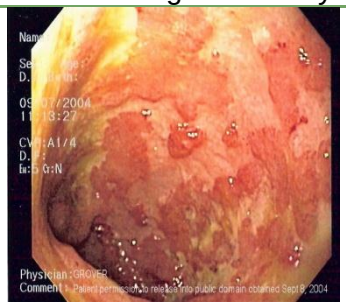
- There is a genetic defect that affects the immune system, so that it attacks the bowel wall in response to stimulation by an offending antigen, like a bacteria, a virus, or a protein in the food

3 ULCERATIVE COLITIS

- An inflammatory disease of the **large intestine** ⓘ
- Recurring and **continuous** Inflammation and ulceration of the **mucosa** of the large intestine ⓘ
- Almost always involve the **rectum** and extend proximally ⓘ
- NB : if the rectum is free of inflammation consider other diagnoses than UC
- It extends in a continuous fashion
- 40-50% of patients have disease limited to the rectum and rectosigmoid
- 30-40% of patients have disease extending beyond the sigmoid
- 20% of patients have a total colitis

3.1 MACROSCOPIC APPEARANCE ⓘ

- Erythematous mucosa, has a granular surface, looks like sand paper
- In more severe diseases hemorrhagic, edematous and ulcerated
- In fulminant disease a toxic colitis or a toxic megacolon may develop



3.2 MICROSCOPIC APPEARANCE ⓘ

- **Crypt abscesses**
- Branching of crypts
- Atrophy of glands
- Loss of mucin in goblet cells

3.3 ULCERATIVE COLITIS PRESENTATION

3.3.1 THE MAJOR SYMPTOMS OF UC ARE:

- Diarrhea (4 to more than 10)
- Rectal bleeding
- Tenesmus (painful passing of the mucus) & Passage of mucus
- Crampy abdominal pain & Fever
- Exam is often normal unless complications occur.

3.4 ULCERATIVE COLITIS COMPLICATIONS

- Hemorrhage: if it's too much, we need surgical intervention to stop it
- Toxic megacolon
- Perforation
- Stricture (due to the fibrosis)
- Cancer (late stages) → so IBD patients need special programs for screening: the screening is biopsy taking – endoscopy is not enough ⓘ

3.5 EXTRA-INTESTINAL MANIFESTATIONS ⓘ

- Uveitis and Episcleritis
- Erythema Nodosum and Pyoderma Gangrenosum
- Arthritis
- Ankylosing Spondylitis
- Sclerosing cholangitis

3.6 ULCERATIVE COLITIS TREATMENT

- Mainly medical treatment but if it fails we use surgical treatment

3.6.1 SURGICAL TREATMENT

- Failure of medical management
- Treating complications
- Prophylaxis for cancer
- Cure after colectomy

Medical therapy of active ulcerative colitis according to disease severity

Disease severity	Medication	Daily dose
Mild-to-moderate disease		
	Sulfasalazine	1 to 1.5 g PO four times daily
	Mesalamine	
	Delayed release EC tablet:	
	- Asacol*	800 to 1600 mg PO three times daily
	- Lialda*	2.4 or 4.8 g PO once daily (2.4 g initially; 4.8 g if no complete response)
	Extended release capsule:	
	- Apriso*	1.5 g orally (four Apriso* capsules) in the morning once daily
	Controlled release capsule:	
	- Pentasa*	500 to 1000 mg PO four times daily
	Olsalazine	1 to 1.5 g PO twice daily
	Balsalazide	2.25 g PO three times daily
	Mesalamine suppository	1000 mg at night
	Hydrocortisone foam 10% (rectal)	90 mg (one applicatorful) at night or twice daily
	Mesalamine enema	4 g at night
	Hydrocortisone enema	100 mg at night
	Sulfasalazine/oral 5-ASA plus 5-ASA enemas/steroid enema	
	Prednisone	40 to 60 mg PO once daily
Severe active disease		
On steroids recently	Methylprednisolone	48 to 60 mg IV once daily
	Hydrocortisone	100 mg IV every 6 hours or as continuous infusion
	Cyclosporine	See topic review for dosing
	Infliximab	See topic on "Anti-tumor necrosis factor therapy in ulcerative colitis"
Toxic megacolon	Intravenous corticosteroids	See topic on "Toxic megacolon"
	Broad-spectrum antibiotics	
Chronic active disease (steroid refractory)	Mercaptopurine	See topic on "Azathioprine and 6-mercaptopurine in ulcerative colitis"
	Azathioprine	
	Infliximab	See topic on "Anti-tumor necrosis factor therapy in ulcerative colitis"

5-ASA: mesalamine, olsalazine, or balsalazide; anti-TNF: anti-tumor necrosis factor; UC: ulcerative colitis; EC: enteric coated.
* United States brand names.

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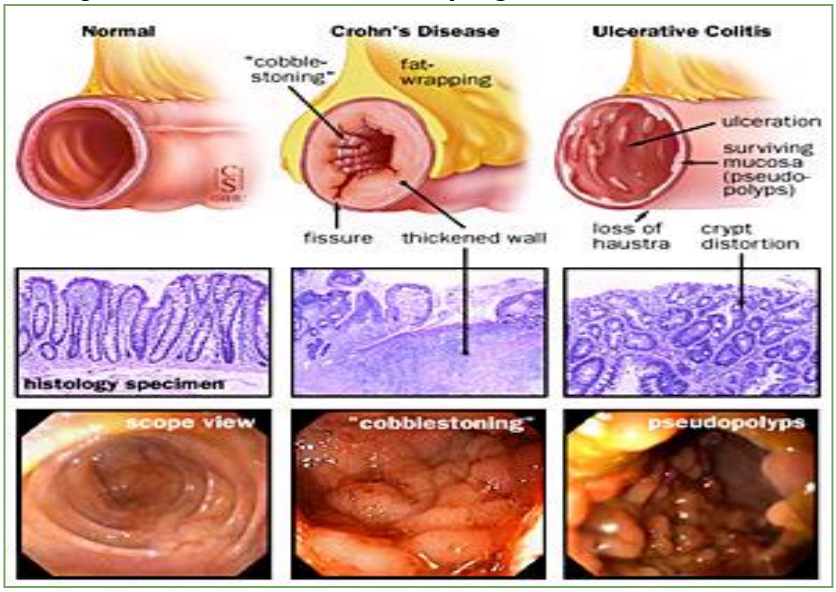
4 CROHN'S DISEASE

- An inflammatory disease that **affects any part of the GI tract** ⓘ
- 80% Small bowel
- 50 % ileocolitis
- 20 % colon
- 30% perianal disease
- UGI < 5 %
- Recurring **transmural** Inflammation of the bowel ⓘ
- About 80% have small bowel involvement, mostly the **terminal ileum** ⓘ
- Characterized by **skip lesions** ⓘ
- 30-40% of patients have small bowel disease alone
- 40-55% of patients have both small and large intestines disease
- 15-25% of patients have colitis alone

ⓘ Research shows a possible link between **Mycobacterium Paratuberculosis** and **Crohn's Disease**

4.1 MACROSCOPIC APPEARANCE

- Mild disease has aphthus or small superfecial ulcers
- In more severe diseases there is the characteristic **cobblestone appearance** ⓘ
- Thickening of the bowel wall with **creeping fat** ⓘ



4.2 MICROSCOPIC APPEARANCE

- **Transmural inflammation** ⓘ
- Focal ulcerations
- Acute and chronic inflammation
- Granulomas may be noted in up to 30% of patients

4.3 CROHN'S DISEASE PRESENTATION

4.3.1 THE MAJOR PRESENTATIONS OF CD ARE

- Crampy abdominal pain ⓘ
- Diarrhea (rarely accompanied with blood ((less than UC)))
- Weight loss
- Colitis and Perianal disease (If a patient presents with a perianal disease that resists treatment then consider CD)
- Duodenal Disease

4.4 CROHN'S DISEASE COMPLICATIONS

- Phlegmons (inflammatory mass) & abcesses
- Fistulas
- Stricture
- Malabsorption
- Perianal disease
- Cancer risk

4.5 EXTRA-INTESTINAL MANIFESTATIONS ⓘ

- Uveitis and Episcleritis
- Erythema Nodosum and Pyoderma Gangrenosum
- Sclerosing cholangitis
- Renal stones
- Gall stones
- Amyloidosis

4.6 CROHN'S DISEASE TREATMENT

- Mainly medical treatment

4.6.1 MEDICAL TREATMENT

- Oral 5-aminosalicylates (sulfasalazine)
- Antibiotics (Cipro, Metronidazole) ⓘ
- Glucocorticoids (Prednisone)
- Immunomodulators(Azathioprine)
- Biologic therapies (infliximab)

4.6.2 SURGICAL TREATMENT

- Failure of medical management
- Treating complications
- Not a Cure ⓘ

5 MCQS

A 22 y/o male presents to the clinic complaining of abdominal pain, diarrhea, and weight loss lasting for one month. He gave a history of occasional occult bleeding in stool. The most likely diagnosis is:

A. Crohn's disease
B. Peptic ulcer
C. Incarcerated hernia
D. Intestinal obstruction

Features of the previous diagnosis include all the followings EXCEPT:

A. Mucosal ulceration separated by normal mucosa
B. All cases should be treated surgically
C. The most common site is the ilium
D. Development of fistulae is a known complication

Crypt abscesses are a feature of:

A. Crohn's Disease
B. Ulcerative Colitis
C. Colon cancer
D. Both A & B

Transmural inflammation of the colon is seen in:

A. Crohn's Disease
B. Ulcerative Colitis
C. Colon cancer
D. Both A & B

	UC	*Crohn's disease*
Blood in stool	Yes	Occasionally
Mucus	Yes	Occasionally
Systemic symptoms	Occasionally	Frequently
Pain	Occasionally	Frequently
Abdominal mass	Rarely	Yes
Perineal disease	No	Frequently
Fistulas	No	Yes
Small Intestine Obstruction	No	Frequently
Colonic Obstruction	Rarely	Frequently
Responce to Antibiotc	No	Yes
Recurrance after surgery	No	Yes
Rectal Sparing	Rarely	Frequently
Continuous Disease	Yes	Occasionally
Cobblestoning	No	Yes
Granuloma on Biopsy	No	Occasionally

🗝 Answer Key →1;A , 2;B , 3;B , 4;A

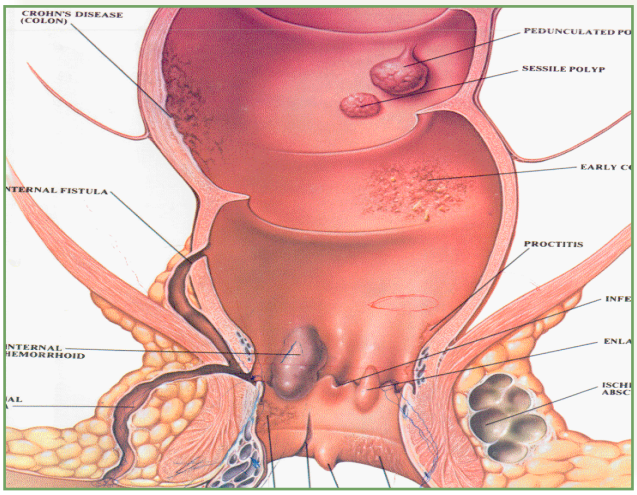
ANORECTAL CONDITIONS

1 OVERVIEW: ANATOMY AND PHYSIOLOGY

- The anal canal is approximately 3-4 cm long and extends from the anorectal junction (dentate/pectinate line) to the anal verge
- **Blood supply** →
  - **a.** The superior rectal (a branch from the inferior mesenteric artery)
  - **b.** The middle rectal (a branch from the internal iliac artery)
  - **c.** The inferior rectal artery (a branch from the internal pudendal artery)
- **Venous drainage** →
  - **a.** The superior rectal (to the portal system by the inferior mesenteric vein)
  - **b.** The middle and the inferior rectal veins (to the systemic circulation by the internal iliac and pudendal vein)
- The **physiology** of anal continence is the result of complex interactions between sensory, involuntary and voluntary motor functions
- The **dentate line** is the transitional zone from columnar rectal epithelium and the squamous anal epithelium
  - Above the line: endodermal origin, lined by columnar rectal epithelium, no sensation of pain except in ischemic cases, it's only sensitive to stretch
  - Below the line: anodermal origin, lined be squamous anal epithelium, sensitive to pain, richly innervated by somatic sensory nerves. Pathologic conditions that arise below the level of the dentate line cause severe pain.
- **Internal anal sphincter** → involuntary sphincter of smooth muscle, autonomic innervation, controls gas and liquid stool.
- **External anal sphincter** → striated voluntary muscle, controls solid stool

2 ANORECTAL ABSCESSES AND FISTULA-IN-ANO

- Both abscess and fistula-in-ano can be considered simultaneously
- The abscess is an **acute** manifestation, & the fistula is a **chronic** condition
- Most surgeons' reputations reportedly have been impugned because of problems with fistula operations than from other operative procedure



2.1 ETIOLOGY

NON-SPECIFIC	SPECIFIC
√ **Crypto-glandular in origin** 90% – Crypto-glandular hypothesis states that infection of anal glands associated with anal crypts is the primary cause of anal fistula & abscess – In the dentate line there are about 9-15 glands inside the crypt, which secrete through ducts. ▪ In case of obstruction → accumulation of secretion & inflammation → causing abscess and anal fistula – An anal fistula is an abnormal connection between the epithelialized surface of the anal canal and (usually) the perianal skin – **Anal fistulae** originate from the anal glands, which are located between the two layers of the anal sphincters, which drain into the anal canal. If the outlet of these glands becomes blocked, an abscess can form which eventually point to the skin surface. The tract formed by this process is the fistula.	√ Crohn's disease (most important; very tough to treat) √ Ulcerative colitis √ TB √ Actinomycosis √ Carcinoma √ Trauma √ Radiation √ Lymphoma, leukemia √ Foreign body √ Pelvic inflammation

2.2 CLINICAL PRESENTATION

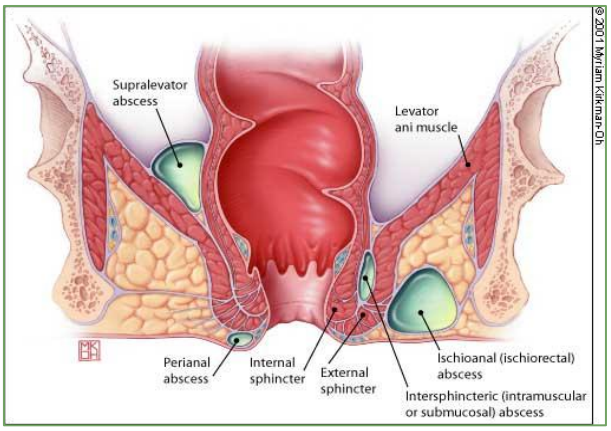
- Abscess (acute) →
  - Patient presents with constant throbbing perianal pain and systemic manifestations as fever if it becomes infected
- Anal fistulae (chronic)→
  - Patient most likely has a history of abscess, pus discharge (bloody/purulent), pruritus ani, perianal discomfort

2.3 DIAGNOSIS

- Done by examination, either in an outpatient setting or under anesthesia (EUA: examination under anesthesia)

2.4 CLASSIFICATION OF **ABSCESSES** (ACUTE PHASE)

1. Ischiorectal/ischioanal (most common) ⓘ
2. Intersphincteric abscess
3. Perianal abscess
4. Supralevator/pelvirectal abscess (rare, difficult to diagnosis; caused by inflammation or a disease of the pelvis)



The process of abscess drainage results in the formation of a communication between the skin and anal canal. Therefore, 50% of abscesses will form a fistula (patient presents after few months from drainage with discharge)

2.5 TREATMENT OF **ABSCESSES**

2.5.1 INCISION AND DRAINAGE

- Prompt surgical drainage to prevent permanent fistula formation
- Determine the most tender point: a 2 cm area of skin is injected with local freezing
- Elliptical or cruciate incision
- Drainage of pus & destroying all loculations

All fistulae open into the dentate line except traumatic fistulae, which are difficult to treat

2.5.2 ANTIBIOTICS

- Alone: has **NO** role in the primary treatment of an abscess
- Except in certain groups of people: ⓘ
  - Immunocomprised
  - Patients with valvular disease
  - Diabetics
  - Extensively diseased patients
  - Associated with systemic manifestations

2.6 CLASSIFICATION OF **FISTULAE** (CHORNIC PHASE)

1. Intersphincteric: via the internal sphincter to the intersphincteric space and then to perineum
2. Transsphincteric: low, via the internal & external sphincters into the ischiorectal fossa and then to the perineum
3. Suprasphincteric: via the intersphincteric space superiorly to above the puborectalis muscle into the ischiorectal fossa and then to the perineum
4. Extrasphincteric (traumatic), as in gunshot wounds: from the perianal skin → levator ani muscles → rectal wall i.e. no specific relation to sphincters.

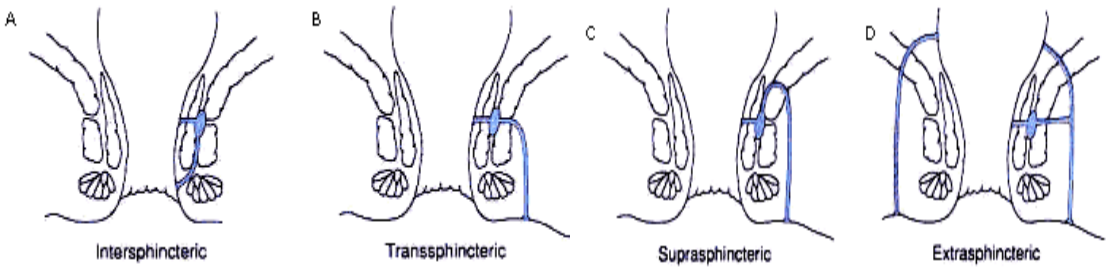
Parks Classification of Fistula-in-ano:

1. Intersphincteric fistulas: simple low tract, high blind tract, and high tract with rectal opening, rectal opening without a perineal opening, extrarectal extension, and secondary to pelvic disease.

2. Transsphincteric fistulas: uncomplicated, high blind tract

3. Suprasphenctric fistulas: uncomplicated, high blind tract

4. Extrasphincteric fistulas: secondary to anal fistula, secondary to trauma, secondary to anorectal disease, caused by pelvic inflammation

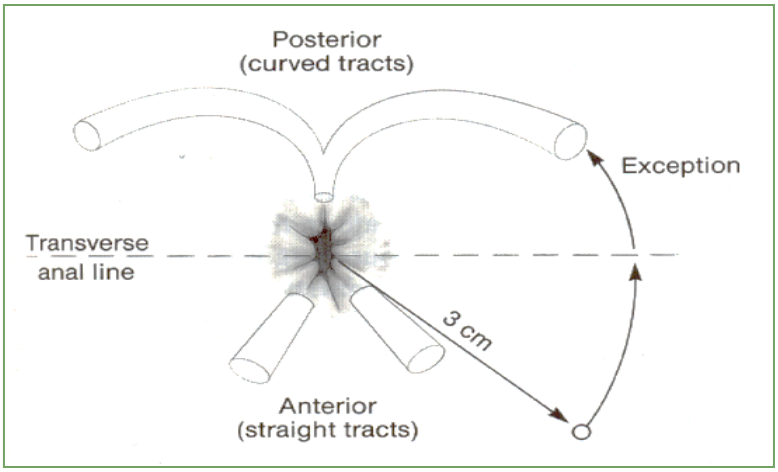


2.7 EVALUATION OF **ANAL FISTULA**

- An accurate pre-operative evaluation is **very important**
- Management depends on muscles involvement
- **Five** essential points of a clinical examination of an anal fistula:
  - √ Location of the internal opening
  - √ Location of the external opening
  - √ Location of the primary tract
  - √ Location of any secondary tracts
  - √ Determination of presence/absence of underlying disease
- **Digital examination** is up to 79%, 84%, and 71% accurate in defining the internal opening, primary track and secondary track, respectively.
- Resting anal tone & voluntary anal contraction before operation should be determined
- Intersphincteric tracks tend to open externally near the anal verge while transsphincteric and more complicated fistulas tend to open further away from the anal verge
- Gentle use of probes along the dentate line or through the external opening may be useful in locating internal openings
- Injection of **hydrogen peroxide** via the external opening into the track may help locate the internal opening and outline the fistula tract course. This may be useful to help delineate missed internal openings. ⓘ

2.7.1 GOODSALL'S RULE ⓘ

- With the patient in knee-chest position, an imaginary horizontal line is drawn at the level of the anus, parallel to the floor.
- For an external opening located:
  - Anterior to this line: the tract passes radially straight towards the internal opening (i.e. 9 to 3 o'clock position)
  - Posterior to this line: the fistulae tract is curved around & internal opening is in a frank midline position (i.e. to 6 o'clock position)



2.8 INVESTIGATIONS

- Fistulography
  - Involves injection of contrast via the internal opening
  - The accuracy rate is 16-48%
- Endoanal/Endorectal US:
  - Involves passage of 7-10 MHz transducer into the anal canal to help define muscular anatomy and differentiating intersphincteric from transsphincteric lesions
  - 50% better than physical examination alone, 94% accuracy rate
  - A standard water filled balloon transducer can help evaluate the rectal wall for any suprasphincteric extension
- CT scan
- MRI **(the best modality)**
  - Findings show 80-90% concordance with operative findings when observing primary tracts course & secondary extensions
  - MRI is becoming the study of choice when evaluating complex fistulae
  - It has been shown to improve recurrence rates by providing information on otherwise unknown extensions

Abscesses & fistulas are usually seen as hypo-echoic perirectal defects. Sometimes, fistulous tracts can't be easily recognized with US. In order to aid identifying these tracts, **hydrogen peroxide** can be injected into the external opening making the tract course visible

2.9 TREATMENT

- Goals of therapy:
  - Cure with lowest possible recurrence rate
  - Minimal, if any, alteration in continence in the shortest period
- The principles are:
  - Identification of primary opening
  - Relationship to puborectalis muscle
  - Least amount of muscle should be divided
  - Side tracts should be sought
  - Presence of underlying disease

To treat any fistula, must identify the external opening by inspection and the internal opening according to goodall's rule.

2.9.1 FISTULOTOMY/FISTULECTOMY

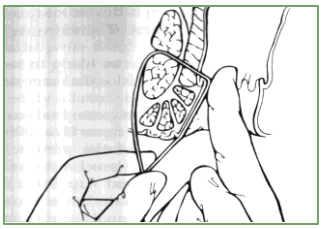
- The **gold** standard
- Considerations:
  - Age, sex, location, type of fistula, previous anorectal surgery, anal manometer
- Complication: Incontinence (by cutting the anal sphincter muscle)

➢ **FISTULECTOMY**:
  - Going around the tract, excising it completely and then close
  - Complete fistulectomy creates larger wounds that take longer to heal & offers no recurrence advantage over fistulotomy

- **FISTULOTOMY** (laying open technique):
  - Fistulous tract is merely laid open to heal
  - Useful for 85-95% of primary fistulae i.e. submucosal, intersphincteric & low transsphincteric.
  - A probe is passed into the tract through internal & external openings
  - Overlying skin, subcutaneous tissue, and internal sphincter are divided, thereby opening the entire fibrous tract
  - At low anal levels, the internal sphincter & subcutaneous external sphincter can be divided at right angles to the underlying fibers without affecting continence
  - Curettage is performed to remove granulation tissue in the tract base
  - Marsupialization of the edges speeds up healing
  - Opening the wound out on the perianal skin for 1-2 cm adjacent to the external opening with local excision of skin promotes internal healing before external closure
  - Perform a biopsy on any firm or suggestive tissue

2.9.2 SETONS

- Placement of a seton which can be made from large silk sutures, silastic vessel markers, or rubber bands that are threaded through the fistula tract
- Most patients prefer this method to avoid incontinence
- It takes 6-8 weeks or more
- Setons purposes:
  1. Visual identification of the amount of sphincter muscle involved (as markers for better postoperative assessment by outlining the track)
  2. Cutting seton (silk) → applied tightly to cut slowly though the tract
  3. Drainage seton (rubber) → applied loosely to serve as drains
  4. Fibrosis induced by the seton prevents separation of the ends of the anal sphincter muscle when fistulotomy is subsequently performed
- A seton can be placed alone, combined with fistulotomy or in a staged fashion. It is useful in these situations:
  - Complex/high fistulae (i.e. high transsphincteric, suprasphincteric, extrasphincteric)
  - Multiple fistulas
  - Recurrent fistula after previous fistulotomy
  - Anterior fistula in female patients
  - Poor preoperative sphincter pressures
  - Crohn's disease (IBD)
  - Immunosuppressed patients
  - Extensive scarring
  - Crazy fistula
  - Lazy sphincter



Every time the patient goes to the washroom, he/she pulls the Seton till he/she can't bear the pain (slipping it through his/her hand)

In females, **NEVER** cut anterior fistulae, they're located in a weak muscle where cutting may lead to incontinence. Therefore, the seton technique is useful here.

2.9.3 ANAL FISTULA PLUG

- Suturable bioprosthetic plug was fashioned to seal and close the primary opening of fistulae tracts
- Made of porcine small intestine submucosa

2.9.4 MUCOSAL ADVANCEMENT FLAP

- Reserved for use in patients with chronic complex high fistulae, but is indicated for the same disease processes as Seton use.
- Technique:
  - Involves total fistulectomy with removal of the primary & secondary tracts, excision of the internal opening, and closure of the rectal defect with a mucosal advancement flap
  - A rectal mucomuscular flap with a wide proximal base (2 times the apex width) is raised.
  - Curettage of the external portion of the tract, as opposed to fistulectomy or excision
  - The internal muscle defect is closed with an absorbable suture, and flap is sewn down over the internal opening so that its suture line doesn't overlap the muscular repair.
- Advantages →
  - Reduced healing time
  - No additional sphincter damage
  - No deformity of the anal canal
  - One stage procedure if primary healing is achieved
- Disadvantages →
  - Poor success in Crohn's or acute infection

2.10 CAUSES OF RECURRENCE:

1. Failure to identify the primary internal orifice
2. Lateral or upward extensions
3. Failure to open fistulous tract
4. Premature closure of wound
5. Etiology
6. Surgeon performing the procedure

2.11 CAUSES OF INCONTINENCE:

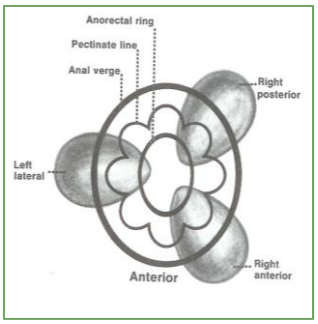
1. 27% overall risk
2. Division of anorectal muscles
3. Severance of motor nerve to sphincter mechanism
4. Prolonged packing after surgery
5. Previous operation
6. Female

Rectovaginal Fistulas:

Constitute 5% of all anorectal fistulas, most commonly due to obstetric trauma. Diagnosed by endorectal US, transvaginal US, and methylene blue enema to confirm the diagnosis.

3 HEMORRHOIDS

3.1 ANATOMY AND CLASSIFICATION



- Vascular cushions located in the anal canal covered by mucosa
- Most hemorrhoids appear in the following sites: ⓘ
  1. Right anterior (11 O'clock)
  2. Right posterior (7 O' clock)
  3. Left lateral (3 O' clock)
- Classification (according to their relation to the dentate line):
  1. **Internal** hemorrhoids → those originating above the dentate line
  2. **External** hemorrhoids → those originating below the dentate line

3.2 PATHOPHYSIOLOGY

- They represent engorgement or enlargement of the **normal fibrovascular cushions** lining the anal canal. (They're not varicosities)
- Chronic straining secondary to constipation or occasionally diarrhea
- Fibrovascular cushions lose their attachment to the underlying rectal wall
- Prolapse of internal hemorrhoidal tissue through the anal canal
- The overlying mucosa becomes more friable and the vasculature increases.
- With overlying thinning of the mucosa and vascular engorgement, subsequent rectal bleeding occurs

3.3 CLASSIFICATION

- **Internal** hemorrhoids are classified by **history** (level of prolapse) and not by physical examination ⓘ
  1. Grade 1 → bleeding without prolapse
  2. Grade 2 → prolapse with spontaneous reduction
  3. Grade 3 → prolapse with manual reduction
  4. Grade 4 → incarcerated, irreducible prolapse

3.4 SYMPTOMS

- Internal hemorrhoids
  - **Bright blood per rectum →** with or following bowel movements, is almost universally bright red, and very commonly drips into the toilet water
  - Blood may also be seen while wiping after defecation
  - **As a prolapsing anal mass →** prolapse usually occurs in association with a bowel movement, during walking or heavy lifting as a result of increased intra-abdominal pressure
  - Extreme pain, bleeding and occasionally signs of systemic illness in case of **strangulation**
- External hemorrhoid appears as a painful skin tag (necrotic old external hemorrhoid) that doesn't bleed or prolapses

3.5 PHYSICAL EXAMINATION:

- **Position**: left lateral decubitus (lithotomy) position

- **Inspection**:
  - Any rashes, condylomata, or eczematous lesions.
  - External sphincter function
  - Any abscesses, fissures or fistulae
- **Palpation**:
  - Gentle digital examination
  - Lubricated finger should be gently inserted into the anal canal while asking the patient to bear down
  - The resting tone of the anal canal should be ascertained as well as the voluntary contraction of the puborectalis and external anal sphincter.
  - Masses should be noted as well as any areas of tenderness. (Abdominal masses that may increase intraabdominal pressure)
  - Internal hemorrhoids are generally not palpable on digital examination.
- **Anoscopic examination:**
  - Anoscopy is mandatory to notice any impalpable mucosal changes
  - The side viewing anoscope should be inserted with the open portion in the right anterior then right posterior and finally the left lateral position
  - Hemorrhoidal bundles will appear as bulging mucosa and anoderm within the open portion of the anoscope.

3.6 EVALUATION OF RECTAL BLEEDING

- Must rule out cancer
- **Patients are divided into 2 categories:** ⓘ
  1. Low risk group
     - A young individual with bleeding associated with hemorrhoidal disease without other systemic symptoms and no family history → Anoscopy and rigid sigmoidoscopy (1st 25cm of rectum and colon)
  2. High risk group
     - An older individual, with either a family history of colorectal cancer, or change in bowel habits → complete colonoscopy should be performed to rule out proximal neoplasia

3.7 TREATMENT

- Varies from simple reassurance to operative hemorrhoidectomy.
- 90% of cases **conservative** only (constipation treatment and banding), 10% require **surgery**
- Treatments are classified into **three** categories:

1. Dietary and lifestyle modification.
2. Non-operative/office procedures.
3. Operative hemorrhoidectomy.

3.7.1 DIETARY AND LIFESTYLE MODIFICATIONS

- The main goal of this treatment is to minimize straining at stool.
- Achieved by increasing fluid and fiber in the diet, recommending exercise, and perhaps adding fiber agents to the diet such as psyllium.
- If necessary, stool softeners may be added.

3.7.2 OFFICE PROCEDURES

1. **Rubber band ligation**
   - For grade I or grade II hemorrhoids &, in some circumstances, Grade III
   - Complications include bleeding, pain, thrombosis and life threatening perineal sepsis.
   - Successful in two-thirds to three quarters of all individuals with first and second-degree hemorrhoids.
   - Only used for internal hemorrhoids
   - Never done in external ones because its very painful (so we just do an incision, evacuate the clot, and then close it under local anesthesia)
2. **Infrared coagulation**
   - Generates infrared radiation, which coagulates tissue protein and evaporates water from cells.
   - Most beneficial in Grade I and small Grade II hemorrhoids.
3. **Bicep electrocoagulation**
   - It works, in theory, similar to photocoagulation or to rubber banding.
   - The probe must be left in place for ten minutes.
   - Poor patient tolerance minimized the effect of this procedure.
4. **Sclerotherapy**
   - Injection of an irritating material into the submucosa in order to decrease vascularity and increase fibrosis.
   - Injecting agents have traditionally been phenol in oil, sodium morrhuate, or quinine urea.

3.7.3 SURGICAL TREATMENT OF HEMORRHOIDS HEMORRHOIDECTOMY

- The triangular shaped hemorrhoid is excised down to the underlying sphincter muscle, and the wound can be closed or left open
- Stapled hemorrhoidectomy has been developed as an alternative to standard hemorrhoidectomy

IN SUMMARY:

- ➢ Grade I and II → lifestyle modification
  - Diet: increase fiber intake and drink lots of water.
  - Supplement fibers and laxatives if he/she constipated
  - Never squeeze or strain
  - Office treatment
- ➢ Grade III → lifestyle modification with banding and if failed, do surgery
- ➢ Grade IV → Immediate surgical intervention

4 ANAL FISSURES

4.1 INTRODUCTION

- A tear in the anal canal extending from just below the dentate line to the anal verge.
- Most commonly in young and middle age adults.
- Cardinal symptom is **PAIN** during & for minutes to hours after defecation
- Bright red blood is common but minimal

Anal fissures are the most common cause of severe localized anorectal pain. They're almost always on the posteroanterior plane. Multiple fissures may be due to Crohn's disease.

- Over 90% of anal fissures are located in the **posterior** midline ⓘ
- Almost all the rest located in the anterior midline.
- The acute fissure is a "mere simple crack" in the anoderm.
- The chronic fissure appears with the following signs:
  1. Distal sentinel tag
  2. Proximal hypertrophied anal papilla
  3. Fibrotic edges of the fissure
  4. Exposed internal sphincter fibers in the base of the fissure

4.2 ETIOLOGY AND PATHOGENESIS

- The initiating factor is trauma, typically overstretching of the anoderm by a large hard stool (constipation)
- The proposed explanation for the posterior midline predominance is a lack of tissue support and maximal stretching at this site.
- Failure to heal is 2ndry to poor perfusion of the anoderm in the post midline.
- Posterior midline ischemia is the result of arterial anatomy and internal anal sphincter hypertonicity.

In simple words,

Constipation → straining → pressure → hypertrophy of the anal sphincter → increased pressure inside lumen → decreased blood supply → ischemia → traumatic bowel movement →sloughing → fissure

- Pain causes them not to want to defecate increasing constipation causing more physical trauma and a cycle occur

4.3 TREATMENT

- 90% of acute fissures settle with conservative management, in those that don't surgery can be done (a lateral internal sphinecterotomy)
- Correcting **constipation** (keeping bowel movements atraumatic)
  - Warm baths (sitz baths) and a high fiber diet to achieve soft bulky stools allow approximately 50% of acute anal fissures to heal within 3 weeks.
  - Stool softeners and fiber supplements are reasonable additions.
  - Recurrence is common, in the range of 30-70%, but can be reduced to 15-20% by maintaining a high fiber diet
- **ACUTE FISSURE**: (Topical application)
  1. Nitroglycerin
     - Topical application of nitroglycerin, a nitric oxide donor, causes a transient lowering of resting anal pressure (relaxing internal sphincter) and an increase in anodermal blood flow (vasodilator)
     - A 92% healing rate within 2 weeks for acute fissures treated with application of 0.2% glyceryl trinitrate ointment t.i.d.
  2. Calcium channel blockers
     - Topical calcium channel blockers (2% diltiazem, 0.3% nifedipine)
     - Heal 65-95% of fissures.
     - The most common side effects are headache, flushing, and symptomatic hypotension.

Topical application→ vasodilation→increase blood supply→help healing of fissure

- **CHRONIC FISSURE:**
  1. Topical Nitroglycerin: At eight weeks healing was observed in 68% of the GTN
  2. Botulinum Toxin: Botulinum toxin has been injected into the external and internal sphincters and, with short term follow up, healing rates of 80% have been achieved.
  3. Internal Sphincterotomy: lateral internal sphincterotomy (LIS) achieves healing in over 95% within several weeks

LIS: is based on cutting a small portion of the internal sphincter, relaxing the muscle and increasing blood supply to allow the fissure to heal, 5% risk of incontinence.

4. Anal dilatation
- Chronic fissures are unlikely to heal with warm baths & a high fiber diet.

5 MCQS

1. The commonest site for anal fissure is:
    a. anterior
    b. lateral
    c. posterior
    d. right posterior
2. The cardinal symptom of anal fissure is:
    a. bleeding
    b. itching
    c. pain
    d. skin tag
3. The anatomical location of hemorrhoids is:
    a. left anterior ,left posterior and right lateral
    b. left lateral, right anterior and right posterior
    c. right anterior, right posterior and left anterior
    d. right lateral, right anterior and left lateral
4. The following symptoms are common in anal problems, except:
    a. Perianal discharge
    b. Perianal itching and irritation
    c. Pain
    d. Nausea and vomiting
5. The proper treatment of a patient with a peri-anal abscess is;
    a. Incision and drainage as soon as fluctuation develops
    b. Incision and drainage with excision of the internal opening
    c. Prompt incision and drainage
    d. Use of antibiotics and sitz bath

🗝 **Answer key:** 1;C , 2;C , 3;B, 4;D, 5;C

COLORECTAL CANCER

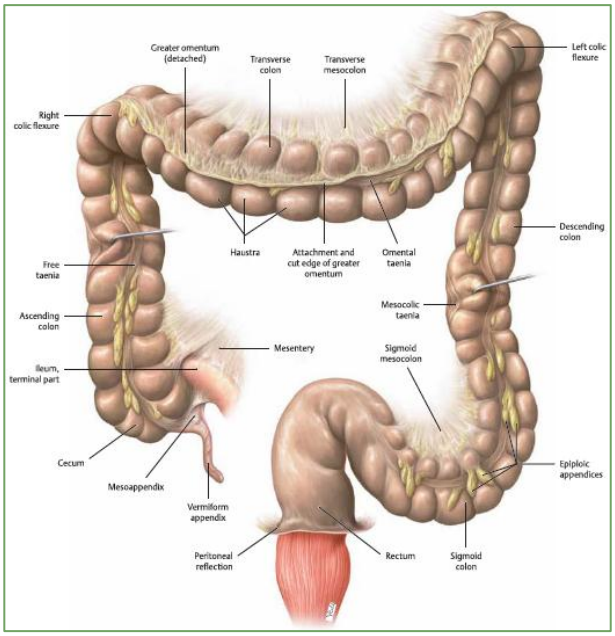
1 INTRODUCTION

1.1 DEFINITIONS:

- Colon = large bowel = large intestine
- Rectum - terminal portion of the colon
- Polyp: is a descriptive term used to describe any mass of tissue that bulges or projects outwards. Colonic polyps are mostly benign outgrowths.
- Adenoma - type of polyp and has chance to develop cancer but not all.
- Cancer - malignant growth; invasive (invades the basement membrane)
- Stage is an estimate to determine how large has the tumor grown.
- Primary - the original tumor, where it started.
- Metastases - where the tumor has spread to.

1.2 COLON AND RECTUM ANATOMY AND CANCER SIGNIFICANCE:

- The management and the characteristics of colon and rectal cancers are completely different
- **Cancer Development:**
  - Most cancers are acquired (sporadic), but some small percentage of cases arise from inherited diseases.
  - Most cancers begin as adenomatous polyps, however only a tiny percentage of adenomas become cancers (1 – 9% become malignant)



1.3 POLYPS:

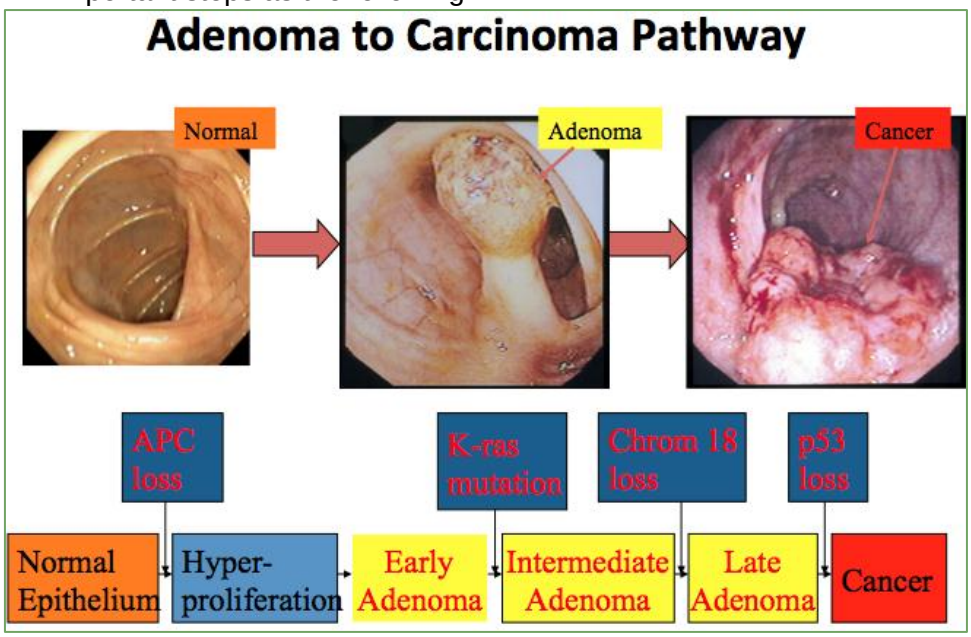
- **Non-neoplastic polyps:**
  - The majority of polyps are non-neoplastic accounting for more than 90% of polyps are benign.
  - These arise as a result of inflammation or improper maturation. These include:
    - Hyperplastic polyps (most commonly seen)
    - Hamartomatous polyps (Juvenile & Peutz-Jeghers polyps)
    - Inflammatory polyps
    - Lymphoid polyps
- **Neoplastic polyps:**

- Account for less than 10% of polyps and these are dysplastic polyps that have malignant potential.
- Adenoma

- **Adenomatous Polyps (adenomas):**
  - Occur mainly in large bowel.
  - Sporadic and familial
  - Vary from small pedunculated to large sessile.
  - Epithelium proliferation and **dysplysia**
  - Divided into:
    - Tubular adenoma: less than 25% villous architecture
    - Villous adenoma villous architecture over 50%
    - Tubulovillous adenoma: villous architecture between 25 and 50%.

1.4 CANCER SEQUENCE:

- The transformation from benign polyps to cancer takes from 7 - 10 years
- The transformation risk into cancer is based on:
  - Size of polyp
  - the histologic subtype of the polyp. They are organized in descending order for cancer development risk: Villus, Tubuloviilus, Tubular polyps
  - Severity of epithelial dysplasia
  - Number of polyps, with multiple polyps holding a greater risk of developing cancer
- The transformation from normal mucosa to cancer undergoes some important steps as the following:

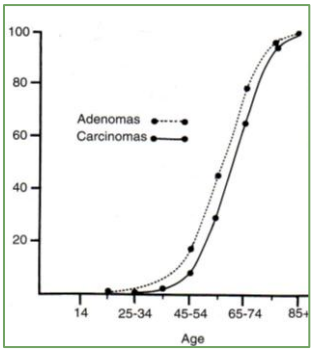


2 EPIDEMIOLOGY OF CRC:

- 3th most common malignancy worldwide.
- Most common in Saudi males.
- Second to lung cancer as a cause of cancer death
- 21,500 new cases, 8900 will die (2008)

- Risk of CRC – women 1/16 , men 1/14
- Median age of diagnosis was 60 according to the Saudi Cancer registry reports.

2.1 EFFECT OF AGE ON THE INCIDENCE OF COLORECTAL CANCER AND COLORECTAL POLYPS:



- As seen on the graph, the incidence of CRC increases tremendously after the age of 50.
- Colonoscopy is advised to be performed at the age of 50 for people with no significant risk factors, and should be performed at a younger age if the person has risk factors.
- Colonoscopy can detect and remove adenomas and thus prevent cancer occurrence.

2.2 CLASSIFICATION OF COLORECTAL CARCINOMA:

1. Adenocarcinoma (>95%)
2. Carcinoid
3. Lymphoma
4. Sarcoma
5. Squamous cell carcinoma

2.3 RISK FACTORS:

- Medical and Family history: (relative risks are extra info)
  - Hereditary colorectal cancer syndromes
  - Personal history: previous polyps (relative risk of 3.5 to 6), occurrence of previous CRC (relative risk of 2 in the first two years)
  - Family history: a first-degree family member doubles risk. Further detail to follow, so when a member of a family is diagnosed with colorectal cancer, it is recommended to screen other members at 10 years younger from their relative's age of diagnosis.
- Inflammatory bowel disease (mainly for cases of disease that extensively involve the colon and pancolitis, these conditions hold a relative risk of around 2.6 – 2.8)
- Other: Diet, nutrients, smoking, alcohol consumption

Hereditary colorectal cancer syndromes are a group of syndromes include hereditary non-polyposis colorectal cancer (HNPCC) syndrome and Familial adenomatous polyposis (FAP). In typical FAP, numerous colonic adenomas appear during childhood. Symptoms appear at a very early age and colonic cancer occurs in 90 percent of untreated individuals by age 45. These patients will have to undergo prophylactic colectomy.

2.4 COLORECTAL CANCER RISK BASED ON FAMILY HISTORY:

• General population " sporadic "	6%
• One 1st degree CRC	2-3X* (12-18%)
• Two 1st degree CRC	3-4X*
• One 1st degree CRC < 50 y	3-4*
• One 2nd or 3rd CRC	1.5X
• Two 2nd degree CRC	2-3X*
• One first degree with polyp	2X*

Hematochezia is more often caused by rectal than colon cancer. Iron deficiency anemia is from unrecognized blood loss and is more common with right sided CRCs and is frequently associated with a delayed diagnostic evaluation.

Abdominal pains is caused by partial obstruction, peritoneal dissemination, or intestinal perforation leading to generalized peritonitis.

3 CLINCAL PRESENTATION:

- Bleeding (melena/hematochezia) - gross, occult, anemia.

- Change in bowel habit – pain, diarrhea, constipation, alternating pattern
- Abdominal pains.
- Obstruction
- Change in caliber of the stools
- Weight loss
- Abdominal mass
- Asymptomatic.
- Some symptoms give clues on the location of the tumor:
- Sigmoid colon: obstruction and change in bowel habits.
- Rectum: bleeding and tenesmus
- Cecum: pain and melena
- Metastasis: weight loss.
- Notes on Clinical Presentation:
  - Symptoms of CRC are typically due to growth of the tumor into the lumen or adjacent structures. As a result, symptomatic presentation is often a manifestation of relatively advanced CRC.
  - In a series of Meta analyses: the previous first three symptoms were the most common upon presentation.
  - Sensitivity of individual symptoms for the diagnosis of CRC was poor, but Dr.AlKhayal mentioned it in the lecture and I thought I should add it

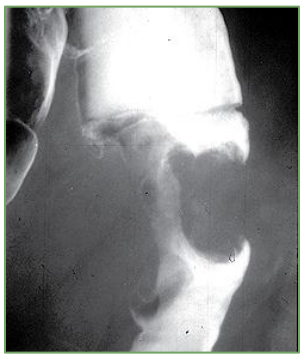
Obstruction is more common with left sided lesions, because fecal contents are liquid in the proximal colon and the lumen caliber is larger, and they are therefore less likely to be associated with obstructive symptoms. CRC is the most common cause of bowel obstruction in the elderly.

4 DIAGNOSIS

- General: Complete history and physical examination including a DRE
- Endoscopic: (identify primary, synchronous lesions)
- Flexible sigmoidoscopy
- Colonoscopy " to rule out other lesions"

4.1 STAGING:

- Endorectal ultrasound (rectal cancer)
- Chest x-ray (metastases)
- Liver ultrasound (metastases)
- Abdominal CT scan (metastases)
- Barium Enema: may show apple-core lesion as seen with this Double contrast barium enema of the descending colon.
- When colorectal cancer is diagnosed, it is almost protocol to perform CT scans of the chest, abdomen, and pelvis to detect or rule out any metastasis.



- Extra notes on Cancer spread:
  - CRC can spread by lymphatic and hematogenous dissemination, as well as by contiguous and transperitoneal routes.
  - The most common metastatic sites are the regional lymph nodes, liver, lungs, and peritoneum.
  - Because the venous drainage of the intestinal tract is via the portal system, the first site of hematogenous dissemination is usually liver, followed by lungs, bone, and many other sites, including brain.

Synchronous lesions: defined as two or more distinct primary tumors separated by normal bowel and not due to direct extension or metastasis. In other words: two or more cancers occurring at the same time.

ⓘ Colonoscopy " to rule out other lesions ": it is the most accurate diagnostic test in symptomatic individuals, since it can localize and biopsy lesions throughout the large bowel, detect synchronous neoplasms, and remove polyps.

- Tumors arising in the distal rectum may metastasize initially to the lungs because the inferior rectal vein drains into the inferior vena cava rather than into the portal venous system.

4.2 BLOODWORK

- CBC, electrolytes, and other function tests
- CEA (CarcinoEmbryonic Antigen) is a known protein molecule that is produced in high levels by CRC cells.
- It is not a specific marker, and can be elevated in many benign conditions like smoking! and other malignant cases like pancreatic cancer; therefore, can never be used as a screening test. However, CEA maybe used as a prognostic factor for evaluation of CRC management. ⓘ

5 THERAPY:

- Surgery is the most important variable in the treatment of colorectal cancer ⓘ
- Radiation and chemotherapy alone cannot cure any stage of colorectal cancer
- The site of tumor dictates the basic procedure

5.1 TREATMENT INDICATIONS FOR DIFFERENT STAGES ⓘ

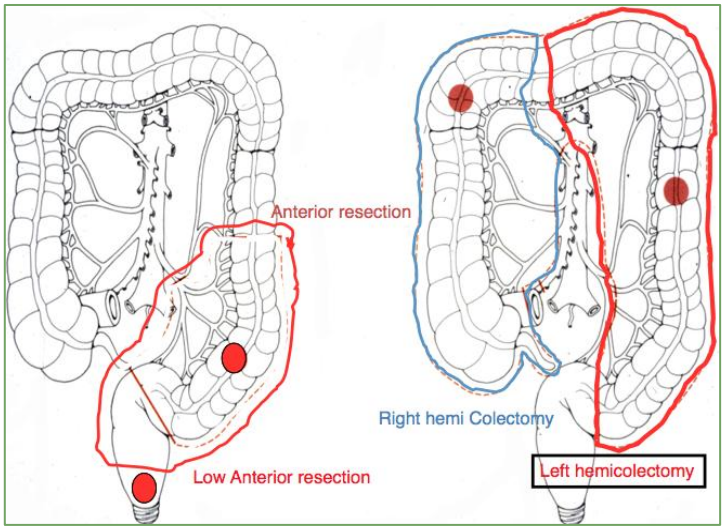
- Stage I and II: surgery
- High risk stage II and stage III: surgery + chemo/radiotherapy
- Stage IV: chemotherapy ± Surgery, depending on withier or not the tumor is resectable and on other factors.

5.2 PREOPERATIVE PREPARATION:

- Evaluation of medical problems. This is important especially for patients who have cardiopulmonary disease, as these patients must be evaluated by concerned specialists.ⓘ
- Mechanical bowel preparation (bowel cleansing by laxatives)
  - Colyte, Oral fleet
- IV antibiotics
- DVT prevention: Heparin shots, Compression stockings
- Foley catheter
- Epidural catheter

5.3 PRINCIPLES OF SURGERY:

- Examine the entire abdomen
- Remove the appropriate segment of the colon with adequate margins
- Remove the corresponding lymph nodes: a minimum of 12 lymph nodes have to be removed in a proper colectomy. ⓘ
- Open vs laparoscopic approach
- The types of colectomies can been seen on this figure:



5.4 OSTOMY INSERTION:

- The intestine is brought out through a hole in the abdominal wall
- Colostomy (colon on the skin)
- Permanent when the rectum is removed
- Temporary when it is unsafe to make a join
- Ileostomy (ileum on the skin)
- Temporary when the join needs time to heal

5.5 RECOVERY:

- Surgery 2 to 4 hours
- Hospital stay 4 to 10 days
  - IV, urine catheter, compression stockings, intravenous pain killers, blood thinner
  - Discharge when ambulating, eating, bowel function, good pain control
  - Recovery 4 weeks

5.6 FOLLOW UP:

- Office visit every 3 months for two years then every 6 months for 3 years
- Regular blood work (CEA)
- Colonoscopy at year 1 and 4 and every 5 years
- CT scan yearly
- **Some points on CEA:**
  - CEA is used to detect the prognosis: higher CEA levels indicate a worse prognosis.
  - It is used to detect recurrence: (CEA levels are usually around 2.5 – 5 ng/ml).
  - If CEA was 50, then after surgery it goes back to 5, then after some time it rises to 50 again. Here we suspect recurrence.
  - If CEA was 100 and after a surgery it is still 100 it can indicates 2 things A) There is another mass, i.e. metastasis and it hasn't been removed or B) the initial mass was not excised properly.

5.7 STAGING: ⓘ (VERY IMPORTANT)

- Staging of CRC is now achieved by using the TNM classification and not the modified Duke classification, as studies have shown that the 2010 modification of the TNM classification had better results. ⓘ
- How far into the wall has it grown?
  - T stage:
    - Tis – invasion of mucosa only
    - T1 – Invasion of submucosa
    - T2 – Invasion of muscularis propria
    - T3 – Full thickness/perirectal fat
    - T4 – Invasion into adjacent organs.
- Take note that adjacent organs does not mean distant metastasis, as that is a different component in the score. Adjacent organs mean structures like: the urinary bladder, uterus, and even the abdominal wall.
- 2. Is it growing in other places?
  - N stage: lymph node involvement, M stage: presence of metastasis
    - N1 – 1-3 lymph nodes
    - N2 - >4 lymph nodes
    - N3 – distant lymph nodes
    - M1 – Distant organ (mostly to the liver, lung)

5.8 TNM STAGING:

- Stage 0 – Tis tumors
  - Invasion of mucosa
- Stage 1 – T1 and T2 tumors
  - Invasion of sub mucosa & muscularis propria
- Stage 2 – T3 and T4 tumors
  - Invasion of full thickness & adjecent organ
- Stage 3 – Any lymph node involvement ⓘ
- Stage 4 – Distant metastases

5.9 WHO GETS ADDITIONAL THERAPY?

- COLON
  - All stage 3 patients (positive nodes) - chemotherapy
  - High risk stage 2 patients. ⓘThese patients include: Cancers with the mucinous subtype, patients with bowl obstructions; perforation, and who have undergone resection with less than 12 resected nodes.
- RECTUM
  - All stage 2 and stage 3 patients should get radiation and chemotherapy.
  - Note: in the rectum there are no serosa layer so the stage 2 patients should receive chemotherapy
- **Survival and TNM staging:**

STAGE	5-Year Survival
1	90%
2	80%^
3	27-69%*
4	8%

^for T3N0 tumors

*depends on # of nodes involved

6 SUMMARY:

- Common Cancer
- Can be prevented through screening and resection of polyps
- Surgery is the primary treatment
- Slow but steady improvement in survival

7 MCQS:

1) How should a patient who had Dukes C colon cancer two years previously be followed for recurrence of liver metastasis?

A. liver enzymes
B. CEA
C. U/S
D. CT
E. Radionuclide imaging

2) An 80-year-old man is admitted to the hospital complaining of nausea, abdominal pain, distention, and diarrhea. A cautiously performed transanal contrast study reveals an "apple core" configuration in the rectosigmoid. Appropriate management at this time would include:

a) Colonoscopic decompression and rectal tube placement
b) Saline enemas and digital disimpaction of fecal matter from the rectum
c) Colon resection and proximal colostomy
d) Oral administration of metronidazole and checking a *Clostridium difficile* titer
e) Evaluation of an electrocardiogram and obtaining an angiogram to evaluate for colonic mesenteric ischemia

3) Correct statements regarding carcinoembryonic antigen (CEA) and colorectal tumors include which of the following?

a) Elevated CEA is indicative of a tumor of gastrointestinal origin
b) A low CEA level after resection of a colon tumor is a poor marker of disease control
c) Ninety percent of colorectal tumors produce CEA
d) There is a high likelihood of liver involvement if the CEA level is high (greater than 100 ng/mL)
e) CEA levels are unusually low in cigarette smokers

4) A definite increased risk of colon cancer is associated with:

a) Diet high in fiber.
b) Diet low in animal fat and protein.
c) Diet low in fiber.
d) Ulcerative Colitis
e) Prior cholecystectomy.

5) A 52 year-old female diagnosed to have sigmoid cancer invading the uterus with no evidence of metastasis based on CT scan and colonoscopy, she underwent sigmoidoctomy and hysterectomy, the histopathology report

revealed invasive moderately differentiated adenocarcinoma involving the entire bowel wall and invading the myometrium, perinural and lymphovascular invasion, 6 out of 15 lymph nodes were positive for metastasis. The TNM classification for this patient will be:

a) T3 N1 M0
b) T3 N2 M1
c) T4 N2 M0
d) T4 N2 M1

6) For the above patient, she recovered well from surgery and visited you in clinic. The action that should be taken at this stage is?

a) Referral to medical oncology for adjuvant chemotherapy
b) Referral to radiation oncology for adjuvant radiotherapy
c) Referral to medical radiation oncology for both chemo-radiation
d) Referral to medical oncology for surveillance only

7) A 39 year-old male presented to the clinic for medical checkup because of significant family history of colon cancer, detailed family history revealed that three of his second degree relatives were diagnosed with colon cancer at age of 50, you counseled him for screening because:

a) He as average risk
b) He as double risk
c) He has 5 times greater risk
d) He has 10 times greater risk

8) Which one of the following factors is most likely to be associated with development of colorectal cancer?

a) Increase Calcium intake
b) Increase fat intake
c) Smoking
d) History of Colonic polyps

🗝 Answer key: 1;B , 2; C , 3;D , 4;D , 5;C , 6;C , 7; , 8;D

CHOLELITHIASIS

1 INTRODUCTION

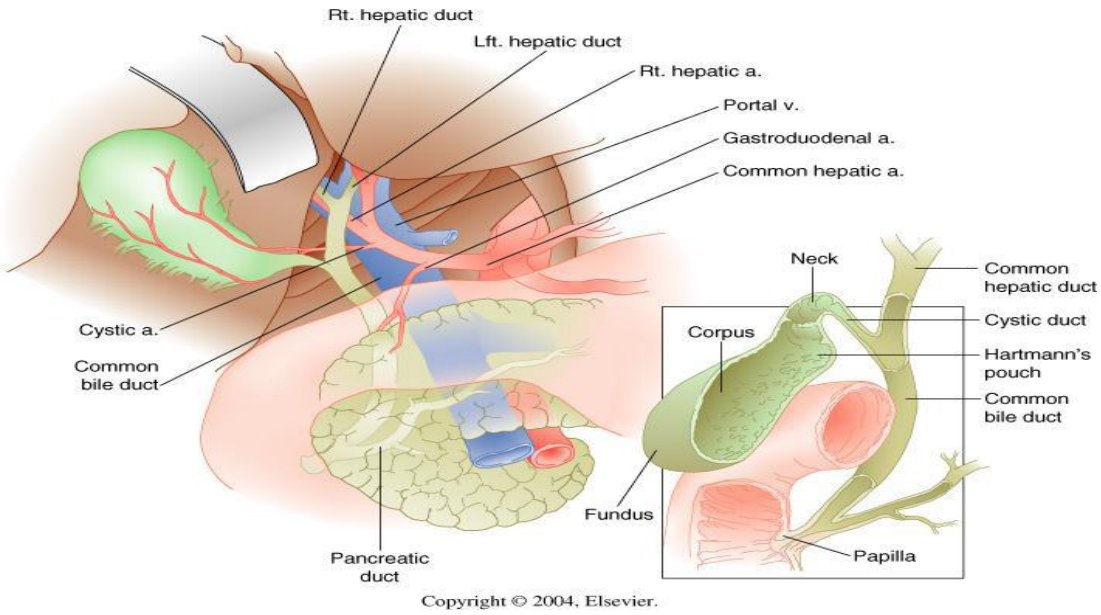
- Cholelithiasis is the presence of gallstones in the gallbladder.
- Presentation and complications:
  - May remain asymptomatic for decades.
  - May cause biliary colic type of pain 📄.
  - May lead to Cholangitis 📄
  - May lead to choledocholithiasis 📄
  - May lead to cholecystitis.

📄 Biliary colic is produced by migration of a gallstone into the opening of the cystic duct that may block the outflow of bile during gallbladder contraction. This results in increase in the gallbladder's wall tension and produces this pain.

📄 cholangitis is infection of the biliary tree.

📄 Choledocholithiasis is the presence of a gallstone in the common bile duct.

2 ANATOMY



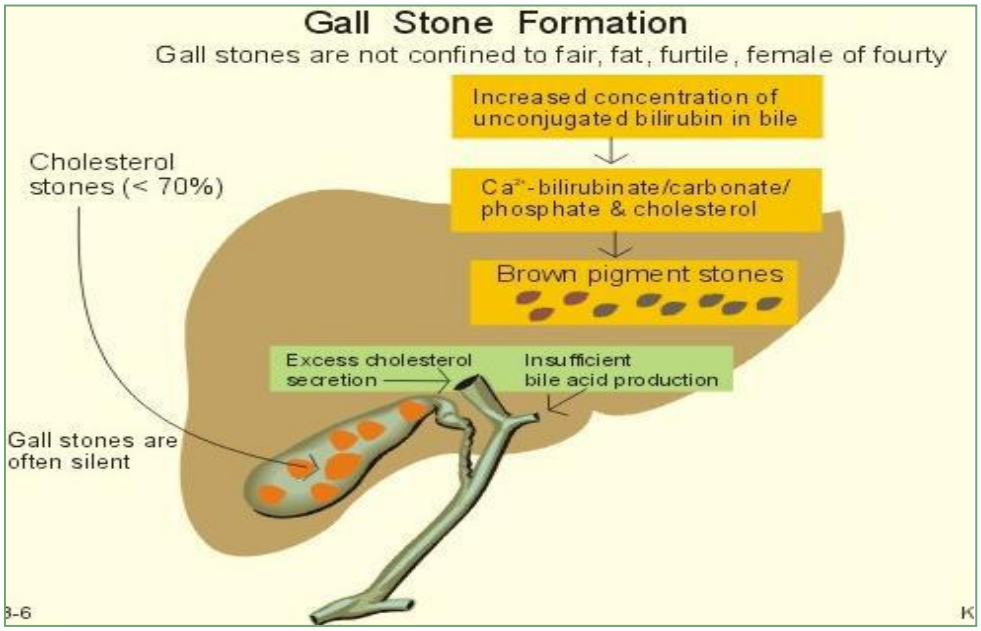
- The porta hepatis or hepatoduedenal ligament contains the portal vein, hepatic artery and bile. ⓘ

3 PATHOPHYSIOLOGY

- There are different types of stones: cholesterol stones which are the most common, pigment stones, and mixed.
- They are formed by crystallization of bile. The bile consists of lethicin, bile acids, and phospholipids in a fine balance.
- The solubility of cholesterol in bile depends on the concentration of lethicin, bile salts and cholesterol. Lethicin and cholesterol are insoluble aqueous solutions but dissolve in bile salt-lethicin micelles.
- Failure of the liver to maintain a micellar liquid can be caused by increase in the concentration of cholesterol or decrease in the
- Concentration of bile salts or lethicin; either way it can result in cholesterol stone formation.

📄 Infections result in an increase in billiary calcium as well as an increase in B-glucuronidase, which converts conjugated bilirubin to the unconjugated form. The calcium binds to unconjugated bilirubin and precipitate to form calcium bilirubinate stones.

- Conversely, increasing the biliary concentration of lethicin and bile salts should hinder cholesterol stone formation.
- Normal bile (normally 1 Liter/day) ⓘcontains glucaro-1,4-lactone, which inhibit the conversion of conjugated to unconjugated bilirubin, and thus stop the formation of calcium bilirubinate stones.
- Impaired motility can predispose to stones.
- Sludge is crystals without stones. It may be a first step in formation of stones, or independent of their formation. It can be found on Ultrasound.



- Pigment stones (15%):
  - Pure pigment (bilirubin) stones:
    - Associated with diseases that increase RBC destruction, such as sickle cell anemia or spherocytosis.
  - Calcium bilirubinate stones:
    - In cirrhotic patients, and parasitic infections 🗎.

4 EPIDEMIOLOGY, CAUSES AND RISK FACTORS

- US: affected by race, ethnicity, sex, medical conditions, fertility.
- 20 million people have cholelithiasis.
- Every year 1-2% of people develop them.
- Internationally: 20% of women get it and 14% of men.

4.1 RACE

- Highest in fair skinned people of Northern European descent and in Hispanic populations.
- High in Pima Indians (75% of elderly).
- Asians with stones are more likely to have pigmented stones than other populations.

4.2 AGE

- It is uncommon for children to have gallstones. If they do, it's more likely that they have congenital anomalies, biliary anomalies, or hemolytic pigment stones.
- Incidence of GS increases with age 1-3% per year.
- In patients over 60 years old, the prevalence of developing gallbladder stone in men is 12.9% and 22.4% in women.

4.3 GENDER

- More common in women. Etiology may be secondary to variations in estrogen causing increased cholesterol secretion, and progesterone causing bile stasis.
- Pregnant women are more likely to have symptoms.
- Women with multiple pregnancies are at higher risk.
- Women who are on oral contraceptives, or estrogen replacement treatment are at higher risk.
- To sum it up, these are the factors that cause and increase the risk of getting stones:
  - Fair skinned people
  - Females
  - Fertile
  - People on a high fat diet and obese people.
  - People with a family history.
  - Rapid weight loss, TPN (total parenteral nutrition), ileal disease, NPO
  - Old people.
  - People who drink alcohol.
  - Diabetics and hemolytics have more complications.

5 PRESENTATION

5.1 HISTORY

- There are 3 clinical stages:
  Asymptomatic, symptomatic, and with complications (cholecystitis, cholangitis, Common bile duct stones).
- Most cases (60-80%) are asymptomatic; such cases are discovered accidently by abdominal sonar.
- Every year 1-3% of patients develop symptoms.
- 60-80% of patients are asymptomatic, 40 -20% develop symptomats, around 20% of the Symptomatic patients will develop complications.
- Most patients develop symptoms before complications but sometimes the patient might develop the complications without having any previous symptoms.
- Once symptoms occur, severe symptoms develop in 3-9% of the cases, with complications in 1-3% per year, and a cholecystectomy rate of 3-8% per year.
- Patients who have small stones are more prone to develop symptoms.
- Asymptomatic GS are not associated with fatalities.

Differential diagnosis:

- AAA (abdominal aortic aneurysm)
- Appendicitis
- Cholangitis, cholelithiasis
- Diverticulitis
- Gastroenteritis, hepatitis
- IBD, MI, SBO (small bowl obstruction)
- Pancreatitis, renal colic, pneumonia

- Morbidity and mortality are associated only with symptomatic patients.
- Symptoms include :
  - Severe Epigastric colicky pain, located in the Right upper quadrant, that lasts for 1 to 5 hours, and wakes the patient up from his sleep at night.
  - Classically the pain is in the Right upper quadrant, however visceral pain and gallbladder wall distension may be only in the epigastric area.
  - A gallstone may impact in the neck of gall bladder or in the cystic duct giving biliary pain or cholecystitis >> Biliary pain usually occurs in the epigastrium and right hypochondrium (RUQ).
  - Other symptoms are related to the site of movement of the stone.
- Indigestion, bloating, and fatty food intolerance occur in similar frequencies in patients without gallstones, and are not cured with cholecystectomy.

5.2 PHYSICAL EXAMINATION

- Vital signs and physical findings in asymptomatic cholelithiasis are completely normal.
- Fever, tachycardia, Murphy's sign and hypotension alert you to more serious infections, including cholangitis, cholecystitis.

Murphy's sign: A sign of gallbladder diseases consisting of pain on taking a deep breath when the examiner's fingers are on approximate location of the gallbladder.

6 INVESTIGATIONS

6.1 LABORATORY STUDIES

- Lab results in asymptomatic patients and patients with biliary colic should be normal.
- WBC, elevated LFTS may be helpful in diagnosis of acute cholecystitis, but normal values do not rule it out.
- Elevated WBC is expected but not reliable.
- ALT, AST, AP more suggestive of CBD stones
- Amylase elevation may be GS pancreatitis

6.2 IMAGING STUDIES

- U/S and Hida are the best. Plain x-rays, CT scans ERCP are adjuncts.
- X-rays:
  - 15% stones are radiopaque, porcelain GB may be seen.
  - Will show air in biliary tree and emphysematous GB wall.
- CT: IT IS THE BEST IMAGING EXAMINATION:
  - Used for complications, ductal dilatation, surrounding organs.
  - Misses 20% of GS.
  - Done if diagnosis is uncertain.
- Ultrasound: IT IS THE FIRST IMAGING TEST YOU DO:
  - It is 95% sensitive for stones
  - It is 80% specific for cholecystitis.
  - It is 98% sensitive and specific for simple stones.
  - Sometimes it might show Wall thickening (2-4mm), might be false positives!
  - Distension
  - Pericholecystic fluid, sonographic Murphy's.

A Study examined the utility of labs with cholethiasis diagnosed with HIDA, and showed no difference in WBC, AST, ALT Bili, & Alk Phos, in patients diagnosed & those without.

In a retrospective study, only 60% of patients with cholecytitis had a WBC greater than 11,000. A WBC greater than 15,000 may indicate perforation or gangrene.

  - If it showed Dilated CBD (7-8mm) , this indicates the presence of an obstruction.
- Hida scan:
  - Documents cystic duct patency.
  - 94% sensitive, 85% specific
  - GB should be visualized in 30 min.
  - If GB is visualized later, it may point to chronic cholecystitis.
  - CBD obstruction appears as non visualization of small intestine.
  - False positives, high bilirubin.
- ERCP (Endoscopic retrograde cholangiopancreatography):
  - ERCP is diagnostic and therapeutic.
  - It provides radiographic and endoscopic visualization of biliary tree.
  - Done when CBD is dilated and LFTs are elevated.
  - Complications include bleeding, perforation, pancreatitis, and cholangitis.
  - **ERCP needs: endoscope + fluoroscopy (X-ray and contrast and guide wire)** ⓘ
  - **If a patient presented with jaundice, you admit him for ERCP**. ⓘ

7 COMPLICATIONS

7.1 CHOLYCYSTITIS

- It's an inflammation of the gallbladder secondary to calculi.
- Characterized by:
  - Continuous pain.
  - Fever.
  - High WBC count due to inflammation.
  - Murphy's sign on examination.
  - Distended gall bladder and thickening of the wall on Ultrasound due to inflammation.
- How to manage this patient?
  - The patient should be admitted to the hospital
  - Stabilized
  - Given IV antibiotics and analgesics.
  - If the patient responded to the treatment, elective cholecystectomy is done after 6 weeks so that the inflammatory process cools down.
  - Urgent surgery is required only if the patient did not respond to the treatment because gangrenous cholecystits may develop📄.

📄 Gangrenous cholecystitis is the most common complication of cholecystitis, particularly in older patients, diabetics, or those who delay seeking therapy. Associated inflammation leads to ischemic necrosis of the wall, with or without associated cystic artery thrombosis.

7.2 OBSTRUCTIVE JAUNDICE

- When obstructive jaundice occurs it means that one of the stones moved down to the common bile duct and caused an obstruction which will obstruct the flow of bile from the liver to the small bowel.
- It can also be a mass that's causing the obstruction.

📄 Stones that block the ampulla of Vater may block pancreatic secretions and predispose the patient to Pancreatitis, as gall stones are the most important risk factor for Pancreatitis

7.2.1 SYMPTOMS AND SIGNS

- **Jaundice:**
  - Look for it in the sclera (specially dark skinned people and during the sun light), skin and mucosa
  - The bilirubin level in the blood is at least double the normal (Upper normal level is 17 mmol)
- **Pale stool**
- **Dark urine**
- **Itching** (due to accumulation of bile salts under the skin)

The whiter your skin is the less bilirubin you need to develop jaundice.
The darker your skin is the more bilirubin you need to develop iaundice.

7.2.2 DIFFERENTIAL DIAGNOSIS

- Painless obstructive jaundice with significant weight loss might be cancer (obstruction develops gradually)
  It could be:
  - Head of pancreas cancer
  - ampulla of Vater cancer
  - Distal CBD cancer

When you take history of a patient with jaundice it is important to ask about eye or skin discoloration, pale stool, dark urine, any itching.

7.2.3 MANAGEMENT

- Stone removal:
  - To relieve obstructive jaundice: admit the patient for ERCP: First shpincterotomy is done to widen the diameter of the sphincter of Oddi then take the stone out by the basket.
  - After doing ERCP we wait for one day before removing the gall bladder (cholecystectomy) to make sure the patient didn't develop pancreatitis.

Both benign tumors and hamartomas are composed of normal cells in excessive quantities, but benign tumors have a normal arrangement whereas hamartomas have an abnormal arrangement of cells.

In involuting hemangioma, the deeper they go the bluer they become, whereas the more superficial the more cherry red they get.

7.3 CHOLYNGITIS

- It's the inflammation of biliary tree.
- Charcot's triad: (it's diagnostic) ⓘ
  1. Fever
  2. Jaundice
  3. Right upper quadrant pain RUQ
- If patient was hypotensive→ send him to the ICU for ionotrops ⓘ
- Treatment: ERCP

7.4 OTHERS

- Sepsis
- **Pancreatitis**
- Perforation (10%)
- GS ileus (mortality 20% as diagnosis difficult).
- Hepatitis
- Choledocholithiasis

8 MANAGEMENT

- Historically cholecystits was operated on emergently which increased mortality.
- Surgical consult is appropriate, and depending on the institution, either medicine or surgery may admit the patients for care.
- Get GI doctor involved early if suspect CBD obstruction

The severity of signs and symptoms /complications determine if this patient should do the surgery electively or operate now.

8.1 EMERGENCY DEPARTMENT CARE

- GB colic is suspected in patients with RUQ pain of less than 4-6h duration and radiating to back.
- Acute cholecystits is suspected in those with longer duration of pain, with or without fever. Elderly and diabetics should be diagnosed as soon as possible because it might proceed to sepsis.
- If a patient presents with acute cholycysitis and hypotension, he must be admitted to the ICU and given ionotrops and then the obstruction must be relieved. ⓘ
- After assessment of ABCs, standard IV, pulse oximetry, EKG, and monitoring must be performed. Also, culture should be included if the patient is febrile.
- The primary goal of the emergency department care is to diagnose acute cholecystitis with labs, US, and or Hida. Once diagnosed, hospitalization is usually necessary. Although Some are treated as OP.
- In patients who are unstable or in severe pain, a bedside US should be considered to exclude AAA and to asses in diagnosing acute cholecystitis.
- Volume should be replaced with IVF, NPO, +/- NGT.
- Administer pain control early. A courtesy call to surgery may give them time to examine without narcotics.

Patients with acute cholycystitis →Admit

→Evaluate patient

-Pain for 24-48 hours : operate

-Pain for more than 48 hours → IV antibiotics→Stabilize the patient → then operate

-Why? Because operating on a patient while the gallbladder is acutely inflamed has been shown to have more complications.

8.2 MEDICATIONS

- Anticholinergics such as Bentyl (dicyclomine hydrochloride) to decrease GB and biliary tree tone. (20mg IM q4-6).
- Demerol 25-75mg IV/IM q3
- Antiemetics (phenergan, compazine).
- Antibiotics (Zosyn 3.375g IV q6) need to cover Ecoli (39%), Klebsiella(54%), Enterobacter(34%), enterococci, group D strep.

8.3 FURTHER INPATIENT CARE

- Cholecystectomy can be performed after the first 24-48h or after the inflammation has subsided. Unstable patients may need more urgent interventions with ERCP, percutaneous drainage, or cholecystectomy. ⓘ
- Laparoscopic cholecystectomy is very effective but it has few complications (4%). 5% convert to open. In acute setting up to 50% open.

8.4 FURTHER OUTPATIENT CARE

- Afebrile, normal VS

- Minimal pain and tenderness.
- No markedly abnormal labs, normal CBD, no pericholecystic fluid.
- No underlying medical problems.
- Next day follow-up visit.
- Discharge on oral antibiotics, pain meds

9 PROGNOSIS

- Uncomplicated cholecystitis has a low mortality.
- The mortality rate for emphysematous GB is 15%
- Perforation of GB occurs in 3-15% of patients with a mortality rate up to 60%
- Gangrenous GB has a mortality rate up to 25%

10 MCQS

1. **A 73-year old previously healthy man presents to the emergency room with several days of jaundice followed by 12 hours of RUQ pain and fever. He is mildly hypotensive. CT scan of the abdomen revealed dilation of the biliary tree.**

· What is the most likely diagnosis?
Answer: Cholyangitis

· Management includes which of the following?
A. Laproscopic cholecystectomy.
B. Open cholecystectomy and T tube replacement.
C. Open cholecystectomy and choledochojejunostomy.
D. Fluid resuscitation, antibiotics, and ERCP.
E. Fluid resuscitation and hepatitis serologies.

2. **What is the most common cause of chronic pancreatitis?**
A. Gall stones
B. Alcohol
Answer: A (80%) gall stones is the cause either in acute or chronic pancreatitis.

3. **How much bile is produced by the liver?**
A. 100 – 200 ml/day
B. 300-400 ml/day
C. 500-1000 ml/day (1Liter)

4. **In Endoscopic ERCP, stone extraction from the common bile duct (CBD) is NOT possible in all of the following except:**
A. Multiple stones in CBD
B. Intrahepatic stone
C. Multiple gall stones
D. Pt has CBD stone with prior gastrectomy

5. **What is the correct procedure to do in the following cases?**
   - A patient had CBD stones but he had prior gastrectomy (or post gastric resection B2 “Billroth's operation II”).
     Answer: No gastric --> we cannot reach the duodenum --> never do ERCP So open the abdomen --> open the CBD and extract the stones (intraoperative cholangiogram).
   - Patient had post gastric resection B1 “Billroth's operation I”?
     Answer: We can do ERCP because duodenum is still open

Q5:

B1: an operation in which the pylorus is removed and the distal stomach is anastomosed directly to the duodenum.

B2: an operation in which the greater curvature of the stomach is connected to the first part of the jejunum in a side-to-side manner. This often follows resection of the lower part of the stomach (antrum). The antrectomy (resection of the stomach antrum) is not part of the originally described procedure. The surgical procedure is called gastrojejunostomy.

6. **A patient presented to the ER with abdominal pain, elevated WBCs and increased serum amylase.**
   What is the most likely diagnosis?
   Answer: Acute pancreatitis

7. **A 45 Y/O obese female with cholelithiasis, presents to the ER complaining of N/V & severe continuous abdominal pain, high grade fever, slightly elevated WBC (12,000), & increased serum amylase.**
   What is the most likely Diagnosis?
   Answer: Acute Pancreatitis

8. **A 70 years old male came with progressive painless jaundice.**
   What are the Differential diagnosis?
   Answer: It could be:
   - Head of pancreas cancer
   - ampulla of Vater cancer
   - Distal CBD cancer

9. **A 70 years old male with progressive painless jaundice is referred to your clinic. You order LFT that shows abnormal pattern of obstruction jaundice, US shows dilated CBD 2 cm.**
   Which procedure do you suggest?
   A. ERCP
   B. Laparoscopic Cholecystectomy
   C. Modified barium swallow
   D. Laparoscopic abdominal exploration
   E. Upper GI endoscopy

10. **A patient came to you complaining of chronic nausea and mild right upper quadrant pain; you suspect the cause of his symptoms is gall stones.**
    What is the first image study in this case?
    Answer: US
    What is the **best** image study in this case?
    Answer: CT

11. Which of the following structures is in not found in the hepatodeudenal ligament?
A. Hepatic vein
B. Hepatic artery
C. CBD

12. Which of the following is not an ultrasonic finding in acute cholecystitis?
A. Sonographic Murphy's sign
B. Pericholecystic fluid
C. Gall bladder wall thickening more than 6 mm
D. Absence of gall stones

13. Signs and symptoms of acute cholecystitis usually include all of the following except:
A. Jaundice
B. RUQ pain
C. Fever
D. Elevated WBC count
E. Nausea and vomiting

14. In obstructive jaundice, LFTs usually shows:
A. Elevated indirect bilirubin and alkaline phosphatase
B. Elevated indirect bilirubin and GGT
C. Elevated direct bilirubin and alkaline phosphatase
D. Elevated direct bilirubin and ALT
E. Elevated direct bilirubin and AST

15. Prolonged PT (INR) in obstructive jaundice is due to decreased absorption of:
A. Vitamin A
B. Vitamin D
C. Vitamin E
D. Vitamin K
E. Calcium

16. In acute cholecystitis, HIDA scan shows
A. Distended gallbladder
B. Contracted gallbladder
C. Non-filling of gallbladder
D. Dilated common bile duct
E. Bile leak

17. Bile contains all of the following except:
A. Bile salts
B. CCK
C. Bile pigments
D. Cholesterol
E. Phospholipids

18. Bile secretion is increased by:

A. Vagus
B. Fasting
C. Sympathetic stimulation
D. Adrenaline
E. Octreotides

19. Risk factors for gallstones include all of the following except:

A. Obesity
B. Contraceptive pills
C. Sickle cell anemia
D. High protein diet
E. Rapid weight loss

20. Which of the following can be diagnostic and therapeutic for common bile duct stones:

A. US
B. CT scan
C. HIDA scan
D. ERCP
E. MRCP

21. A 25 Years old lady presented to ER with 2 days history of right upper quadrant pain and fever. She has no Murphy's sign and WBC count is 7. The best management will be

A. PO Analgesia
B. IV analgesia
C. Admission and start IV antibiotics
D. Admission and start PO antibiotics
E. IV antibiotics and follow up in clinic

22. The following are indications for cholecystectomy in asymptomatic gall bladder stone patients except:

A. Diabetes
B. During surgery
C. Stone 4 cm in size
D. Ischemic heart disease
E. Hemolytic anemia

🗝 Answer Key: 1=D, 2=A, 3=C, 4=A, 9=A, 11=A, 12=D, 13=A, 14=C, 15=D, 16=C, 17=B, 18=A, 19=D, 20=D, 21=C, 22=D

PORTAL HYPERTENSION

1 INTRODUCTION

Portal hypertension (defined as hydrostatic pressure >5 mmHg) results initially from obstruction to portal venous outflow. Obstruction may occur at a presinusoidal (portal vein thrombosis, portal fibrosis, or infiltrative lesions), sinusoidal (cirrhosis), or postsinusoidal (veno-occlusive disease, Budd Chiari syndrome) level. Cirrhosis is the most common cause of portal hypertension; in these patients, elevated portal pressure results from both increased resistance to outflow through distorted hepatic sinusoids, and enhanced portal inflow due to splanchnic arteriolar vasodilation.

1.1 CAUSES:

Causes of Portal hypertension can be classified as:

- Cirrhotic (definition)
- Non-cirrhotic: most important non-cirrohtic causes are: shistosomaiasis and splenic vein thrombosis (mainly caused by hypercoaguable state and pancreatitis)

1.2 SYMPTOMS OF PORTAL HYPERTENSION

- Asymptomatic: portal hypertension is asymptomatic until complications develop, where patients present according to the ongoing pathological process. These complications are in the form of:
- Gastroesophageal varices
- Ascites
- Splenomegaly: can sometimes cause dull abdominal pain.
- Underlying disease

2 VARECIAL BLEEDING

- Approximately one-third of all patients with varices will develop variceal hemorrhage
- A major cause of morbidity and mortality in patients with cirrhosis. Veins don't have much smooth muscles and as a result do not go into spasm once they bleed. With this lack of smooth muscle and engorgement of the esophageal veins with, varecies tend to bleed profoundly, when they rupture.

2.1 PREVENTION OF VARECIAL BLEED

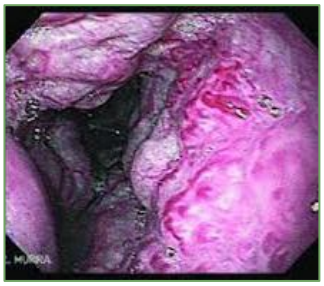
- AASLD RECOMMENDATIONS — Recommendations for prevention of variceal bleeding have been issued by the American Association for the Study of Liver Diseases
- These Recommendations are as follows:

ⓘ No treatment is given to people who haven't developed Cirrhosis.

Varices develop in order to decompress the hypertensive portal vein and return blood to the systemic circulation. They are seen when the pressure gradient between the portal and hepatic veins rises above 12 mmHg; patients with lower values do not form varices and do not bleed.

The risk of Esophgeal Varecies development can be predicted by the Child-Pugh score, calculated by computing different values for certain conditions like: presence of Ascites, encephalopathy, bilirubin and albumin levels, and other factors.

Red wale signs are longitudinal red streaks seen in endoscopies on varices that resemble red corduroy wales. See below>



- In patients who have compensated cirrhosis and small varices that have not bled but have criteria for increased risk of hemorrhage (Child B/C or presence of red wale marks on varices), nonselective beta blockers
- In patients with medium/large varices that have not bled, nonselective beta blockers (propranolol or nadolol) is recommended or undergo EVL
- In patients who receive beta-blockers, a follow-up EGD is not necessary.
- If a patient is treated with EVL, it should be repeated until the varices are obliterated. EGD should performed one to three months after obliteration and then every 6 to 12 months to check for variceal recurrence.

3 TREATMENT OF ACTIVE VARECIAL BLEED

3.1 PRIMARY GOALS

There are three primary goals of management during the active bleeding episode:

1. ⓘ ABCs, especially hemodynamic resuscitation: this is achieved by two large bore peripheral lines, where fluids or blood should be administered. In some cases, clotting factors/platelets might be needed due to the massive blood transfusion and exhausted clotting factors/platelets.
2. Prevention and treatment of complications
   - Prophylactic antibiotics, preferably before endoscopy (although effectiveness has also been demonstrated when given after). Suggest intravenous ceftriaxone (1 g IV) or Cipro (400 mg IV BID) 📄 About 20% of patients with varecial bleeding will have a infection. Most commonly a UTI, but other more serious conditions like a respiratory infection or peritonitis may develop.
3. Arresting Varecial bleeding:
   - Vasoactive substances: Suggest terlipressin in countries where it is available and somatostatin or octreotide (50 mcg bolus followed by 50 mcg/hour by intravenous infusion) where terlipressin is unavailable.
   - ⓘ Endoscopic treatment: is the treatment of choice, where it can be diagnostic and also therapeutic. Endoscopic therapy can either be Endoscopic variceal ligation (EVL) or Endoscopic scelotherapy.
   - If the patient's bleeding is still not controlled, Surgery mostly in the form of TIPS is usually performed.

3.2 SALVAGE TREATMENT

- ⓘTIPS (transjugular intrahepatic portosystemic shunt) <u>used primarily as a salvage therapy in patients with recurrent variceal bleeding</u> despite an adequate trial of endoscopic and pharmacologic treatment (usually defined as two failed attempts of endoscopic treatment)
- The best candidates for surgery are patients with well preserved liver function who fail emergent endoscopic treatment and have no complications from the bleeding or endoscopy.
- The choice of surgery usually depends upon the availability, training, and expertise of the surgeon.

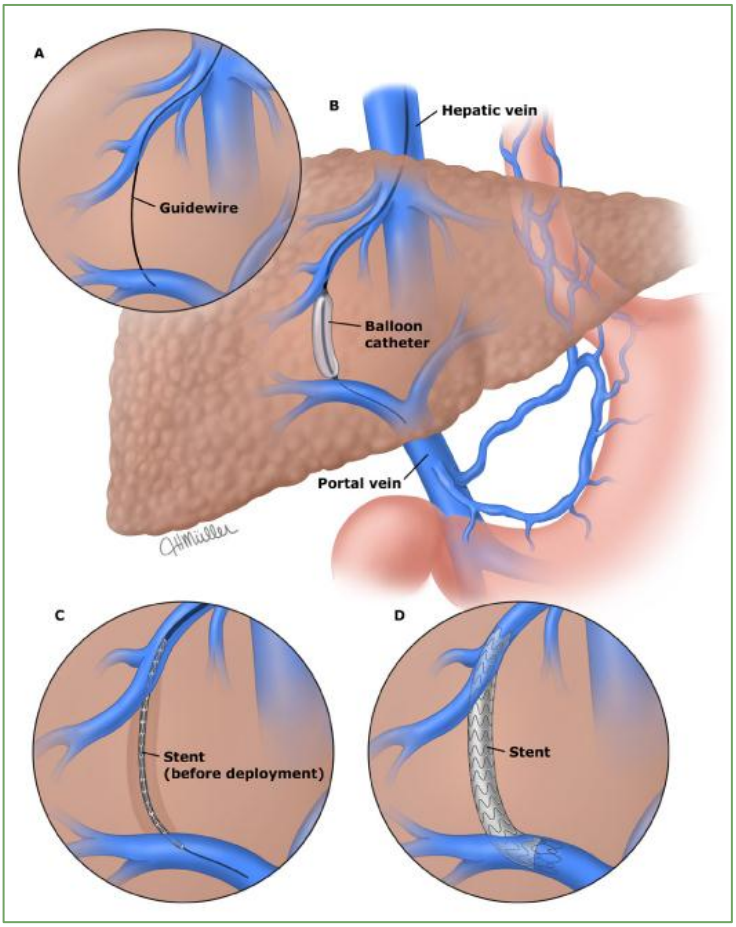
📄 The principal complications that cause death are aspiration pneumonia, sepsis (antibiotic administration), acute-on-chronic liver failure, hepatic encephalopathy (lactulose and treatment of other precipitating factors), and renal failure (careful fluid balancing and avoid giving nephrotoxic substances)

📄 Vasoactive substances agents directly constricts mesenteric arterioles and decreases portal venous inflow, thereby reducing portal pressure. However, Terlipressin is the only pharmacological agent shown to reduce mortality in compared to placebo.

📄 EVL should be performed as soon as possible. It involves the placement of rubber bands around a portion of oesophageal mucosa that contains the varix. EVL is superior to sclerotherapy in general, but sclerotherapy maybe used in cases when esophageal visualization is limited due the bleeding mainly because scleortherapy is quicker and provides better visualization of the esophagus.

- Although a selective shunt has some physiologic advantages, it may significantly exacerbate marked ascites. Thus, a portacaval shunt would be preferable in patients with marked ascites

3.3 PORTAL HYPERTENSION OPERATIONS



A. Shunt Surgery:

Definition: Transjugular intrahepatic portosystemic shunts (TIPS) involve creation of a low-resistance channel between the hepatic vein and the intrahepatic portion of the portal vein (usually the right branch) using angiographic techniques. The tract is kept patent by deployment of an expandable metal stent across it, thereby allowing blood to return to the systemic circulation. Portosystemic shunts are classified as nonselective, selective, and partial, depending on how much hepatic portal flow is preserved.

- Types of shunts:
  1. Nonselective — those that decompress the entire portal tree, such as portacaval shunts
  2. Selective — those that compartmentalize the portal tree into a decompressed variceal system while maintaining sinusoidal

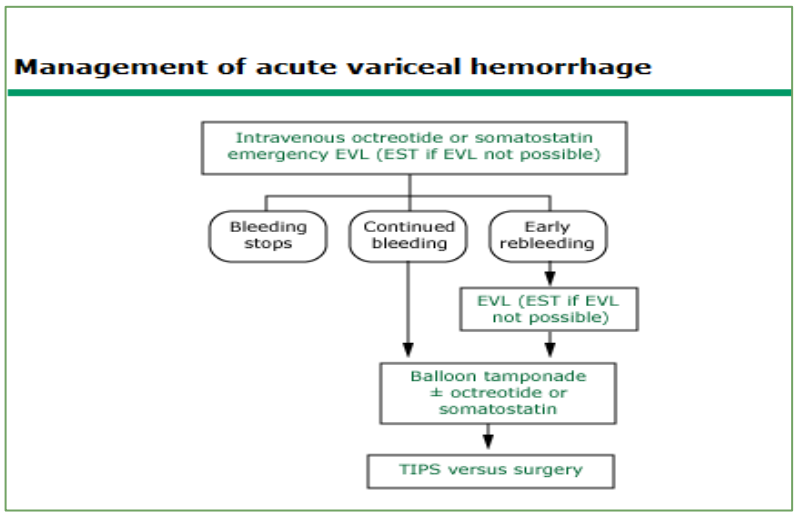
perfusion via a hypertensive superior mesenteric-portal compartment, such as a distal splenorenal shunt

3. Partial — those that incompletely decompress the entire portal tree and thereby also maintain some hepatic perfusion

B. Nonshunt operations generally include either esophageal transection (in which the distal esophagus is transected and then stapled back together after varices have been ligated) or devascularization of the gastroesophageal junction (Sugiura procedure).

3.4 GENERAL OUTLINE OF VARECIAL BLEEDING TREATMENT

- Maintain a hemoglobin level of approximately 8 g/dL. A threshold above this may actually increase mortality.
- Pharmacologic therapy (somatostatin or its analogue octreotide) should start as soon as bleeding is suspected and continue for 3-5 days after confirmation.
- Short-term (maximum seven days) antibiotic prophylaxis should be instituted in any patient with cirrhosis and GI hemorrhage.
- Upper endoscopy, performed within 12 hours, should be used to make the diagnosis and to treat variceal hemorrhage either with endoscopic variceal ligation or sclerotherapy.
- TIPS is indicated in patients in whom hemorrhage from esophageal varices cannot be controlled or in whom bleeding recurs despite combined pharmacological and endoscopic therapy.
- Balloon tamponade should be used as a temporizing measure (maximum 24 hours) in patients with uncontrollable bleeding for whom a more definitive therapy (eg, TIPS or endoscopic therapy) is planned.
- ⓘ Note: Dr.Mazen mentioned that Proton pump inhibitors should be added even in cases with known Liver disease. The rationale behind this is A) that peptic ulcer bleeding hasn't been ruled out and B) decreased acid secretion may help in the healing of these gastrointestinal bleeds. I did not know where to add this point, so I did here.



2 ASCITES:

- Cirrhosis is the most common cause of ascites in the United States, accounting for approximately 8%5
- Ascites is the most common complication of cirrhosis
- Fluid leaks from the surface of the liver and intestine.
- Factors responsible: portal hypertension, decreased ability of the blood vessels to retain fluid, fluid retention by the kidneys, and alterations in various hormones and chemicals that regulate bodily fluids

2.1 TREATMENT OF ASCITES:

- Dietary sodium restriction is a central component, 2000 mg / day
- Patients should be instructed to avoid NSAIDs, which can cause sodium retention and affect renal function
- Fluid restriction is equivocal and not strongly recommended
- Diuretic therapy, a single morning oral doses of spironolactone and furosemide, beginning with 100 mg and 40 mg
- Serial therapeutic paracentesis and TIPS are usually reserved for patients with refractory ascites.
- Peritoneovenous shunts (LeVeen or Denver) or surgical portosystemic shunts have very limited indications

2.2 COMPLICATIONS OF ASCITES:

- Spontaneous bacterial peritonitis: (SBP) is an infection of preexisting ascitic fluid without evidence for an intra-abdominal secondary source such as a perforated viscus
- The diagnosis is established by:
    - positive ascitic fluid bacterial culture, and/or
    - elevated ascitic fluid absolute polymorphonuclear leukocyte (PMN) count (≥250 cells/mm3)

3 PORTAL VEIN THROMBOSIS

Causes:

- Can be picked up Ultrasound with Doppler flow studies, CT scanning, and magnetic resonance angiography
- UGD should be performed to establish wither varices are present
- In cases of detected acute thrombosis (e.g. pancreatitis) Anticoagulation therapy for at least three months starting with low molecular weight heparin and shifting to oral anticoagulation as soon as the patient's condition has stabilized.
- Anticoagulation should be continued long-term in patients with acute portal vein thrombosis who have a permanent thrombotic risk factor that is not correctable.

Causes of portal vein thrombosis
Abdominal sepsis
Behcet's disease
Cirrhosis
Collagen vascular diseases (eg, lupus)
Compression or invasion of the portal vein by tumor (eg, pancreatic cancer)
Endoscopic sclerotherapy
Factor V Leiden
Hepatocellular carcinoma
Hyperhomocysteinemia
Inflammatory bowel disease
Myeloproliferative syndromes
Omphalitis
Oral contraceptives
Pancreatitis
Paroxysmal nocturnal hemoglobinuria
Pregnancy
Protein C deficiency
Prothrombin gene mutation
Retroperitoneal fibrosis
Transjugular intrahepatic portosystemic shunt
Trauma

3.1 BLEEDING FROM PORTAL VEIN THROMBOSIS:

- Gastric fundal varices: endoscopic variceal obturation using tissue adhesives such as cyanoacrylate is preferred, where available. Otherwise, endoscopic variceal ligation is an option.
    - ⓘ Splenectomy is curative for cases of splenic vein thrombosis and gastric varices formation.
- TIPS should be considered in patients in whom hemorrhage from fundal varices cannot be controlled or in cirrhosis whom bleeding recurs despite combined pharmacological and endoscopic therapy.

PANCREATIC DISEASES

1 ACUTE PANCREATITIS

1.1 DEFINITION

Acute non-bacterial inflammation caused by activation of pancreatic enzymes and auto-digestion of the pancreas by its own enzymes. ⓘ

1.2 ETIOLOGY

1. Gall stones **(most common)** ⓘ:
    a. Small stones can lodge in the Ampulla of Vater and block both the common bile duct (CBD) & pancreatic duct
    b. Small stones eventually pass and can be found in stool
2. Alcohol (2nd most common) ⓘ: underlying mechanisms are still unclear, but 2 effects are proposed to be involved:
    a. Direct toxic effect on pancreatic cells
    b. Transient ischemia (cutaneous vasodilation → blood diverted away from splanchnic circulation → pancreatic ischemia)
3. Hypercalcemia: Ca^{++} activates enzymes
    a. Excessive calcium causes:
        i. Deposition of Ca in soft tissues leading to obstruction of the pancreatic duct
        ii. Trypsinogen activation before it reaches the intestines
    b. With severe inflammation: Ca^{++} + fat = saponification (soap formation) → serum Ca^{++} will be depleted in the process (low-normal serum Ca^{++} levels)
4. Hyperlipidemia
    a. Mechanism unclear.
    b. Could be a cause: Elevations greater than 1,000 mg/dL can lead to pancreatitis
    c. Could be a result: TG serum levels increase with inflammatory processes– but the elevation will be moderate (<1000 mg/dL)
5. Familial (rare)
6. Iatrogenic
    a. Drugs: diuretics (**lasix** and **thiazides**), **HRT** (hormone replacement therapy)/**OCP** (oral contraceptive pill), **azathioprine**, and **steroids**
    b. ERCP (endoscopic retrograde cholangiopancreatography): ↑ Pressure with duct cannulation or contrast injection
7. Obstruction (1%): tumor at Ampulla of Vater
8. Viral infection: Coxiella, mumps
9. Trauma
10. Scorpion bite
11. Idiopathic

The most important enzymes for protein digestion are trypsin, chymotrypsin, and carboxypolypetidase. These enzymes are not in the active form until they reach the duodenum. Trypsin is activated (from its precursor: trypsinogen) by an enzyme called enterokinase that is secreted from the intestinal mucosa when chyme enters the intestine. Trypsin then cleaves the other two enzymes and trypsonigen into their active forms.

Antitrypsin is a substance secreted from the pancreatic acini that prevents the activation of trypsin and subsequently the other enzymes, which prevents auto digestion of the pancreas itself. When the duct gets obstructed or pancreatic cells gets damaged lysosomal enzymes activate trypsin. This is initially controlled by antitrypsin, but its quantities are soon overwhelmed by the amount of activated enzymes, until these enzymes digest the pancreas and cause the condition of acute pancreatitis.

1.3 CLINICAL MANIFESTATIONS

1.3.1 HISTORY

- Acute epigastric pain, radiating to **back** (pancreas is a retroperitoneal organ)
  - Patient will be leaning forward *(↓ pain as pancreas moves away from the nerves*)
- Nausea & vomiting
- Previous attacks (untreated underlying disease e.g. gall stones)
- Symptoms of underlying cause e.g. gall stones

Courvoisier sign: is case of painless jaundice and a palpable gallbladder. It is not caused by stones but most often by malignancies like pancreatic cancer and cholingeocarcinoma.

1.3.2 EXAMINATION

- Hypotension, ↑ peripheral resistance, tachycardia & fever
- Dehydration – can progress to → shock
- Epigastric tenderness
- Pleural effusion *"sympathetic effusion"* (Left lower lobe)

Hemorrhagic pancreatitis ⓘ:

- Grey Turner sign: bruising of the **flanks**; sign of retroperitoneal hemorrhage
- Cullen's sign: superficial edema and bruising in the subcutaneous fatty tissue around the **umbilicus** – indicating pancreatic necrosis & retroperitoneal bleeding

Shock in acute pancreatitis can be attributed to a few factors: **(a)** Massive exudation and hemorrhage in the retroperitoneal space **(b)** Release of a number of vasodilators, like bradykinin and PAF and in the systemic circulation **(c)** The ileus which causes the accumulation of fluid in the intestine

Figure 1: Grey Turner's Sign

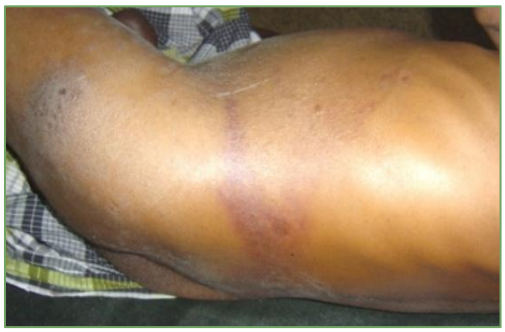
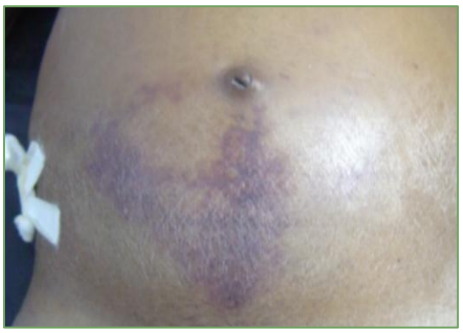


Figure 2: Cullen's Sign



1.3.3 LAB TESTS

- ↑ WBC
- ↑ **Amylase** (most **sensitive;** shorter $t_{1/2}$): >1000
- ↑ **Lipase** (more **specific** than amylase)
- Serum calcium & lipids (*see section 1.2*)

Amylase:

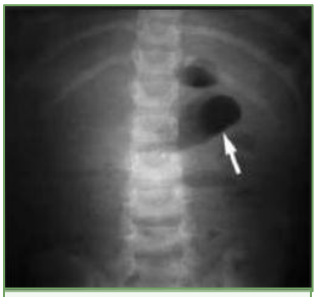
- It goes up quickly & down quickly.
- Secreted everywhere in the GI, and in the ovaries and fallopian tubes.
- Elevated in GI diseases & ectopic pregnancy.
- >1000 elevation occurs only with pancreatitis.

1.3.4 RADIOLOGY

- Plain erect chest & **abdominal X-ray:**

- **Sentinel loop:** 1-2 inflamed bowel loops dilated around pancreas causing ileus (painful obstruction); localized peritonitis causing localized ileus

- CT scan (**BEST**): **Phlegmon**; edematous, inflamed pancreas, "dirty mesentery"

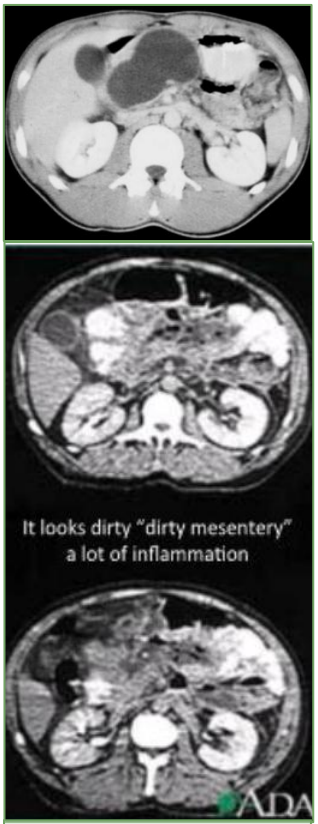


AXR: Sentinel loop

1.3.5 RANSON'S CRITERIA ⓘ

Assess severity & prognosis

1. On admission
   a. Age >55 years
   b. WBC > 16,000
   c. Glucose >11 mmol/L (x 18 = 198 mg/dL) (no insulin secretion)
   d. AST >250
   e. LDH >350
2. 36-48 hours after admission
   a. Urea >8 mg/dL (dehydration)
   b. Hematocrit: >10% decrease (hemorrhage)
   c. Fluid sequestration >6 L (patient needed 6 L of fluid)
   d. PO_2 <60
   e. Base deficit >4 (acidosis)
   f. Serum calcium <8 mg/dL (saponification)



CT scan: Phlegmon

1.4 MANAGEMENT ⓘ

- Acute pancreatitis is the only acute abdomen emergency that **DOESN'T NEED SURGERY**
- Most important: **IV FLUID REPLACEMENT**
  - Patients lose a lot of fluid (~3-4 L) to the interstitium "3rd spacing" = massive **edema** +/- retroperitoneal bleeding (due to vessel wall digestion be activated enzymes), leading to **hypovolemia** → replace fluid with normal saline or Ringer's lactate
- Then:
  a) Rest the patient: Analgesics
  b) Rest the bowel: Nasogastric tube
  c) Rest the pancreas: NPO (Nil per Os: nothing by mouth)
- Do not administer antibiotics
- 90% will improve with conservative management; surgery rarely indicated (only to debride necrotic tissue in advanced stages "necrosectomy")

1.5 COMPLICATIONS

The only indications for antibiotics ⓘ (+/- surgery, if no improvement w/antibiotics):

1. Necrosis
2. Infected necrosis
3. Abscess
4. Pseudocyst

Figure 3: Infected Necrosis (gas formation)

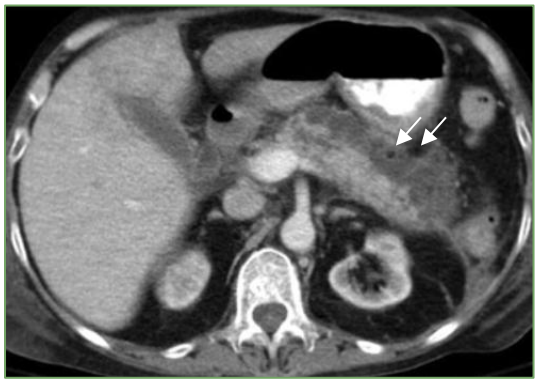
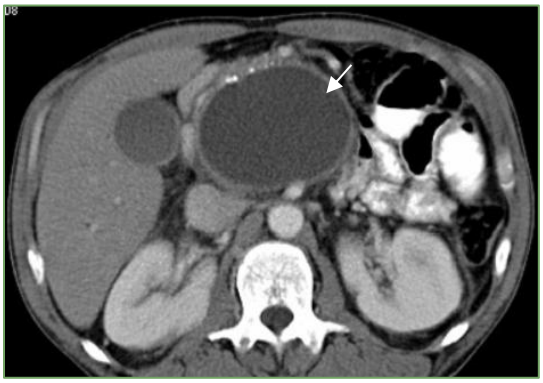


Figure 4: Pseudocyst



2 PSEUDOCYST

"Failure of pancreas to recover/recurrence of symptoms"

- A collection of amylase-rich fluid enclosed in a wall of fibrous or granulation tissue (not epithelium) that develops following an acute pancreatitis attack (>4 wks from onset)
- 50% are found to have a communication with the main pancreatic duct.

2.1.1 PRESENTATION

- Abdominal pain
- Pressure symptoms e.g.
  - Stomach: nausea
  - Bile duct: obstructive jaundice
- Epigastric mass

2.1.2 INVESTIGATIONS

- ↑ Lipase/WBC
- **CT scan (BEST)**

2.1.3 COMPLICATIONS

- Infection --→ abscess
- Rupture → pancreatic ascites
- Bleeding (erode the vessels, esp. **gastroduodenal artery**)

2.1.4 MANAGEMENT

- Observe for 6-12 weeks (50% resolve spontaneously) then repeat CT scan
- Surgery (drainage) indications:
  - Infection (external)
  - Symptomatic (internal)
  - > 5 cm (internal)

3 CHRONIC PANCREATITIS

Chronic pancreatitis is a progressive inflammatory disease of the pancreas causing fibrosis and loss of endocrine & exocrine functions of the pancreas.

Most common cause: Chronic alcoholism

3.1.1 SIGNS & SYMPTOMS:

- Abdominal pain
- Malabsorption
- Diabetes

3.1.2 DIAGNOSIS:

- Lipase & amylase: usually normal
- ↑ Glucose
- Abdominal x-ray: calcification, stones
- CT scan: calcifications, atrophy, dilated ducts

3.1.3 COMPLICATIONS:

- Biliary obstruction (due to fibrosis of the head of the pancreas)
- Pseudocyst (due to rupture of a stricture)
- Carcinoma (due to repeated inflammation)
- Splenic vein thrombosis (lies on top of the pancreas)

3.1.4 TREATMENT:

- Pancreatic enzymes (for malabsorption)
- Insulin (for diabetes)
- Analgesics (narcotics) or celiac block (injection of analgesics)
- Surgery
    - Pancreaticojejunostomy (pancreatic duct drainage procedure to decompress the dilated pancreatic duct)—most common procedure
        - Bypasses pancreatic duct & relieves pain
    - Pancreatic resection (last resort; will lead to “brittle diabetes” which is unstable diabetes with recurrent swings in glucose levels)

4 PANCREATIC ADENOCARCINOMA

- 3rd leading cause of **cancer death** in men aged 35-55 years

4.1.1 RISK FACTORS

- Most important: **smoking** ⓘ
- Fatty food
- Remote gastrectomy
- Race: Black
- Chronic pancreatitis

- Polyposis syndromes
- Family history
- Cholecystectomy

4.1.2 PRESENTATION

Arise most commonly in the head of the pancreas (70%) → present w/jaundice
Other (tail, body) usually presents late w/metastases.

- Weight loss
- Deep seated pain
- Back pain (sign of retroperitoneal invasion)
- Gastric outlet obstruction

Physical examination:

- **Jaundice**
- Hepatomegaly
- Palpable gallbladder (distended GB due to obstruction)
- Succession splash (gastric outlet obstruction)

ⓘ **Jaundice + fever = cholangitis**

- Cholangitis: inflammation of the biliary tree. **It is a medical emergency.**
- Obstruction of the biliary duct by a pancreatic head tumor promotes infection, leading to cholangitis.

4.1.3 INVESTIGATIONS

- Lab
  - ↑ WBC (w/cholangitis)
  - CA 19-9 >100 (tumor marker)
- Imaging: double-duct sign (dilated bile duct & pancreatic duct) on U/S & CT
  - U/S: dilated bile duct
  - ERCP (esp. cholangitis)
  - CT scan (**BEST**)

4.1.4 TREATMENT

- Treatment is **surgical**
  - Assess resectability (rule out local invasion & distant metastases)
  - Whipple's resection (pancreatectomy)
  - Palliative biliary & gastric drainage

POOR LONG TERM SURVIVAL

5 MCQs

1. The most specific blood test in diagnosing acute pancreatitis is:
    a. Serum amylase
    b. Urinary amylase
    c. Serum lipase
    d. CA 19-9
    e. CEA
2. The most important step in the management of acute pancreatitis is :
    a. IV fluids
    b. Antibiotics
    c. NG tube
    d. ERCP
    e. Pain medications
3. Ranson's criteria include the following except:
    a. WBC
    b. Age
    c. Serum glucose
    d. LDH
    e. Serum Lipase
4. The following are causes of acute pancreatitis except:
    a. Alcohol
    b. Gall stones
    c. Trauma
    d. Viral infections
    e. Hypocalcemia
5. The most important factor in pancreatic adenocarcinoma is :
    a. Alcohol
    b. Smoking
    c. Chronic pancreatitis
    d. Diabetes
    e. Gastrectomy
6. Pancreatic pseudocyst might be complicated with all of the following except:
    a. Malignant transformation
    b. Rupture
    c. Bleeding
    d. Jaundice
    e. Infection
7. Symptoms of chronic pancreatitis include all of the following except:
    a. Diabetes
    b. Constipation
    c. Diarrhea
    d. Abdominal pain
8. Which of the following is most helpful in diagnosing pancreatic adenocarcinoma:
    a. CA 125
    b. Serum amylase
    c. CEA
    d. CA 19-9

9. Pain in chronic pancreatitis could be improved with, except:
    a. Antibiotics
    b. Narcotics
    c. Celiac block
    d. Surgical drainage
    e. Pancreatectomy
10. Pancreatic adenocarcinoma can present with, except:
    a. Hematemesis
    b. Jaundice
    c. Abdominal pain
    d. Abdominal mass
    e. Weight loss

🗝 **Answers: 1:c, 2:a, 3:e, 4:e, 5:b, 6:a, 7:b, 8:e, 9:a, 10:a**

SUPERFICIAL LUMPS

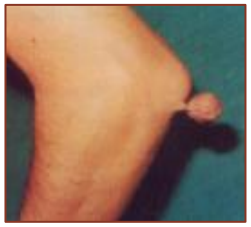
1 OVERVIEW: ANATOMY OF THE SKIN

- **SKIN ANATOMY:**
  - Epidermis → openings of glands
  - Papillary dermis → basal cell layer
  - Dermis → contains sweat and sebaceous glands

2 BENIGN SKIN LUMPS

2.1 PAPILLOMA (WART):

- Finger-like projections of all skin layers
- Usually infective (papilloma virus)
- Pedunculated or sessile
- Treatment:
  - Cauterization → if small or multiple
  - Excision → if large or sessile

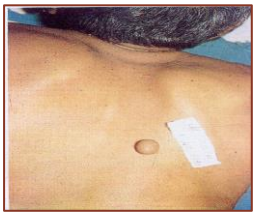


Pedunculated = attached by a peduncle/stalk

Sessile= attached directly by its base without a stalk

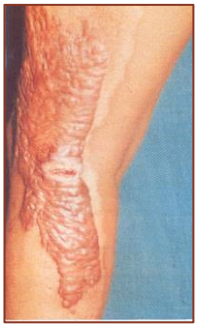
2.2 SCARS:

- A **scar** is considered a fibrous tissue proliferation following:
  - Trauma
  - Surgery
  - Infection
- It is usually flat



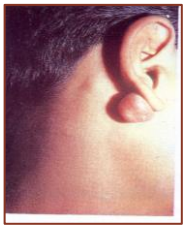
2.3 HYPERTROPHIC SCAR

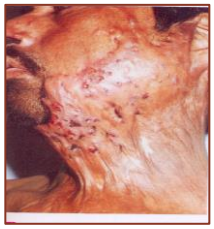
- Excessive fibrous tissue in a scar
- Confined to the scar ⓘ
- No neovascularization
- Wound infection is an important factor
- Clinically:
  - Raised
  - Non tender swelling
  - Not itchy
- It my regress gradually in six months
- Does not recur after excision



2.4 KELOID

- Excessive fibrous and collagen tissue with neovascular proliferation in a scar (enabling it to continue to grow and extend)
- Usually extends beyond the original scar ⓘ
- Clinically:
  - Initially raised





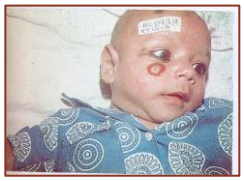
  - Pink and tender
  - Itchy and may ulcerate
- More common in dark skinned people
- Progressive vs. non-progressive
- Acquired vs. spontaneous
- Keloids can recur after excision
- Treatment:
  - Injection (hyaluronidase, steroids etc.)
  - Excision and grafting

2.5 PYOGENIC GRANULOMA

In pyogenic granuloma, patient complains of a rapidly growing lump on skin, which bleeds easily.

- Excessive granulation tissue growth in ulcers
- Clinically:
  - Firm and bright
  - Red swelling that bleed on touch
  - Recurrent bleeding when exposed to minor trauma
- Treatment:
  - Cauterization (if small)
  - Excision (if large)

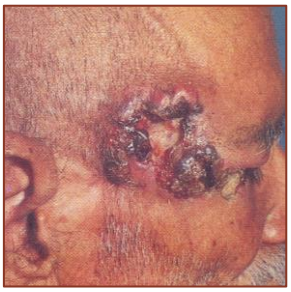
2.6 HAEMANGIOMA



- A developmental malformation of blood vessels rather than a tumor
- Types: capillary, cavernous, arterial
- It commonly occurs in skin and sub-cutaneous tissue but can occur in other organs e.g. lips, tongue, liver, and brain may be affected

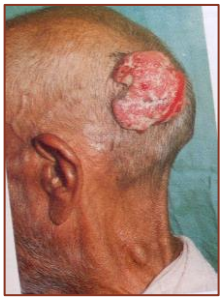
3 MALIGNANT SKIN TUMORS

3.1 BASAL CELL CARCINOMA



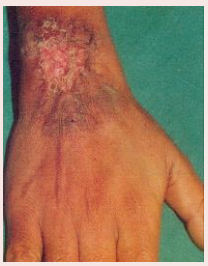
- Ulcerated tumor of basal cell layer of skin
- Middle aged white tropical males (Australia) (high UV light exposure)
- Common in face (triangle of face: nose, forehead, and eyelids)
- Low grade and slowly growing tumor (years)
- Clinically:
  - Rolled-in edges (inverted) with attempts of healing ⓘ
  - Floor shows an unhealthy granulation with a scab
  - The base is indurated and may be fixed to bone
  - Spreads locally (usually no lymph nodes metastases)
- Treatment:
  - Radio-therapy & surgery

3.2 SQUAMOUS CELL CARCINOMA (EPITHELIOMA)



- Arise from squamous cell layer of skin or mucus membrane
- It may arise from metaplasia of columnar epithelium due to chronic irritation (gall bladder, bronchus, stomach etc.)
- It can occur anywhere in the body
- Males are more affected than females
- More malignant and rapidly growing than BCC
- Clinically:
  - Edges are rolled out (everted) ⓘ
  - Spreads: locally, lymph nodes, and blood
- Treatment:
  - Radio-therapy and surgery

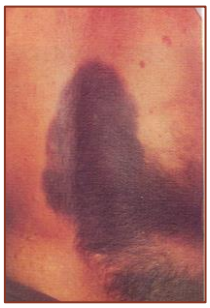
3.3 MARJOLIN ULCER



This picture shows a patient who started to develop marjolin ulcer on top of his 20 years old burn scar.

- It is a low grade squamous cell carcinoma arising in chronically inflamed ulcers or scars
- Treatment:
  - Radiotherapy and surgery

3.4 NAVEUS (MOLE)



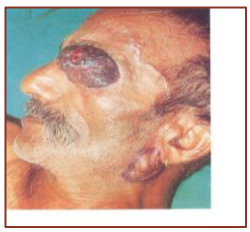
- A localized cutaneous malformation
- Includes moles and birth marks
- They may present at birth, or even later in life
- Types:
  - Junctional, intradermal, compound, blue naveus
  - Juvenile and freckle
- Evidences of malignant change: ⓘ
  - Increase in size
  - Change to irregular edge
  - Change in thickness
  - Change in color (deepening in color)
  - Change in surrounding tissue
  - Symptoms e.g. itching, bleeding discharge
  - Lymphadenopathy
  - Microscopic evidence

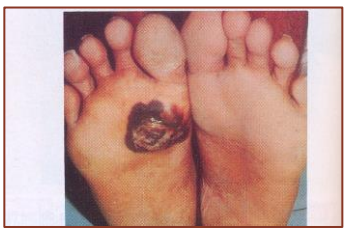
Junctional=located in dermo-epithelial junction, may turn into malignancy.

Compound=located in both dermis and junction, may turn into malignancy.

Intradermal=located mostly in dermis, has no malignancy potential.

3.5 MALIGNANT MELANOMA





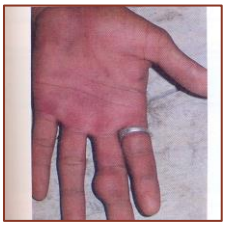
- It's a rare but most rapidly infiltrating skin tumor
- 90% of the time it arises from a pre-existing naevus
- 10% De-novo

- Metastasis:
  - Local and satellite nodules

- Lymphatic (early metastasis to LN)
- Blood (liver, lung, bone etc.)

4 SKIN CYSTS

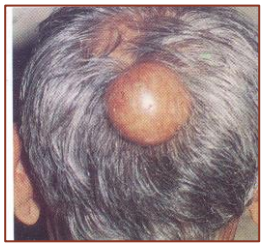
4.1 IMPLANTATION DERMOID



- It is a post-traumatic dermoid
- Commonly in fingers and hands of farmers and tailors ⓘ
- Tense, may be hard tender swelling
- Attached to skin which may be scarred
- Contains desquamated epithelial cells
- Pain and ulceration may occur following repeated trauma
- Treatment:
  - Excision is curative

4.2 SEBACEOUS CYST

Sebaceous cysts have two important features:
1. Skin adherence
2. Punctum



- It is a retention cyst due to blockage of its duct
- Lined by squamous epithelium and contains sebum and desquamated epithelium
- Commonly in scalp, face, scrotum and vulva (NEVER in palm and sole) ⓘ
- Clinically: ⓘ
  - Spherical, cystic or tense swelling, attached to skin with punctum (very diagnostic) that may discharge sebum upon squeezing
  - Indentation and fluctuation tests may be positive but transillumination test is negative (opaque fluid)

- Complications:
  - Cosmetic
  - Infection
  - Ulceration
  - Cock peculiar tumor (granuloma due to ulceration)
  - Sebaceous horn (inspissated secreted sebum)
- Treatment:
  - Excision (uninfected cyst)
  - Drainage followed by excision (infected)

5 SUBCUTANEOUS LUMPS

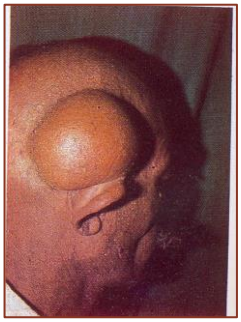
- **Cystic swellings:**
  - Congenital:
  - Dermoid cyst
  - Cystic hygroma
  - Haemangioma
  - Aquired:
    - Parasitic
    - Haematoma
    - Abscess

- **Solid swellings:**
  - Commonly benign: Schwanoma, neurofibroma, lipoma
  - Rarely malignant

5.1 DERMOID CYST

- Clinically **four** variants:
  - Sequestration dermoid
  - Implantation dermoid
  - Tubulo-dermoid
  - Terato-dermoid

5.1.1 SEQUESTRATION DERMOID



- It is a true congenital cyst (c.f. implantation dermoid)
- Ectodermal tissue buried in mesoderm forming a cyst lined by squamous epithelium and contains paste-like desquamated epithelium
- Common at lines of embryonic fusion sites ⓘ
  - Midline: neck and root of nose
  - Scalp
  - Inner or outer angles of eyes
- Clinical features: ⓘ
  - Painless, spherical, cystic mass
  - Smooth surface
  - Not attached to skin (c.f. sebaceous cyst)
  - No punctum (c.f. sebaceous cyst)
  - Not compressible (c.f. meningocele)
  - Cough impulse and bone indentation (scalp)
  - Transillumination test? positive

5.1.2 TUBULO-DERMOID:

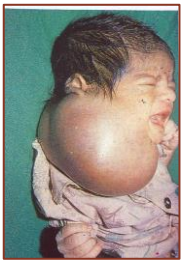
- Cystic swelling arising from the non-obliterated part of congenital duct or tube which fills up by secretions of lining epithelium
- Examples:
  - Thyroglossal cyst (remnant of thyroglossal duct)
    - Most common midline neck swelling and usually presents as a painless, rounded cystic lump, which moves on swallowing or protruding the tongue.
  - Post-anal dermoid (remnant of neuro-enteric canal)
  - Epindymal cyst tin brain (remnant of neuro-ectoderm canal)

5.1.3 TERATO-DERMOID:

- Cystic swelling arising from totipotent cells with ectodermal preponderance
  - Ovary; ovarian cyst
  - Testes; teratoma
  - Mediastinum
  - Retroperitoneum
  - Pre-sacral area

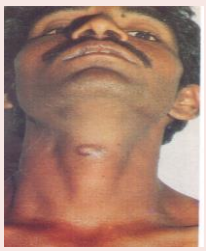
- They usually contain derivatives of mesoderm (cartilage, bone, hair, cheesy material)

5.2 CYSTIC HYGROMA

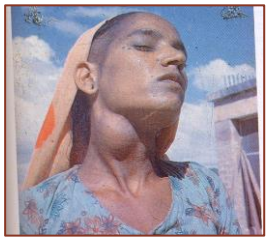


- A congenital malformation affecting lymphatic channels
- Clinically:
  - it appears early, multilocular, filled with clear fluid (transillumination positive)
- Lined by columnar epithelium
- Common in: neck, axilla, groin, mediastinum and tongue

This picture shows thyroglossal cyst, which characteristically moves with tongue protrusion.

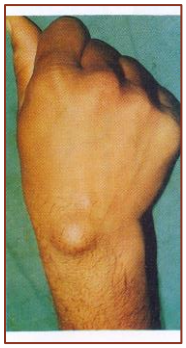


5.3 BRANCHIAL CYST



- A congenital cyst in persistent cervical sinus
- Located below angle of mandible, behind mid sternocleomastoid muscle
- Clinically:
  - Tense, distinct edges, positive fluctuation and negative transillumination
- Contains cholesterol crystals (diagnostic)
- Differential diagnosis:
  - Cold abscess, dermoid cyst, plunging ranula, cystic hygroma
  - Carotid body tumor, lymph node, submandibular gland

5.4 GANGLION



- A cystic swelling of synovial membrane of tendon or capsule in small joints
- Myxomatous degeneration
- May be communicating
- Common sites: ⓘ
  - Dorsum of wrist
  - Dorsum of foot and ankle
  - Palmar aspect of wrist and fingers
- Clinically:
  - Slowly growing lump
  - Common in females
  - Spherical, firm, cystic swelling
  - Mobile across tendon axis but limited along longitudinal axis
- Treatment:
  - Excision

5.5 LIPOMA

- Benign tumor of adipose tissue
- The most common benign tumor in subcutaneous tissue ⓘ
- Common in trunk, neck and limbs
- Encapsulated vs. diffuse

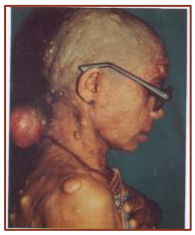
- May be mixed e.g. fibrolipma, neurolipoma (with neural tissue), and haemangioma-lipoma (with vascular tissue)
- **Dercum's disease** = multiple lipomatosis
- Clinically:
  - Painless, soft and lobulated lump.
  - Well-defined edges and skin is free.
  - Slipping sign positive.
  - Freely mobile.
  - Fluctuation test is negative.
  - Tranillumination test is negative.
- Complications:
  - Necrosis, calcification, hemorrhage, infection, and rarely malignancy
- Treatment:
  - Small symptomatic → reassurance only
  - Symptomatic → surgical excision (if encapsulated) or liposuction (if diffuse)

5.6 NEUROFIBROMA

- Tumor of nerve connective tissue (not necrosis)
- Types:
  - Localized or solitary NF
  - Generalized multiple neurofibromatosis type 1 (Von-Recklinghausen's disease)
  - Plexiform NF
  - Elephantiasis NF
  - Cutaneous NF
- Clinically:
  - Encapsulated, rounded or elliptical swelling
  - Smooth, firm with well defined edges
  - Tenderness and parasthesia may be present
  - Mobility may be diminished along nerve-axis
- Treatment:
  - Excision

5.7 MULTIPLE NEUROFIBROMATOSIS (VON-RECKLINGHAUSEN'S DISEASE)

  - Inherited as an autosomal disease
  - More common in males
  - Multiple tumors with Café-au-leit spots
  - Peripheral and cranial nerves maybe affected
  - May be associated with other tumors (e.g. endocrine)



6 MCQS

1. The finger like projections of connective tissue core that is lined with an epithelium is called:
   A. Fibroma
   B. Papilloma
   C. Lipoma
   D. Ganglion

2. The most common midline single neck swelling is:
   A. Pharyngeal pouch
   B. Dermoid cyst
   C. Laryngocele
   D. Thyroglossal cyst

3. Basal cell carcinoma:
   A. Metastasize very quickly
   B. Aggressive tumor that grows rapidly
   C. Surgery is the best treatment for local lesions
   D. A tumor of infancy

4. All of the following are common sites of squamous cell carcinoma, except:
   A. Neck
   B. Back of the hand
   C. Lower lip
   D. Lower back

5. A 40 years old male presented with 10x10 cm, soft non-compressible, mobile mass that was not attached to the skin. The most likely diagnosis is:
   A. carbuncle
   B. hemangioma
   C. Lipoma
   D. Dermoid cyst

6. Marjolin ulcer:
   A. Is a type of basal cell carcinoma
   B. Is a type of squamous cell carcinoma
   C. Is a type of melanoma
   D. Is a type of an ulcer in a dysplastic navus

🗝 Answers: 1=B, 2=D, 3=C, 4=D, 5=C, 6=B

ABDOMINAL MASSES AND HERNIAS

1 RIGHT UPPER QUADRANT MASS

1.1 HEPATIC MASSES:

- Congestive heart failure
- Macronodular cirrhosis
- Hepatitis
- Hepatoma or secondary carcinoma
- Hydatid cyst
- Liver abscess
- Riedel's lobe: an extension of the right lobe down below the costal margin along the anterior axillary line

1.1.1 PHYSICAL SIGNS

- Can't go above it, and moves with respiration
- Dull to percussion up to the level of the 8th rib in the midaxillary line
- Edge: Sharp or rounded
- Surface: Smooth or irregular

1.2 GALLBLADDER MASSES

- Mucocele: Containing Mucus
- Empyema: Containing pus
- Courvoisier law:
  - If the gallbladder is palpable and the patient is jaundiced, the obstruction of the common bile duct causing the jaundice is unlikely to be a stone because the previous inflammation will have made the gallbladder thick and non-distensible

1.2.1 PHYSICAL SIGNS

- Moves with respiration
- Not dull because it is covered by the colon
- It can be balloted i.e. felt bimanually

2 LEFT UPPER QUADRANT MASSES

2.1 SPLEEN

- Typhoid
- Tuberculosis
- Syphilis
- Glandular fever
- Malaria
- Ka lazar

- Myeloid and lymphatic leukemia
- Spherocytosis
- Thrombocytopenia purpura
- Portal hypertension
- True solitary cyst
- Hydatid cyst
- Lymphoma

2.1.1 PHYSICAL SIGNS

- Appears from below the costal margin and enlarges towards the umbilicus
- Firm, smooth and has a defined notch on its upper edge
- Cannot get above it, and dull on percussion

2.2 ENLARGED LEFT KIDNEY

3 EPIGASTRIC MASSES

3.1 CARCINOMA OF THE STOMACH

- Abdominal pain/mass
- Indigestion
- Loss of weight and appetite

3.1.1 PHYSICAL FINDINGS

- When palpable it is hard and irregular and disappears below the costal margin i.e. cannot go above it
- Moves with respiration

3.2 PANCREATIC PSEUDOCUST

- Collection of pancreatic secretion, caused by pancreatitis, on the surface of the pancreas or in part of the whole lesser sac.
- There is history of acute pancreatitis followed by epigastric fullness, pain, nausea and sometimes vomiting.

3.2.1 PHYSICAL FINDINGS

- Firm mass in the epigastric region with indistinct lower edge.
- The upper limit is not palpable.
- Usually resonant because it is covered by the stomach
- Moves very slightly with respiration

4 RIGHT ILIAC FOSSA MASSES

4.1 APPENDICULAR MASS

- Central abdominal pain shifting to the right iliac fossa associated with nausea, vomiting and loss of appetite
- Physical finidings:
  - Tender indistinct mass, dull to percussion and fixed to the right iliac fossa posteriorly

4.2 APPENDICULAR ABSCESS

- As for appendicitis with additional symptoms of an abscess such as fever, rigors, sweating and increased local pain
- Physical findings:
  - A tender mass which in its late stages may fluctuate and be associated with edema and reddening of the overlying skin

4.3 TUBERCULOSIS

- Inflamed ileocecal lymph nodes, parts of and the terminal ileum and the cecum
- Vague chronic central pain for months
- General ill health and weight loss
- The pain eventually becomes intense and settles in the iliac fossa
- Physical findings:
  - The mass is firm, distinct and hard
  - It is not tender and does not resolve with observation

4.4 CHRON'S DISEASE

- Recurrent episodes of pain in the right iliac fossa, malaise, loss of wight and episodes of diarrhea and melena
- Physical findings:
  - The elongated terminal ileum forms an elongated sausage-shaped mass which is rubbery and tender

4.5 PSOAS ABSCESS

- General ill feeling for months, night sweats and weight loss
- Physical findings:
  - Soft, tender, dull and compressible
  - There may be fullness in the lumbar region
  - The swelling extends below the groin and it may be possible to empty the swelling

4.6 OTHERS

- Cecal carcinoma
- Actinomycosis
- Ruptured epigastric artery
- Iliac lymphadenopathy
- Iliac artery aneurysm

5 LEFT ILIAC FOSSA MASSES

5.1 DIVERTICULITIS

- Recurrent lower abdominal pain and chronic constipation for years
- The acute episodes starts suddenly with severe pain, nausea, loss of appetite and constipation

- Physical finding:
  - Tender indistinct mass, with sings of general or local peritonitis

5.2 CARCINOMA OF THE SIGMOID COLON

- General cachexia
- Lower abdominal pain associated with rectal bleeding
- Change in bowel habits and sometimes intestinal obstruction
- Physcial findings:
  - Hard mass, non tender
  - May be mobile or fixed
  - The colon above the mass may be distended with indentable feces

5.3 OTHERS

- Chron's disease
- Psoas abscess
- Same masses of the right iliac fossa

6 HYPOGASTRIC MASSES

6.1 URINARY BLADDER

- Acute retention: the bladder is full and tender
- Chronic retention: Painless
- History of prostatism
- Physical findings
  - Arises out of the pelvis and so it has no lower edge
  - Not mobile and dull to percussion
  - Direct pressure often produces a desire to micturate

6.2 PREGNANT UTERUS

- The uterus enlarges to the xiphisternum by the 36th week of pregnancy, at this stage the fetus is palpable
- A pregnant uterus is smooth, firm and dull

6.3 FIBROIDS

- They cause irregular and heavy periods, disturbed micturation, lower abdominal pain and backache
- Physical findings:
  - Arises out of the pelvis and so the lower edge is not palpable
  - Firm or hard, moves slightly in transverse direction and dull on percussion

7 ABDOMINAL HERNIA

7.1 DEFINITION

- An abnormal protrusion of intra-abdominal contents through a defect in the abdominal wall.

- Protrusion of a viscus or part of it through an opening in the wall of its containing cavity

7.2 ETIOLOGY

7.2.1 CONGENITAL DEFECTS:

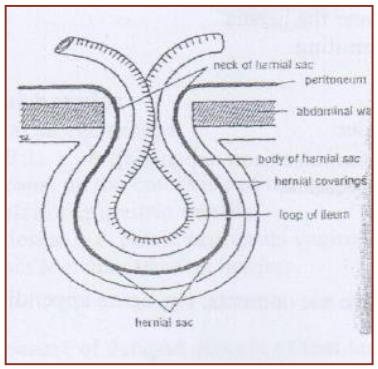
- Indirect inguinal hernia, umbilical hernia
- Patent processus vaginalis: almost always causes indirect inguinal hernia

7.2.2 ACQUIRED

- Loss of tissue strength and elasticity, due to aging or repetitive stress:
  - *hiatal hernia*
- Operative Trauma, in which normal tissue strength is altered surgically:
  - *incisional hernia*
- Increased intra-abdominal pressure:
  - Heavy lifting
  - Coughing, asthma, and COPD
  - Straining at defecation or urination (e.g. Benign prostatic hypertrophy, constipation, colon/rectal cancer)
  - Multiparity (Multiple pregnancies)
  - Ascites and abdominal distension
  - Obesity

7.3 COMPOSITION

- The sac: diverticulum of peritoneum consisting of a mouth, neck, body and fundus
- The body: varies in sice snd is not necessarily occupied
- The coverings: derived from the layers of the abdominal wall
- The contents: may be omentum, bowel, ovary, bladder… etc.



7.4 COMMON CLINICAL PRESENTATION OF ABDOMINAL WALL HERNIA

- Swelling
- Reduction
- site

7.5 ABDOMINAL WALL SITES

- Mid-line
- Umbilical area
- Inguinal region
- Femoral canal
- Para-median lineObturator

- Lumber area
- Obturator foramen
- Incisional or scar line

7.6 CLASSIFICATION

7.6.1 REDUCIBLE:

- The contents of the sac are reduced spontaneously or manually

7.6.2 IRREDUCIBLE

- The contents remain constantly outside

7.6.2.1 INCARCERATED

- Trapped or imprisoned
- Initially it is reducible, then it becomes irreducible → cannot be reduced (either spontaneously or manually).
- Does not denote obstruction
- Blood supply remains intact
- Nausea, vomiting, and symptoms of bowel obstruction (possible).

7.6.2.2 OBSTRUCTED

- Contains obstructed intestine
- Small intestine obstruction presents with pallor and vomiting
- Large intestine obstruction presents with distention and constipation
- Blood supply remains intact

7.6.2.3 STRANGULATED

- A surgical emergency
- Likely in hernias with narrow necks
- Blood supply is seriously impaired rendering the contents ischemic ⓘ
- Gangrene may occur within 5-6 hours after the inset of symptoms
- Symptoms of an incarcerated hernia present combined with a toxic appearance.
- Strangulation is probable if pain and tenderness of an incarcerated hernia persist after reduction.
- The femoral hernia is the most liable to strangulation due to its narrow neck and its rigid surroundings ⓘ
- The constricting agents that compress the blood supply are: (In order of frequency)
  - The Neck
  - External ring in children
  - Adhesions with the sac (rare)
- Symptoms
  - Sudden pain over the hernia
  - Nausea and vomiting
- Signs
  - Tense and tender
  - Absent cough impulse (non expansile) ⓘ

7.6.2.4 INFLAMED

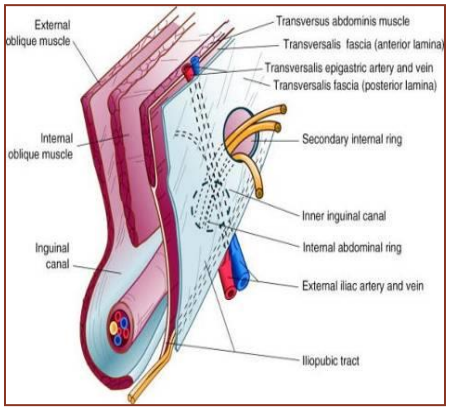
- Rare
- Due to inflammation on the sac contents, e.g. acute appendicitis or salpingitis

8 INGUINAL HERNIA

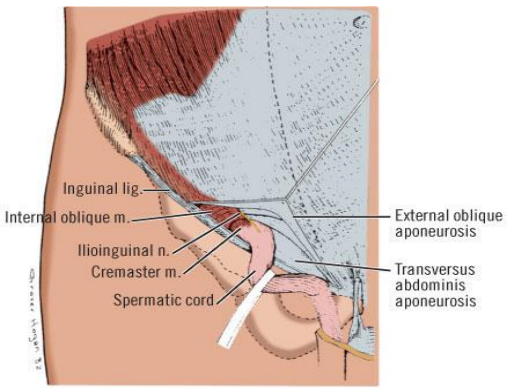
- The most common form of hernia in both sexes
- Subdivided into direct and indirect
- In adult males it is most commonly indirect

8.1 SURGICAL ANATOMY

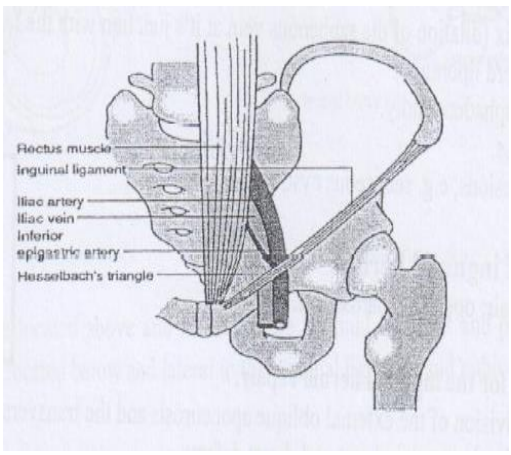
- Superficial ring: triangular aperture in the external oblique aponeurosis 1.25 cm above the pubic tubercle
- Deep ring: U-shaped condensation of the transversalis fascia 1.25 cm above the inguinal ligament
- The inguinal canal:
  - In infants the two triangular aperture are superimposed and the canal is slightly oblique
  - In adults it is 3.75-4 cm long
  - In females, it contains the round ligament of the uterus Contains the spermatic cord and round ligament of the uterus
  - In males, it contains
    - The ilioingunal nerve
    - The spermatic cord and its contents, which are
      - ✓ Genital branch of the genitofemoral nerve
      - ✓ Testicular artery
      - ✓ Pampiniform plexus of veins
      - ✓ Cremasteric muscle fibers
      - ✓ Cremasteric vessels
      - ✓ Vas deferens



  - Boundaries of the inguinal canal
    - Anteriorly: external oblique aponeurosis
    - Posteriorly: facia transversalis and conjoined tendon
    - Superiorly: internal oblique aponeurosis
    - Inferiorly: inguinal ligament



Skandalakis JE, Colborn GL, Weidman TA, et al: *Skandalakis' Surgical Anatomy*: http://www.accesssurgery.com



8.2 INDIRECT (OBLIQUE) INGUINAL HERNIA

- Most common of all forms at all age groups
- The male: female ratio is 20:1
- Travels down the inguinal canal on the outer side of the spermatic cord
- Its neck lies lateral to the inferior epigastric vessels
- Can be due to a congenital lesion i.e patent processus vaginalis
- Strangulation is common, but less than in femoral hernia
- Seen in young patients
- In adult males
  - Mostly on the right side because of delayed decent of the right testicle
  - 12% bilateral

8.3 DIRECT INGUINAL HERNIA

- Comes out forward via the posterior wall of the inguinal cana, at Hasselbach's (i.e. ingunal) triangle due to a defect of weekness of the facia transversalis
- Always acquired, never congenital ⓘ
- It has a wide neck and therefore there is no hazard of strangulation ⓘ
- The neck is medial to the inferior epigastric vessels
- Does not attain a large size

8.4 CLINICAL PRESENTATION OF INGUINAL HERNIA

- Groin pain referred to the testicle
- Cough impulse (Expensile) ⓘ
- A large hernia causes dragging pain
- Presents as a swelling or fullness at the hernia site
- Aching sensation (radiates into the area of the hernia)
- No true pain or tenderness upon examination
- Enlarges with increasing intra-abdominal pressure and/or standing

8.5 DIFFERENTIAL DIAGNOSIS

- Hydrocele

Pantaloon (Saddlebag) hernia is the simultaneous occurrence of a direct and an indirect hernia.
It causes two bulges (medial and lateral) that straddle the inferior epigastric vessels

Hasselbach's triangle is bounded by: ⓘ
-Inguinal ligament inferiorly
-Inferior epigastric artery laterally
-Lateral border of rectus muscle medially

- Encysted hydrocele of the cord
- Varicocele
- Epididymoorchitis
- Testicular torsion
- Undescended testis
- Ectopic testis
- Testicular tumor
- Pseudohernia
- Femoral artery aneurysm
- Saphena varix (dilation of the saphenous vein at it's junction with the femoral vein in the groin)
- Spermatic cord lipoma
- Inguinal lymphadenopathy
- Psoas abscess
- Cutaneous lesions, e.g. sebaceous cyst, skin tumor

8.6 TREATMENT OF INGUINAL HERNIA

- Surgical repair: open vs laproscopic

In adults, we do herniotomy and herniorrhaphy (repair) because the problem is due to weakness.

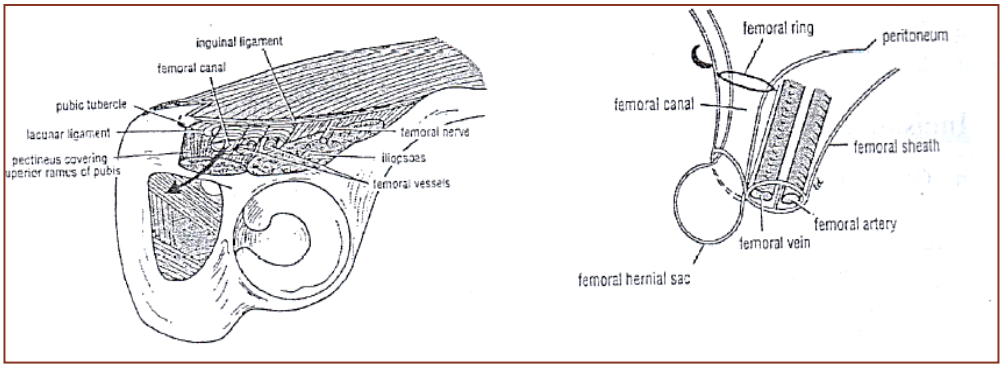
In children, we do herniotomy only; because the problem is congenital, not muscle weakness.

8.7 ESSENTIAL STEPS FOR THE INGUINAL HERNIA REPAIR

- Complete division of the external oblique aponeurosis and the transversalis fascia
- Differentiation between indirect and direct defects
- Isolation of the spermatic cord
- Ligation and removal of the sac at the deep inguinal ring flush with peritoneum
- Oblique reconstruction of the inguinal canal with an anterior and posterior wall and an internal and external ring

9 FEMORAL HERNIA

- Commonly affecting females ⓘ
- Most liable to strangulationⓘ
- The hernia descends vertically to the saphenous opening
- **Surgical anatomy**
  - Boundaries of the femoral sheath
    - Anteriorly: inguinal ligament
    - Posteriorly: Iliopectineal ligament, pubic bone and pectineus muscle fascia
    - Medially: lacunar ligament
    - Laterally: femoral nerve
  - The femoral canal
    - The most medial compartment of the femoral sheath
    - Extends from the femoral ring to the saphenous opening
    - 1.25 cm long and 1.25 cm wide at the base
    - Contains fat, lymphatic vessels and the lymph node of Cloquet



- **Differential diagnosis**
  - Inguinal hernia
  - Inguinal hernias are located above and medial to the inguinal ligament and pubic tubercle, whereas femoral hernias are located below and lateral to the inguinal ligament and pubic tubercle ⓘ
  - Saphena varix
  - Femoral lymphadenopathy
  - Femoral artery aneurysm
  - Psoas abscess
- **Complications**: Strangulation due to a narrow unyielding femoral ring
- **Treatment**: Surgical repair

10 UMBILICAL AND PARAUMBILICAL HERNIA

- Umbilical hernia is seen in infants and children
- The female: male ratio is 20:1
- PUH affects adults. The defect is either supra or infraumbilical through the linea alba
- When enlarged, it becomes rounded or oval shaped
- May contain omentum, small intestine or transverse colon
- **Etiology**
  - Obesity
  - Flabbiness of the abdominal muscles
  - Multiparity
- **Clinical Features**
  - Irreducibility in PUH is due to omental adhesions within the sac
  - Pain may be colicky due to parital or complete intestinal obstruction
- **Treatment**: Open (Mayo's repair) or laproscopic repair (if the defect is more than 4 cm)

11 INCISIONAL HERNIA

- Occurs in surgical scars and it has no actual neck (or its neck is wide), so it does not lead to complications
- **Causes**
  - Mechanical factors (increase in intraabdominal pressure postoperatively)
    - Prolonged ilius
    - Chronic cough

        - Repeated vomiting
        - Lifting heavy objects in the immediate postoperative period
    - Patient factors
        - Infection
        - Malnutrition
        - Diabetes and chronic illness
        - Steroid treatment
    - Technical factors
        - Too much tension on closure, or closure with absorbable sutures
        - Ischemia
- **Clinical features**: swelling at the scar associated sometimes with pain
- **Treatment**: Open or laparoscopic repair

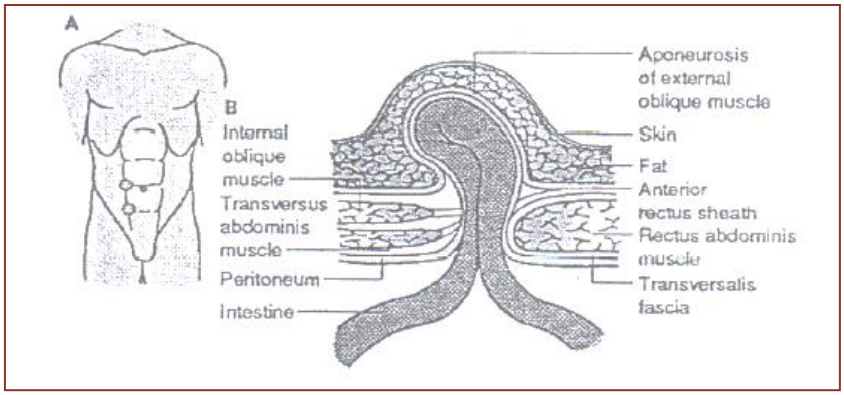
12 EPIGASTRIC HERNIA

- Due to a defect in the linea alba between the xiphoid process and the umbilicus
- Starts as a protrusion of the extraperitoneal fat at the site where a small blood vessel pierces the linea alba
- If the protrusion enlarges, it drags a pouch of peritoneum after it
- **Clinical features**
    - May be asymptomatic or painful, either locally or simulates peptic ulcer pain
- **Treatment**: Mayo's repair

13 RARE EXTERNAL HERNIAS

13.1 SPIGELIAN HERNIA

- Occurs at the space between the semilunar line and the lateral adge of the rectus muscle (Inferior to the arcuate line)
- The posterior recuts sheath is lacking which contributes to the inherent weakness in this ares
- Preoperative diagnosis is correct in only 50% of patients
- US and CT scan are helpful to confirm the diagnosis
- Approximation of the tissues adjacent to the defect with interrupted sutures is curative. If the defect is large, it can be covered with mesh



13.2 LUMBAR HERNIAS

- Broad bulging hernias
- Usually don't get incarcerated
- Petit's hernia
    - Occurs in the inferior lumbar triangle which has the following boundaries
        - Laterally: external oblique muscle
        - Medially: latissimus dorsi
        - Inferiorly: iliac crest
- Grynfeltt's hernia
    - Less common
    - Occurs in the superior lumbar triangle which is bounded:
        - Superiorly: inferior margin of the 12th rib
        - Medially: sacrospinalis muscle
        - Laterally: internal oblique muscle

Secondary lumbar hernia develops as a result of trauma, mostly surgical (e.g. renal surgery) or infection.

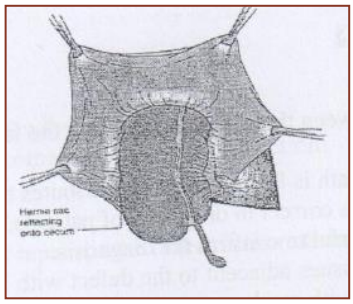
Lumbar hernias were encountered relatively frequently in the past in cases of spinal tuberculosis with paraspinal abscesses

13.3 OBTURATOR HERNIA

- The obturator canal is covered by a membrane pierced by the obturator nerve and vessels. Weakening of the obturator membrane and enlargement of the canal may result in the formation of a hernia sac. Which can lead to intestinal herniation and obstruction
- Presentation could be with evidence of compression of the obturator nerve leading to pain in the medial aspect of the thigh
- Treated by surgery

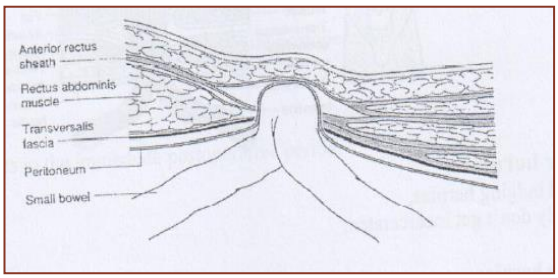
13.4 SLIDING HERNIA

- It is a hernia in which part of the posterior wall of the sac is formed by a viscus (intraabdominal organ), e.g. sigmoid colon, cecum, ovary or portion of the bladder
- The wall of the hernial sac, rather than being formed completely by peritoneum, is in part formed by a retroperitoneal structure
- Bladder slides posterio-medially (PM) and the colon posterio-lateral (PL)



13.5 OTHER

1. **Richter's hernia**
    a. It is a hernia at ant site in which only part of the circumference of the bowel (usually jejunum) is involved
    b. Only one side of the bowel wall is trapped in the hernia, rather than the entire loop of bowel.
    c. Does not usually obstruct but can strangulate or become incarcerated ⓘ
    d. This is especially dangerous because the incarcerated portion of bowel can necrose and perforate in the absence of obstructive symptoms.



2. **Littre's Hernia**
    a. Any groin hernia that contains a Meckel's Diverticulum,
    b. Rare.
    c. Usually incarcerated or strangulated
    d. If the diverticulum is symptomatic or strangulated, it is mandatory to excise it at the time of repair.
3. **Divarication (Separation) of the recti abdominis (Diastasis recti)**
    a. Only a facial weakness, not a true hernia ⓘ
    b. Seen more in elederly multiparous patients
    c. A gap in the linea albe (medial margin of the recti) seen on straining through which the abdominal contents bulge.
    d. No treatment is necessary ⓘ
4. **Perineal Hernias**
    a. Occur in the pelvic floor usually after surgical procedures such as an abdominoperineal resection.
5. **Peri- or para-stomal Hernia**
    a. Hernia adjacent to an ostomy "e.g. colostomy".
6. **Amyand's Hernia**
    a. Hernia sac containing a ruptured appendix.
7. **Hesselbach's Hernia**
    a. Hernia under the inguinal ligament lateral to femoral vessels.
8. **Cooper's Hernia**
    a. Hernia through the femoral canal & tracking into the scrotum or labia majus.

14 METHODS OF HERNIA REPAIR

14.1 OPEN TECHNIQUE (HERNIOTOMY AND REPAIR)

- Bassini repair
- Draning
- Shouldice
- McVay (Cooper's ligament repair)
- Mesh (i.e. hernioplasty)

14.2 LAPAROSCOPIC REPAIT

- Two types
    - TAPP (transabdominal preperitoneal) repair
    - TEP (totally extraperitoneal) repair
- Indicated in only two conditions: ⓘ

- Bilateral hernia
- Recurrent hernia

15 HISTORY

15.1 LUMP

- duration, first symptoms, associated symptoms, progression, persistent ,other sites ,cause
- Does it reduce on lying down?
- Has there been an episode of pain in the swelling?
- Has there been an episode of abdominal pain?
- Does the patient have any febrile symptoms?

15.2 HISTORY OF RECTAL BLEEDING

- Causes of increase of intrabdominal pressure.
- Previous surgeries?

16 PHYSICAL EXAM

- Examine the patient in the **standing and supine positions.**
- Examine the patient from the front

Inspection

- lump: site shape
- scrotum: does it extend to the scrotum

Palpation

- Ask the patient about pain before you palpate
- Can you go above it
- Can you palpate the testis
- If it is a hernia ➔ type
- Define pubic tubercle

Feel from the sides

- Aim to examine the lump.
- Tenderness, temperature, size ,shape, site, composition
- Reducible. Ask the pts if you couldn't
- Controlled when you pressure over deep inguinal ring.
- Expensile cough impulse.
- Direction of reappearance.

Investigations

- CBC
- *Leukocytosis may occur with strangulation*.
- Electrolytes, BUN, creatinine levels :
- *Assess the hydration status of the patient with nausea and vomiting.*
- Urinalysis: *narrowing the differential diagnosis of genitourinary causes of groin pain*.

- Imaging studies:
  - *Imaging studies are not required in the normal workup of a hernia.*
  - Ultrasonography. (obese)
  - If an incarcerated or strangulated hernia is suspected:
    - *Flat and upright abdominal films to diagnose a small bowel obstruction.*

17 MCQS

1. **Which of the following is true regarding femoral hernia?**
   a. Commonly seen in children.
   b. It is the commonest hernia seen in females
   c. usually presents with inguinal swelling
   d. it is liable for complications
   e. usually treated conservatively

2. **The most common cause of an enlarged lymph node in the femoral triangle is**
   a. Tuberculosis lymphadenitis
   b. Brucella
   c. Neoplastic
   d. Nonspecific lymphdenitis
   e. Sarcoidosis

3. **The first symptoms of strangulated Inguinal Hernia is:**
   a. Vomiting
   b. Fever
   c. Septic shock
   d. Constipation
   e. Pain

4. **Inguinal Hernia:**
   a. Is more common in girls.
   b. Hernioraphy is the treatment of choice.
   c. Ultrasound is required to diagnose it.
   d. Hernia sac may contain ovary, appendix, or omentum.
   e. Direct inguinal hernia is more common than indirect.

5. **Patent processus vaginalis results in:**
   a- indirect inguinal hernia
   b- direct inguinal hernia
   c- femoral hernia
   d- umbilical hernia

6. **The following are important steps in the management of strangulated hernia except:**
   a. Nasogastric tube
   b. Antibiotics
   c. Conservative treatment till obstruction is relieved

d. Intravenous fluids
e. Consent for possible bowel resection

7. **All of the followings are external hernias except:**
   a. Obturator hernia
   b. Hiatal hernia
   c. Femoral hernia
   d. Lumbar hernia

8. **The differential diagnosis of an inguinal swelling could include all of the followings except:**
   a. Lipoma of the cord
   b. Indirect inguinal hernia
   c. Encysted hydrocele
   d. Undescended testis
   e. Varicocele

9. **A 41 y/o woman is a known case of femoral hernia and was scheduled to be operated later. She presented in the ER with severe pain over the hernia and fever. On examination, the hernia was tense and tender, and the cough impulse was negative. The diagnosis is:**
   a. Inflamed hernia
   b. Strangulated hernia
   c. Obstructed hernia
   d. Incarcerated hernia

10. **Boundaries of Hesselbach's triangle include all the followings EXCEPT:**
    a. Lateral border of rectus muscle
    b. Inferior epigastric artery
    c. External iliac artery
    d. Inguinal ligament

11. **Which one of the following clinical future helps to differentiate between inguinal hernia and hydrocele in children?**
    a. Reducibility
    b. Scrotal swelling
    c. Tenderness
    d. Transillumination

🗝 **Answer Key → 1D, 2D, 3E, 4D, 5A, 6C, 7B, 8E, 9B, 10C, 11A**

INTRODUCTION TO TRAUMA

1 MECHANISMS AND PATTERNS OF INJURY ⓘ

1.1 BLUNT INJURY

Classified into:

1. High-energy transfer (e.g. Car Accident).
2. Low energy transfer (e.g. Fall from a bicycle).
   - Associated with multiple widely distributed injuries because the energy is transferred over a wider area during blunt trauma.

1.2 PENETRATING INJURY

Classified into:

1. Stab wound.
2. Gunshot wound.
3. Shotgun.
   - Damage is localized to the path of the bullet or knife.

2 PRE-HOSPITAL CARE

The objective of pre-hospital care is to prevent further injury, initiate resuscitation and transport the patient safely and rapidly to the most appropriate hospital.

- Most important things in pre hospital care:
  - Airway control.
  - Fluid resuscitation.
- Transportation either by:
  - Ground Ambulance, or
  - Helicopter.

Initial evaluation and resuscitation of the injured patient in the ER is done using **Primary survey** and **secondary survey.**

3 PRIMARY SURVEY "THE MOST IMPORTANT" ⓘ

- The goal of primary survey is to **identify** and **treat** conditions that constitute an immediate threat to life.
- **Advanced trauma life support** (ATLS) provides a structured approach to the trauma patient with standard Algorithms of care.
- It emphasizes the "**golden hour**" concept that timely prioritized interventions are necessary to prevent death.
- The **ATLS** Course refers to the primary survey as assessment of the "**ABC**" (**A**irway with cervical spine protection, **B**reathing and **C**irculation). ⓘ
- Although the concepts within primary survey are presented in a sequential fashion in reality they often proceed simultaneously. In details:

The **golden hour**: it's the first hours of the patient arrival to the ER.

3.1 AIRWAY MANAGEMENT WITH CERVICAL SPINE PROTECTION

I. Conscious patient who do not show tachypnea and have **normal voice** do not require early attention to the airway. (So you proceed to the next step!)

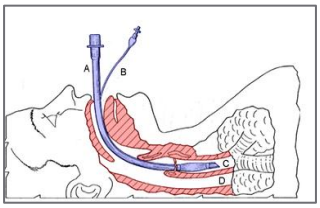
II. Patients with penetrating neck injuries and:
- An expanding hematoma.
- Evidence of chemical or thermal injuries to mouth, nares or hypopharynx.
- Extensive subcutaneous air in the neck.
- Complex maxillofacial trauma.
- Airway bleeding.
  - In these cases elective intubation should be performed. These patients may initially have a satisfactory airway but they may become obstructed if soft tissue swelling, hematoma formation, or edema progress.

III. Establishment of a definitive airway, immediate intubation, (i.e. endotracheal intubation) is indicated in:
- Patients with apnea.
- Inability to protect the airway due to altered mental status.
- Impending airway compromise due to inhalation injury.
- Hematoma.
- Facial bleeding.
- Soft tissue swelling or aspiration.
- Inability to maintain oxygenation.

ⓘ: Altered mental status is the most common indication for intubation in the ER for traumatic patients.

3.1.1 OPTIONS FOR ENDOTRACHEAL INTUBATION INCLUDE:

3.1.1.1 NASOTRACHEAL INTUBATION:



It can be accomplished **only in patients who are breathing spontaneously**. The primary application for this technique in Emergency Department (ED) is in those patients requiring emergent airway support in whom chemical paralysis cannot be used.

It is contraindicated in extensive **maxillofacial** injuriesⓘ. Why? It may cause further injury.

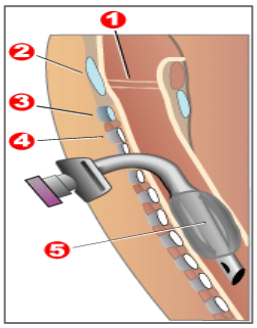
3.1.1.2 OROTRACHEAL INTUBATION:

It is **the most common** technique used to establish a definitive airway.

- Because all patients are presumed to have cervical spine injuries, manual in-line cervical immobilization is essential especially in unconscious patients in which we must protect the cervical spine.
- Correct endotracheal placement is verified with:
  - **Direct laryngoscopy**, you see the tube heading to the vocal cords.
  - **Capnography**, if you connect the patient on a ventilator you will see high CO_2, at least it's in the trachea.
  - **Clinically**: Audibility of bilateral breath sounds, by auscultation.
  - And finally **Chest X-Ray**, only if the patient is stable.

3.1.1.3 SURGICAL ROUTES:

3.1.1.3.1 CRICOTHYROIDOTOMY:



Patients in whom attempts at intubation have failed or who are precluded from intubation due to extensive facial injuries ⓘ.

It has no complications that affect the vessels because you go to the laryngeal membrane directly (no stenosis).

3.1.1.3.2 EMERGENT TRACHEOSTOMY:

Is indicated in a patient with extensive laryngeal injury

It may cause complications that damage the vessels (stenosis).

3.2 BREATHING AND VENTILATION

Once a secure airway is obtained, adequate oxygenation and ventilation must be assured. All injured patients should receive supplemental oxygen and be monitored by pulse oximetry.

The following conditions constitute an immediate threat to life due to inadequate ventilation and should be recognized during the primary survey. The main 3 conditions that you must take care of are ⓘ:

1. Tension pneumothorax
2. Open pneumothorax
3. Flail chest with underlying pulmonary contusion

3.2.1 TENSION PNEUMOTHORAX

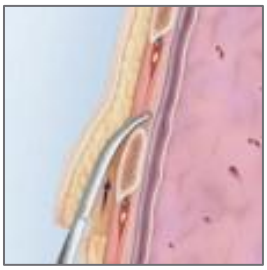
Tension pneumothorax is the accumulation of air in the pleural cavity with lung making a one-way valve, allowing the air to enter without going out.

Diagnosis: Respiratory distress and hypotension in combination with any of the following physical signs in patients with chest trauma:

- **Tracheal deviation** away from the affected side (e.g accumulation of air in the right lung will deviate the trachea to the left side).
- Lack or decreased breath sounds on the affected side. ⓘ
- Subcutaneous emphysema on the affected side.
- **Distended neck veins** due to impendence of superior vena cava, but the neck veins may be flat due to systemic hypovolemia.

  1. In cases of tension pneumothorax, the parenchymal tear in the lung act as a one-way valve, with each inhalation allowing additional air to accumulate in the pleural space.
  2. The normally negative intrapleural pressure becomes positive which depresses the ipsilateral hemidiapgragm and shift the mediastinal structures into the contralateral part of the chest.
  3. Subsequently the contralateral lung is compressed and the heart rotates about the superior and inferior vena cava, this decreases venous return and ultimately decreases cardiac output, which results in an immediate cardiovascular collapse.

If there is a tension pneumothorax on the right side, this will push the mediastinum to the left and kink the SVC which lead to congestion of the face and upper limbs and distension of neck veins b/c there is obstruction of venous return. This condition is known as **“Superior Vena Cava Syndrome"** But if the patient is hypovolemic, the veins will not distend.



Treatment:

- **Immediate needle thoracostomy** decompression with a 14-gauge angiocatheter in the second intercostal space in the midclavicular line.
- **Tube thoracostomy (chest tube)** in the fifth intercostal space in the midaxillary line immediately in the emergency department before the chest radiograph.

3.2.2 OPEN PNEUMOTHORAX (OR SUCKING CHEST WOUND)

This occurs with full-thickness loss of the chest wall, permitting free communication between the pleural space and the outer atmosphere.

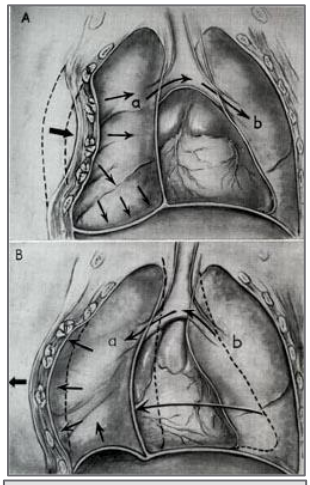
This compromises ventilation due to equilibration of atmospheric and pleural pressures, which prevents lung inflation and alveolar ventilation and result in hypoxia and hypercarbia (↑CO_2).

Treatment:

- Closure of the chest wall defect if it's small and tube thoracostomy if it's large defect.

3.2.3 FLAIL CHEST

- It occurs when three or more contiguous ribs are fractured in at least two locations
- Paradoxical movement of this free-floating segment of chest wall. ⓘ
- Rarely the additional work of breathing and chest wall pain caused by the flail segment is sufficient to compromise ventilation.
- Resultant hypoventilation and hypoxemia may require intubation and mechanical ventilation.
- Most of the time flail chest it associated with contusion of the lung parenchyma.



Diagrams depicting the paradoxical motion observed during respiration with a flail segment

- 🗎 It occurs when multiple adjacent ribs are broken in multiple places, separating a segment, so a part of the chest wall moves independently.
- The number of ribs that must be broken varies by differing definitions: some sources say at least two adjacent ribs are broken in at least two places, some require three or more ribs in two or more places.
- The flail segment moves in the opposite direction as the rest of the chest wall: because of the ambient pressure in comparison to the pressure inside the lungs, it goes in while the rest of the chest is moving out, and vice versa.
- This so-called "paradoxical motion" can increase the work and pain involved in breathing.

3.3 CIRCULATION WITH HEMORRHAGE CONTROL

I. Blood pressure and pulse should be measured manually at least every 5 minutes for patient with significant blood loss until normal vital signs values are restored.
II. Two peripheral catheters, 15- 16 gauge or larger in adults.
III. **Fluid resuscitation**. We start with crystalloids like normal saline or ringer's lactate but ringer's is better. Normal saline contains large amount of Cl^-

which can lead to hyperchloremic metabolic acidosis and the patient is already has metabolic acidosis ⓘ.

IV. Blood should be drawn simultaneously and send for measurement of hematocrit level, as well as for typing and cross-matching for possible blood transfusion in patient with evidence of hypovolemia.

V. If peripheral angiocatheter access is difficult, saphenous vein cutdown at the ankle provide excellent access.

VI. Additional venous access through femoral or subclavian vein (can be used for central venous pressure (CVP) measurement).

VII. Intraosseous needle can be placed in the proximal tibia (preferred) or distal femur of an unfractured extremity for fluid resuscitation in patient under 6 years of age.

VIII. External control of hemorrhage should be achieved promptly while circulating volume is restored. Manual compression of open wounds with ongoing bleeding should be done with single 4 x 4 gauze and a gloved hand ⓘ. Blind clamping of bleeding vessels should be avoided because you may damage the vessels. ⓘ

- During the circulation section of the primary survey ***FOUR*** life-threatening injuries must be identified:
    a. **Massive hemothorax** “bleeding in the thorax”
    b. **Cardiac tamponade** “bleeding in the myocardium”
    c. **Massive hemoperitoneum** “bleeding in the abdomen”
    d. **Mechanically unstable pelvic fracture** “bleeding in the pelvis”
- Those are the causes of massive hypotension in traumatic patients.
- If the patient has hypotension and you couldn’t find a source of bleeding when looking in the abdomen and chest. Think of cardiac tamponade, as it is very commonly missed. ⓘ
- ***THREE*** critical tools used to differentiate these in multisystem trauma patient
    a. **Chest** radiograph
    b. **Pelvis** radiograph
    c. Focused Abdominal Sonography for Trauma (**FAST**)

    The **FAST** is performed as part of the initial evaluation of the trauma patient in the emergency center. It consists of four separate views of four anatomic areas:

    1. The right upper abdomen (Morison's space between liver and right kidney).
    2. The left upper abdomen (perisplenic and left perirenal areas).
    3. Suprapubic region (perivesical area).
    4. Subxyphoid region (pericardium).

3.3.1 IMMEDIATE TREATMENT:

3.3.1.1 MASSIVE HEMOTHORAX

- Clinically, if you listen to the chest there will be no breathing sounds on the affected side.
- Tube thoracostomy to facilitate lung re-expansion.
- Massive hemothorax (if the tube drain >1500 ml).
- If blood is seen it’s an indication for operative intervention.

3.3.1.2 CARDIAC TAMPONADE

- Pericardial drain under ultrasound guidance.
- Followed by operative intervention.

3.3.1.3 MECHANICALLY UNSTABLE PELVIS FRACTURE

- Pelvis fracture.
- Immediate external fixation.

3.3.1.4 MASSIVE HEMOPERITONEUM WITH HEMODYNAMIC UNSTABILITY

- Fluid resuscitation
- Immediate surgical intervention with shock patients

3.3.1.4.1 SHOCK CLASSIFICATION AND INITIAL FLUID RESUSCITATION

Classic signs and symptoms of shock: are tachycardia, hypotension, tachypnea, mental status changes, diaphoresis and pallor. The quantity of acute blood loss correlates with physiologic abnormalities.

1. **Tachycardia** is often the earliest sign of ongoing bleeding. And it is not reliable in old patients or patients on beta-blockers.
2. **Hypotension** is not reliable early sign of hypovolemia, because blood volume must decrease by >30% before hypotension occurs.

Signs and Symptoms of Advancing Stages of Hemorrhagic Shock ⓘ				
	Class I	Class II	Class III	Class IV
Blood loss (ml)	Up to 750	750 – 1500	1500 – 2000	> 2000
Blood loss (% BV)	Up to 15%	15 – 30%	30 – 40%	>40 %
Pulse Rate	<100	>100	>120	>140
Blood Pressure (mmHg)	**Normal**	**Normal**	Decreased	Decreased
Pulse Pressure	Normal or Increased	Decreased	Decreased	Decreased
Respiratory Rate	14 – 20	20 – 30	30 – 40	> 35
Urine Output (ml/hr)	>3	20 – 30	5 – 15	Negligible "Anuria"
CNS/Mental Status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and Lethargic

- Fluid resuscitation begins with a 2 L (Adult) or 20 ml/kg (child) IV bolus of isotonic crystalloid, typically Ringers's Lactate.
- For persistent hypotension, this is repeated once in an adult and twice in a child before red blood cells (RBCs) are administered.
- Urine output is a quantitative reliable indicator of organ perfusion. Adequate urine output is 0.5 ml/kg per hour in an adult, and 1 ml/kg per hour in a child. ⓘ

- Based on the initial response to fluid resuscitation, hypovolemic injured patients can be separated into three broad categories:
  1. **Responders** "BP will stabilize"
  2. **Transient responders** "BP will improve and then it will fall down again which means there an active bleeding"
  3. **Non-responders** "which means there is a major bleeding that you can't control by resuscitation."

Before you go to the secondary survey you have to make sure that there is no life threatening condition is missed.

4 SECONDARY SURVEY

- Once the immediate threats to life have been addressed, a thorough history is obtained and the patient is examined from top to toe to ensure that no wound, bruise or swelling is missed.
- The back and spine are examined with the patient "log-rolled", looking specifically for localized tenderness, swelling, bruising or a "step".
- The perineum is examined and a rectal examination is performed to evaluate for sphincter tone, presence of blood, rectal perforation, or high riding prostate, this is particularly critical in patients with suspected spinal cord injury, pelvic fracture, or transpelvic gunshot wounds. ⓘ
- Vaginal examination with speculum should be performed in women with pelvic fractures to exclude an open fracture.
- **<u>In edition to physical examination the following should be done:</u>**
  1. Continuous vital signs monitoring.
  2. Central venous pressure (CVP) Monitoring.
  3. ECG Monitoring.
  4. Nasogastric Tube Placement, which is contraindicated in complex maxillofacial injury or fractures of the base of the skull and should be passed orally.
     - It evaluates the stomach content for blood, which may suggest gastro- duodenal injury.
     - If it passed to the chest it may suggest diaphragmatic injury.
  5. Foley Catheter Placement:
     - To monitor the urine output-Foley Catheter placement should be deferred after urological evaluation in patients with signs of urethral injury (Blood *at the meatus, perineal or scrotal hematoma, or a high riding prostate on the PR exam.*).
  6. Repeat FAST as needed.
  7. Laboratory Measurement.
  8. Radiographs:
     - Selective radiography and laboratory tests are done early after the primary survey.
     - For patients with severe blunt trauma the following radiograph should be done:
       1. Lateral Cervical Spine X-R
       2. Chest X-R
       3. Pelvis X-R

- For patients with truncal gunshots wound, anteriorposterior and lateral radiographs of the chest and abdomen should be done with marking the entrance and exit sites with metallic clips or stables.
- In critically injured patient we obtain blood sample for:
    1. Type and Cross- Matching.
    2. Complete Blood Count
    3. Blood Chemistry
    4. Coagulation Studies
    5. Lactate Level
    6. Arterial Blood Gas Analysis “ABG”

5 MCQS

1. Which of the following is true concerning traumatic pneumothorax:
   a. Needle aspiration is the treatment of choice
   b. The commonest type of chest injury
   c. Definitive treatment by intercostals tube drainage
   d. The main factor causing respiratory distress in flail chest
   e. Present with stridor

2. Two hours post Rt subclavian central venous catheter insertion, patient started to complain of Rt sided chest pain, shortness of breath, taccypnea, and he is tacchycardic. There is decrease air entry to the Rt side. The most likely diagnosis is:
   a. Hemothorax
   b. Air embolism
   c. Line was inserted in the artery
   d. Pneumothorax

3. The proper management of the previous patient is:
   a. Chest tube
   b. Removal of the line
   c. Intubation
   d. Aspiration of the gas from the line

4. Immediate life-saving attention is required for a trauma victim who suffers any of the following conditions except:
   a. Airway obstruction
   b. Fracture of the femoral shaft
   c. Massive flail chest
   d. Open pneumothorax
   e. Tension pneumothorax

5. The most common chest injury is:
   a. Pneumothorax
   b. Hemothorax
   c. Sternal fracture
   d. Rib fracture
   e. Pulmonary contusion

6. In laparoscopic procedures:
   a. CO is used for insufflation
   b. The umbilical trocar is commonly used for the camera
   c. Bowel perforation occurs more commonly with the open method for trocar insertion
   d. Diathermy is not used because of risk of explosion
   e. The pressure in the abdomen can be raised safely up to 35mmHg

7. The following are adverse prognostic factors in head injury except:
   a. Hypertension.
   b. Poor Glasgow Coma Score on admission.
   c. Hypotension.
   d. Age > 65 years.
   e. Non of the above

8. The Glasgow coma scale (GCS) is dependent upon the following except:
   a. Response to speech
   b. Response to pain
   c. Response of the pupils
   d. Best response
   e. Response of the patient

9. Severe head injury is defined as Glasgow Coma Score (GCS) of:
   a. 3
   b. 3-9
   c. 10
   d. 11-12
   e. 13-15

10. The outcome of head injuries:
    a. Depends upon the severity of head injury
    b. Most of minor head injured are able to return to work
    c. Only about a third of severe head injured make good recovery
    d. All the above
    e. None of the above

11. The adverse prognostic factors for the development of acute subdural haematoma include the following except:
    a. Old age
    b. Young age
    c. Chronic alcoholism
    d. Skull fracture
    e. Anticoagulation therap

12. The source of bleeding in the subdural space are all true except:
    a. Bridging veins into the superior sagittal sinus.
    b. Bridging vein of Labbe
    c. Cerebral contusion
    d. Sinus bleeding
    e. Fracture haematoma

13. Extradural haematoma, (the following are correct except):
    a. Is more common than acute subdural haematoma
    b. Is more often associated with vault skull fracture
    c. Is caused by rupture of bridging veins
    d. Is more likely to expand
    e. Is more likely to be arterial in origin

14. Mortality from trauma occurs mostly:
    a. Immediately at the scene
    b. During the first hour from the accident
    c. After admission to the hospital
    d. In the intensive care unit (ICU)
    e. Just before discharge from the hospital

15. Which one of the following is a life threatening injury
    a. Pneumothorax
    b. Hemothorax
    c. Head injury
    d. Fractured ribs
    e. Compound fracture of the leg

16. Paradoxical breathing is associated with:
    a. Bilateral ribs fracture
    b. Massive hemothorax
    c. Tension pneumothorax
    d. Hypoxemia
    e. Pulmonary contusion

17. A polytraumatized patient who arrives to the emergency department alive but unconscious is best managed by:
    a. Adrenalin infusion
    b. Blood transfusion
    c. Cardiopulmonary compressions
    d. Endotracheal intubation
    e. Intravenous fluids resuscitation

18. In hemodynamically stable trauma patient, intraabdominal injury is best assessed by:
    a. Clinical abdominal examination
    b. CT scan
    c. Diagnostic peritoneal lavage (DPL)
    d. Four quadrants peritoneal tapping

19. The best method to stop continuous bleeding from pelvic fracture is by:
    a. Applying mass trousers
    b. Insertion of external fixators
    c. Internal fixation
    d. Internal pelvic packing

🗝 Answers: 1:d, 2:a, 3:a, 4:b, 5:d, 6:b, 7:e, 8:c, 9:b, 10:d, 11:b, 12:e, 13:c, 14:b, 15:c, 16:a, 17:d, 18:b, 19:b.

TRAUMA CARE

1 INTRODUCTION

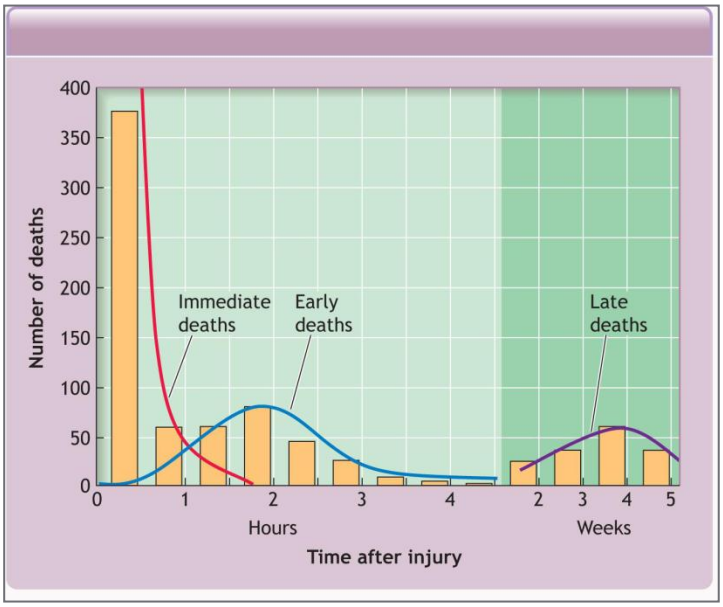
1.1 COURSE OBJECTIVES

- Importance of Trauma Care
- Principles of primary and secondary assessments.
- Establish management priorities

1.2 THE NEED

- The leading cause of death in the first four decades of life
- More than 5 million trauma-related deaths each year worldwide.
- Motor vehicle crashes cause over 1 million deaths per year. (we don't call it accidents b/c it's preventable)
- Injury accounts for 12% of the world's burden of disease.

1.3 TRIMODAL DEATH DISTRIBUTION



Immediate: you have to prevent it (seatbelt)!

- Early: Get the patient to the hospital (golden hour)

- Late: complications e.g. infection, multi-organ failure

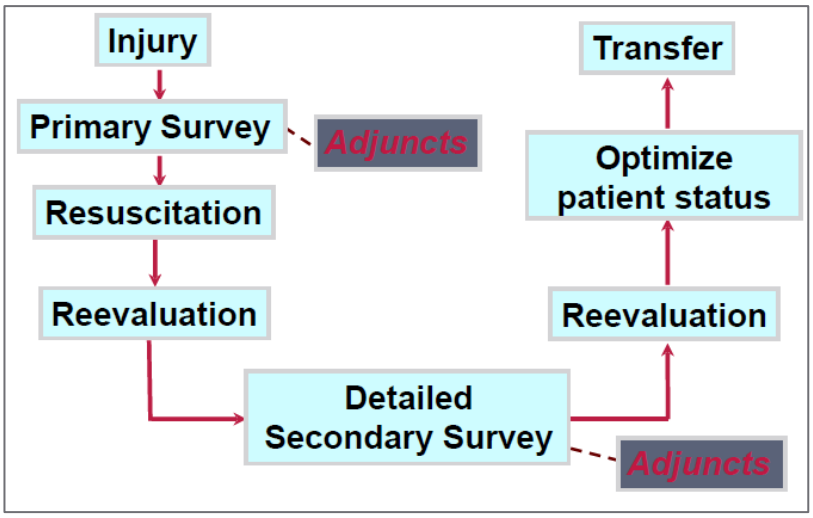
2 ADVANCED TRAUMA LIFE SUPPORT CONCEPT (ATLS)

- ABCDE approach to evaluation and treatment
- Treat greatest threat to life **first**
- Definitive diagnosis **not** immediately important
- Time is of the essence
- Do no further harm

2.1 ABCDE APPROACH

- **A**irway with c-spine protection
- **B**reathing / ventilation / oxygenation
- **C**irculation: stop the bleeding!
- **D**isability / neurological status
- **E**xpose / **E**nvironment / body temperature

2.2 INITIAL ASSESMENT AND MANAGEMENT



- Primary survey and resuscitation of vital functions are done simultaneously using a team approach.

2.2.1 QUICK ASSESSMENT

What is a quick, simple way to assess a patient in 10 seconds?

- Identify yourself
- Ask the patient his or her name
- Ask the patient what happened

An appropriate response to the previous question confirms the following:

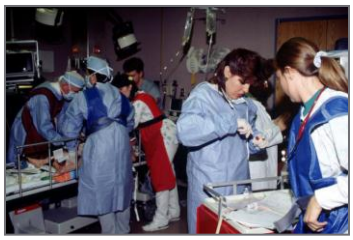
- Patent's Airway
- Sufficient air reserve to permit speech
- Sufficient perfusion to permit cerebration
- Clear sensorium

2.3 ADVANCED TRAUMA LIFE SUPPORT OBJECTIVES

- Apply principles of primary and secondary surveys
- Identify management priorities
- Institute appropriate resuscitation and monitoring procedures
- Recognize the value of the patient history and biomechanics of injury
- Anticipate and manage pitfalls

2.4 STANDARD PRECAUTIONS

- Cap
- Gown
- Gloves
- Mask
- Shoe covers
- Goggles / face shield



3 PRIMARY SURVEY

The priorities are the same for all patients

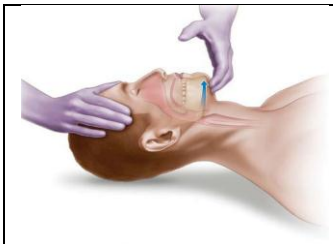
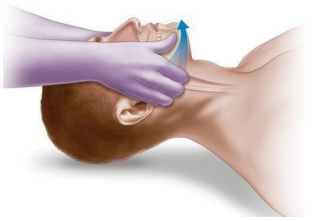
- **A**irway with c-spine protection
- **B**reathing with adequate oxygenation
- **C**irculation with hemorrhage control
- **D**isability
- **E**xposure / **E**nvironment

Special Considerations

- Trauma in the elderly
- Pediatric trauma
- Trauma in pregnancy

3.1 AIRWAY

- Establish patent airway and protect c-spine
- Basic Airway Techniques:
    1. Chin-lift Maneuver
    2. Jaw-thrust Maneuver

	*Chin-lift Maneuver*
	*Jaw-thrust Maneuver*

- Advanced Airway Techniques: orotracheal intubation
- Pitfalls (Unexpected difficulties):
  - Occult airway injury
  - Progressive loss of airway
  - Equipment failure
  - Inability to intubate

3.2 BREATHING

Assess and ensure adequate oxygenation and ventilation

- Respiratory rate
- Chest movement
- Air entry
- Oxygen saturation
- Pitfalls (Unexpected difficulties):
  - Airway versus ventilation problem?
    - Iatrogenic pneumothorax or tension pneumothorax

3.2.1 THE IMMEDIATE LIFE THREATENING INJURIES

- Laryngeotracheal injury / Airway obstruction
- Tension pneumothorax
- Open pneumothorax
- Flail chest and pulmonary contusion
- Massive hemothorax (> 1.5 L)
- Cardiac tamponade

3.2.2 MANAGEMENT

- **Where to insert chest tube?**
  - *5th intercostal space, anterior to the mid axillary line.*
- **How to manage tension pneumothorax?**
  - Needle to the 2nd intercostal space at the mid axillary line (needle thoracostomy) followed by Chest tube!
- **How to manage open pneumothorax?**
  - *Placement of dressing secured on 3 sides to create (flutter-valve) because securing on 4 sides will cause tension pneumothorax, a chest tube distant from injury must then be placed.*
- **How to manage hemothorax?**
  - *Chest tube, if the bleeding didn't stop, the patient must be taken to the OR*
- **How to manage cardiac tamponade in trauma?**
  - *Heart injured, needle pericardiocentasis or pericardial window can be immediately life-saving. Thoracostomy is the definitive treatment with repair of injury*

3.3 CIRCULATION

- Level of consciousness
- Skin color and temperature
- Pulse rate and character

3.3.1 CIRCULATORY MANAGEMENT:

- Control hemorrhage
- Restore volume
- Reassess patient
- **Lethal triad: [Hypothermia, Coagulopathy and Acidosis]** ⓘ
- Pitfalls (Unexpected difficulties):
  - Elderly
  - Children
  - Athletes
  - Medications

3.3.2 WHAT ARE THE CAUSES OF HYPOTENSION IN TRAUMA? ⚐

- Bleeding in the chest - Dx: by Examination & X-ray
- bleeding in the abdomen - Dx: Fast , DPL, abdominal distention
- Bleeding in the pelvis - pelvis is moving with hypotension!
- External bleeding
- bleeding at the site of trauma

3.4 DISABILITY ⚐

- Baseline neurologic evaluation
- Glasgow Coma Scale Score
- Pupillary response
- Observe for neurologic deterioration

Table 21.21 Glasgow Coma Scale

	Score
Eye opening (*E*)	
Spontaneous	4
To speech	3
To pain	2
No response	1
Motor response (*M*)	
Obeys	6
Localizes	5
Withdraws	4
Flexion	3
Extension	2
No response	1
Verbal response (*V*)	
Orientated	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1
Glasgow Coma Scale = $E + M + V$ (GCS minimum = 3: maximum = 15)	

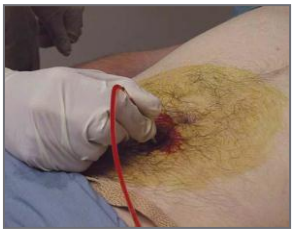
3.5 EXPOSURE / ENVIRONMENT ⚐

- Completely undress the patient
- Prevent hypothermia
- Check for missed injuries

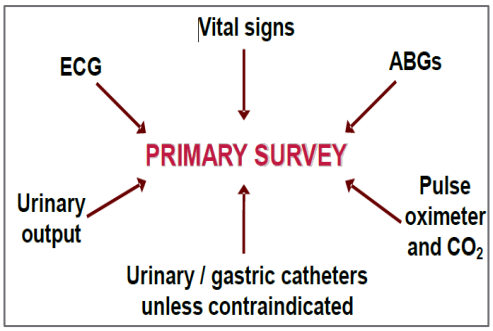
4 RESUSCITATION

- Protect and secure airway
- Ventilate and oxygenate
- Stop the bleeding!
- Vigorous shock therapy
- Protect from hypothermia

5 ADJUNCTS TO PRIMARY SURVEY



- **X-rays to any trauma patient:**
  - C-spine x-ray
  - Chest x-ray
  - Pelvic x-ray
- **Diagnostic Tools:**
  - *FAST* (Focused Assessment with Sonography for Trauma)
  - *DPL* (diagnostic peritoneal lavage)
- **Consider Early Transfer**
  - Use time before transfer for resuscitation
  - Do not delay transfer for diagnostic tests
- **Primary survey should take 10 – 20 minutes**



6 SECONDARY SURVEY

- The **complete** history and physical examination.

When do I start the secondary survey?

- Primary survey is completed
- ABCDEs are reassessed
- Vital functions are returning to normal

6.1 COMPONENTS OF THE SECONDARY SURVEY:

- History: Allergies, Medications, Past illnesses, Last meal, Events / Environment / Mechanism.
- Physical exam: Head to toe
- Complete neurologic exam
- Special diagnostic tests
- Reevaluation

6.1.1 HEAD

- External exam
- Scalp palpation
- Comprehensive eye and ear exam
- Including visual acuity

Pitfalls:

- Unconsciousness
- Periorbital edema
- Occluded auditory canal

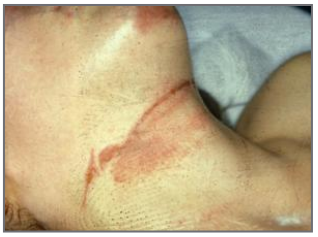
6.1.2 MAXILLOFACIAL

- Bony crepitus
- Deformity
- Malocclusion

Pitfalls:

- Potential airway obstruction
- Cribriform plate fracture
- Frequently missed

6.1.3 NECK (SOFT TISSUES)

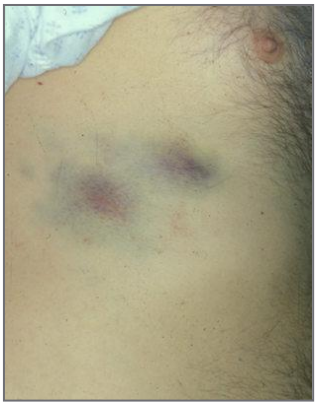


- Mechanism: Blunt vs penetrating
- Symptoms: Airway obstruction, hoarseness
- Findings: Crepitus, hematoma, stridor, bruit

Pitfalls:

- Delayed symptoms and signs
- Progressive airway obstruction
- Occult injuries

6.1.4 CHEST

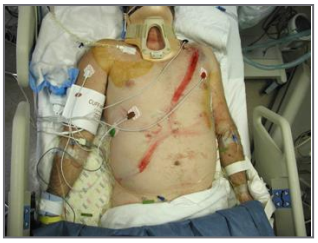


- Inspect
- Palpate
- Percuss
- Auscultate
- X-rays

Potential life threatening injuries:

- Blunt cardiac injury
- Traumatic aortic disruption
- Blunt esophageal rupture
- Traumatic diaphragmatic injury

6.1.5 ABDOMEN



- Inspect / Auscultate
- Palpate / Percuss
- Reevaluate
- Special studies

Pitfalls:

- Hollow viscous injury
- Retroperitoneal injury

6.1.5.1 INDICATIONS FOR LAPAROTOMY – BLUNT TRAUMA:

- Hemodynamically abnormal with suspected abdominal injury (DPL /FAST)
- Free air
- Diaphragmatic rupture
- Peritonitis
- Positive CT

6.1.5.2 INDICATIONS FOR LAPAROTOMY – PENETRATING TRAUMA:

- Hemodynamically abnormal
- Peritonitis
- Evisceration
- Positive DPL, FAST, or CT

6.1.6 PERINEUM

- Contusions, hematomas, lacerations, urethral blood

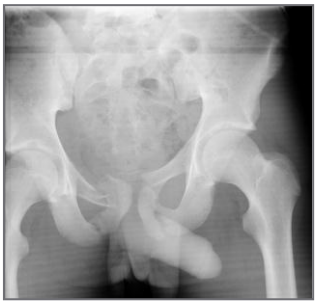
6.1.7 RECTUM

- Sphincter tone, high-riding prostate, pelvic fracture, rectal wall integrity, blood

6.1.8 VAGINA

- Blood, lacerations, Urethral injury, Pregnancy

6.1.9 PELVIS

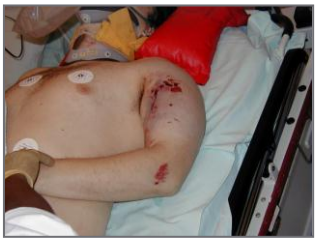


- Pain on palpation
- Leg length unequal
- Instability
- X-rays as needed

Pitfalls:

- Excessive pelvic manipulation
- Underestimating pelvic blood loss

6.1.10 EXTREMITIES



- Contusion, deformity
- Pain
- Perfusion
- Peripheral neurovascular status
- X-rays as needed

6.1.11 MUSCULOSKELETAL

- Potential blood loss
- Missed fractures
- Soft tissue or ligamentous injury

- Compartment syndrome (especially with altered sensorium / hypotension)

6.1.12 NEUROLOGICAL EXAM

- Brain
  - GCS
  - Pupil size and reaction
  - Lateralizing signs
  - Frequent reevaluation
  - Prevent secondary brain injury (Early neurosurgical consult)
- Spinal Assessment
  - Whole spine
  - Tenderness and swelling
  - Complete motor and sensory exams
  - Reflexes
  - Imaging studies
- Spine and Cord: Conduct an in-depth evaluation of the patient's spine and spinal cord

Early neurosurgical consult

Altered sensorium
- Inability to cooperate with clinical exam

Early neurosurgical orthopedic consult

6.2 ADJUNCTS TO SECONDARY SURVEY

- Special Diagnostic Tests as Indicated

Pitfalls:

- Patient deterioration
- Delay of transfer
- Deterioration during transfer
- Poor communication

6.3 HOW TO MINIMIZE MISSED INJURIES?

- High index of suspicion
- Frequent reevaluation and monitoring

7 PAIN MANGMENT

- Relief of pain / anxiety as appropriate
- Administer intravenously
- Careful monitoring is essential

8 TRANSFER

Which patients do I transfer to a higher level of care?

- Those whose injuries exceed institutional capabilities:
  1. Multisystem or complex injuries
  2. Patients with comorbidity or age extremes

When should the transfer occur?

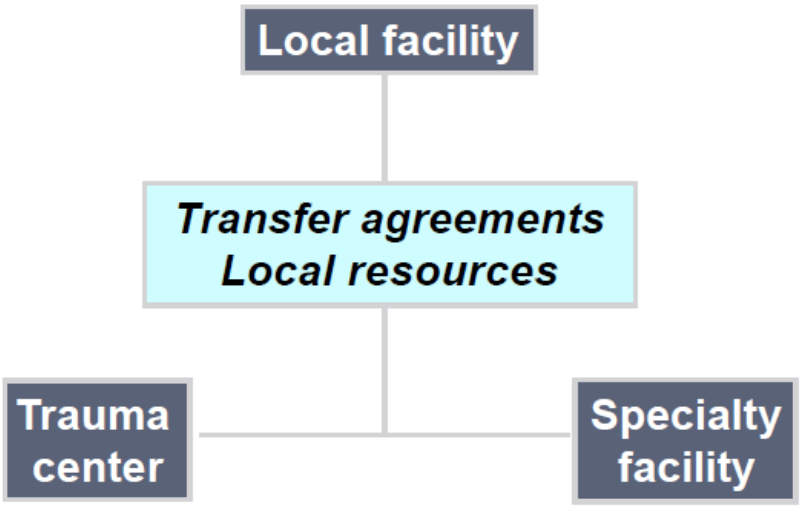
- As soon as possible after stabilization

Which patients do I transfer to a higher level of care?

- Airway and ventilatory control

- Hemorrhage control

8.1 TRANSFER TO DEFINITIVE CARE



SPECIFIC ORGAN ABDOMINAL TRAUMA

1 OVERVIEW

- Trauma is a major cause of death after IHD and malignancies.
- Trauma is considered the leading cause of death in the young population with ages that vary between 1 and 35 years.
- If trauma didn't cause death, it can cause any type of disability for a large group of people per minute.
- Trauma care account up to 7% of all hospital care which is a big budget.

There is no specific organ trauma, so abdominal trauma comes with multiple traumas.

1.1 ROAD TRAFFIC ACCIDENTS

- Road traffic accident (RTA) is one of the good examples of trauma and it is the most common trauma.
- RTA is the third leading cause of death after IHD and cancer.
- How to reduce trauma due to RTA?
  - Roads should be in a good shape.
  - Every person should make a complete check up for his car every once in a while, such as: breaks, water the car, wheels... etc.
  - Drivers should follow the rules which include: wearing the seat belt, not to drive while person is drunk or on drugs, follow the road signs, not to exceed the assigned speed and so on.
  - Medical care: there should be very good equipments/machines at the ERs with qualified staff.

2 TYPES OF TRAUMA

- There are different types of traumas; the types are classified according to the Mechanism of trauma.
- A patient can either get one of these types or a combination of them.
- Types of trauma:
  - Blunt trauma: Road traffic accident is the major cause of blunt trauma.
  - Penetrating
  - Burns
  - Blast

Blunt trauma: injury incurred when the human body hits or is hit by a large outside object (as a car).

Blast trauma: injury caused by the explosion of a bomb (especially in enclosed spaces)

3 ABDOMINAL TRAUMA

- The majority of abdominal injuries are due to blunt abdominal trauma (90%) secondary to high speed automobile accidents.

3.1 ANATOMICAL REGIONS OF THE ABDOMEN

- Peritoneum:
  - Intra thoracic abdomen:
    It is under the costal margin. Contains the liver, spleen, stomach, and pancreas.
    It is hard to examine it.
  - True abdomen:
    It can be clinically examined.
- Retroperitoneal:
  - Pancreas & Duodenum
  - Bowel
  - Vascular(IVC , aorta)
  - Kidneys, ureter
  - Pelvic abdomen: bladder, female genital system
  - It is not accessible during physical examination, investigations are needed.

3.2 TYPES OF THE ABDOMINAL TRAUMA

- Blunt abdominal trauma: (take about 90 % of trauma)
  - Sometimes doctors miss such cases because a patient with a blunt abdominal trauma can come walking to the ER. Some doctors take superficial history and physical examination and let the patient go home without admitting him. At the mean time, the patient would be bleeding slowly from the inside and in an hour he would collapse. Patients who come to the ER because of trauma should be examined from head to toe whether they came walking and conscious or not.
- Penetrating abdominal trauma: It is to diagnose and manage.

3.3 MANAGEMENT OF TRAUMA PATIENTS

- The primary management of abdominal trauma is determination that an intra abdominal injury exists and operative intervention is required.
- Many deaths would have been preventable if there wasn't a failure in managing the abdominal injuries.
- Causes of the failure of management includes:
  1- Delay in ambulance to arrive, traffic jam, wrong place of hospitals, no good qualified hospital, and non well equipped hospitals.
  2- Many patients die because doctors don't do ABC
- When you receive a trauma case always assume that there is injury even if the patient came walking to you until proven otherwise by history clinical presentation and investigations.

3.3.1 PRIMARY SURVEY

- The resuscitation & Management priorities of patient with major abdominal trauma are:
  1) ABCDE of emergencies (must be done to all trauma patients):
     - Airway: intubation if the airway is damaged.
     - Breathing: if breath sounds were absent, insert a chest tube immediately**.** No O2 for 15 minutes will cause a disability**.**
     - Circulation: If there was bleeding (hemorrhage), control should be initiated. Give IV fluids (usually crystalloids and normal saline) and control the bleeding.
     - Disabilities
     - Exposure: cut the clothes.
  2) Usage of Nasogastric tube. It is contraindicated if there was bleeding from the nose or mouth.
  3) Usage of urinary catheter to monitor the output and input. It is contraindicated If there was bleeding from the urethra.

3.3.2 SECONDARY SURVEY

- History: History is taken from the patient himself, if he was conscious, if not it is taken from the person who attended or the paramedic.
  - Blunt trauma
  - Penetrating trauma → immediately to surgery
- Physical examination: General and abdominal examination
- Sometimes there is no time for Secondary survey.
- Abdominal Examination: Inspection, Palpation, Percussion, Auscultation, Rectal Examination, and Vaginal Examination.

3.3.2.1 INVESTIGATIONS:

- Blood Tests
- Radiological Studies (Plain abdominal X-ray , CXR)
- Diagnostic Peritoneal Lavage (DPL):
  - Indicated when the patient is in a shock or suffering from abdominal distention.
  - It is extremely reliable; it can determine the presence of blood in the peritoneal cavity up to 98% of the cases.
  - When positive take the patient to the OR immediately.
  - If the results weren't so accurate and clear, insert a liter of saline and if fresh blood appears then it is positive.
- If the patient is stable you do:
  - USG abdomen
  - CT abdomen
  - Peritoneoscopy (diagnostic laparoscopy)

3.3.3 INDICATIONS FOR SURGERY- LAPROTOMY

1. If there were any signs of peritoneal injury such as tenderness, distention, guarding, bruising and so.
2. Unexplained shock (If you give a lot of fluid but your patient is still in a shock).
3. Evisceration of viscus (If the bowel was out).
4. Positive DPL – diagnostic peritoneal lavage.
5. Determination of finding: During routine follow up on investigations.
    a. Sometimes you need to admit the patient for observation or admit them to the ER for 6 hours then signs will start to appear.
    b. Ex: Patient came conscious with injury for conservative therapy to the ER and got admitted, after 4-6 hours he went into a shock.

4 SPECIFIC ORGAN TRAUMA

- Peritoneal:
    - Liver: protected by ribs.
    - Spleen: it is a mobile organ.
    - Kidneys: in the retroperitoneal, it is not easy to injure so if it was injured it will be a severe trauma.
    - Bowel
- Retroperitoneal:
    - Pancreas & Duodenum
    - Bowel
    - Vascular(IVC , aorta)
    - Kidneys, ureter
- Geneto-urinary system:
    - Urinary bladder, urethera (it is easy to diagnose if there was a fracture in the pelvis)
    - Female reproductive system

4.1 LIVER TRAUMA

If you found a gunshot or a stab in the fifth intercostal space, assume that the liver is injured.

- Liver is the largest organ in the abdominal cavity "5th intercostal space"
- Any trauma under the nipple we expect liver; it means the liver is injured.
- Most commonly injured organs in all patients with abdominal Trauma.
- Commonest organ injured in case of penetrating trauma.

4.1.1 MECHANISM OF INJURY

- In blunt trauma:
    - Hepatic injuries result from direct blows
    - compression between the lower ribs on right side and the spine
    - Shearing at fixed points secondary to deceleration.
    - Any penetrating gunshot, stab or shotgun wound below the right nipple on right upper quadrant of the abdomen is also likely to cause a hepatic injury.

4.1.2 DIAGNOSIS AND INVESTIGATIONS

- Clinical manifestations:
  - Often made at laparotomy in patients presenting with penetrating injuries requiring immediate Surgery or in shock.
  - Blunt Trauma: patients who remain in a shock or present with abdominal rigidity, you do no further investigation and you take him to the OR immediately.
- Investigations:
  - Diagnostic peritoneal lavage (DPL)
  - CT Scan abdomen: used to diagnose intra peritoneal injuries in stable patients after blunt trauma

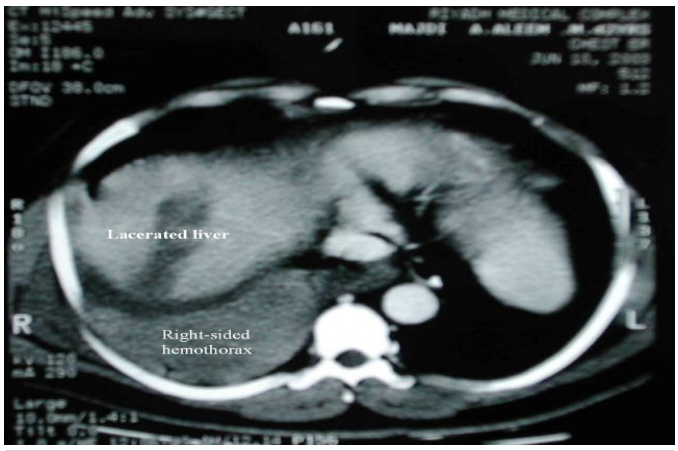


Figure: Gunshot below the nipple, Right side hemothorax, grade 3. The patient is stable; there is no blood in the peritoneal cavity. In this case the patient will go with conservative management, if he bleeds, he must be taken to the OR and if there was another injury take him to the OR and deal with all the injuries.

4.1.3 MANAGEMENT

- When the patient comes to ER, the initiate management should be uniform:
- ABCDE: regardless what injury you have.
- Non-operative approach:
  - Not all patients with liver injury need operation. It is determined by CT scan.
  - The criteria for non-operative approach is:
    - Simple hepatic laceration Or intra hepatic hematoma
    - No evidence of active bleeding
    - Intra peritoneal blood loss less than 250 ml
    - Absence of other Intra peritoneal injuries " spleen , bladder,.." that requires surgery
- Operative approach:

- Persistent hypotension, despite adequate volume replacement, suggests ongoing blood loss and mandates immediate operative intervention
- Classification: This classification is based on operative findings and management. So hepatic injury is classified into:
  a. Grade 1: Simple injuries – non bleeding. Conservative treatment if no bleeding or other injuries.
  b. Grade 2: Simple injuries managed by superficial suture alone if you opened the patient. Conservative treatment if no bleeding or other injuries.
  c. Grade 3: Major Intra parenchymal with active bleeding but not requiring inflow occlusion (Pringle maneuver) to control hemorrhage. Some of the patients go for conservative treatment others go for OR.
  d. Grade 4: Extensive Intra parenchymal injury with major active bleeding requiring inflow occlusion for haemostatic control. Needs operation and do Pringle manoeuvre.
  e. Grade 5: Juxtahepatic venous injury (injuries to retrohepatic cava or main hepatic veins) portal vein injury. Patients in this grade are less likely to survive
- All patients undergoing laparotomy for trauma should be explored through midline incision (from xiphisternum to pubic" around the umbilicus go up or down) because you do not know where is the lesion.
- Management according to classification:
  a. Grades 1 & 2: Simple injuries can be managed by any one of variety of methods: if we open it simple suture, electrocautery, tropical hemostatic agents, etc. Does not require drainage.
  b. Grade 3: Major intra-parenchymal injuries with active bleeding can be managed best by Finger Fracturing the hepatic parenchyma and ligating or repairing lacerated blood vessels & bile ducts under direct vision
  c. Grade 4: Extensive intraparenchynal injuries with major rapid blood loss require occlusion of portal trial to control hemorrhage. It might need liver resection, lobe resection, and ligation of intrahepatic artery. It is rarely saved.

The Pringle maneuver is a surgical maneuver used in some abdominal operations. A large hemostat is used to clamp the hepatoduodenal ligament interrupting the flow of blood through the hepatic artery and the portal vein and thus helping to control bleeding from the liver.

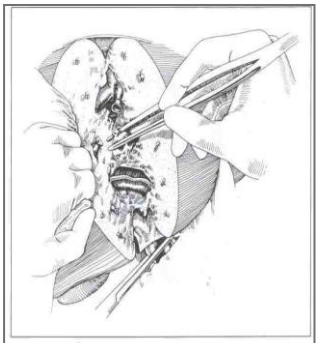


Figure 30–4.
Nonviable portions of the liver are debrided. Debridement ceases only when bleeding viable hepatic parenchyma is encountered.

Figure:

Finger fraction: the injury in the liver is small, you will open the liver according to the injury, start ligating the blood vessels then ligating the ducts. Then omental packing: put omentum in between and suture it because it will cause hemostasis.

Summary:

- Simple techniques: Simple techniques include drainage only of non-bleeding injuries, application of fibrin glue, sutures “hepatorrhaphy” and application of surgical (I & II).
- Advanced techniques: Advanced Techniques of Repair (III & IV) all performed with Pringle Maneuver in place
- Types of repair:
    1) Extensive hepatorrhaply
    2) Hepatotomy with selective vascular ligation
    3) Omental pack
    4) Resectional debridement with selective vascular ligation
    5) Resection
    6) Selective Hepatic Artery Ligation “remember liver is regenerate”
    7) Peri-hepatic packing: If you can’t deal with a patient, just pack the patient and send him to a center where he will be treated. Also, if you did what you have to do but the bleeding didn’t stop, pack your patient and send him to another hospital.

4.1.4 COMPLICATIONS AND MORTALITY

- Recurrent bleeding
- Hematobilia: blood will go to the bile duct and the patient will bleed per rectum.
- Perihepatic abscess then Biliary Fistula later on.
- Billiary Fistula
- Intrahepatic Haematoma
- Pulmonary Complications
- Coagulopathy: because of a lot of blood transfusion.

4.2 SPLENIC TRAUMA

4.2.1 GENERAL CONSIDERATIONS

- The spleen remains the most commonly injured organ in patients who have suffered blunt abdominal trauma
- Involved frequently in penetrating wounds of the left lower chest and upper abdomen.
- Management of the injured spleen has changed radically over the past decade.
- The spleen is now recognized as an important immunological factory as well as a reticuloendothelial filter. The problem is when spleen has a disease; splenomegaly;- malaria, portal hypertension makes it more susceptible to be damaged from simple trauma and you will find the patient collapsed.
- Although the risk of over whelming post- splenctomy sepsis (OPSS) is greatest in children less than 2 years, recognition of OPSS has stimulated efforts to (Conserve spleen) by splenorrhaphy (either by repair or leave according to grade).

4.2.2 MECHANISM OF INJURY

- Commonly injured in patients with blunt abdominal trauma because of its mobility.
- Most civilian stab wounds and gunshot wounds cause simple lacerations through injuries.
- It is of interest that 2% of patients who are undergoing a surgery in the LUQ of the abdomen can get injured in the spleen by the surgeon causing a small injury by any of the surgical equipment being used by the doctor using or the assistant.

4.2.3 CLASSIFICATIONS

- The Magnitude of splenic disruption depends on the patient's age, injury mechanism and presence of underlying disease.
- Splenic injury has been classified according to its pathological anatomy into:
    - Grade I: Subcapsular hematoma.
    - Grade II: Sub segmental parenchgmal injury.
    - Grade III: Segmental devitalization (part of it)
    - Grade IV: Polar disruption (complete pole)
    - Grade V: Shattered or devascularized organ (autosplenectomy), Patient is in a shock but he can survive because of the blood supply.

4.2.4 DIAGNOSIS AND INVESTIGATIONS

- History
- Physical examination:
    - Sign & symptom: if you find any of these, you presume spleen and kidney injury:
        - LUQ bruising or abrasion
        - Left lower ribs fracture on CXR
        - Kehri's sign : shoulder tip pain (L shoulder)
        - Balance's sign : LUQ mass (hematoma)
- Radiological:
    - CXR " very important in case of spleen injury "
    - Plain abdominal X-Ray
    - CT Scan: it is the most important investigation in spleen injury. (Done if the patient is stable)
    - Angiography: it is very important for grading. It can be used for diagnosis and a therapeutically. Done if the patient is stable.
    - Picture -1-: it shows grade 1 hematoma – patient is stable so go with conservative therapy.

Kehri's sign is the occurrence of acute pain in the tip of the shoulder due to the presence of blood or other irritants in the peritoneal cavity when a person is lying down and the legs are elevated.

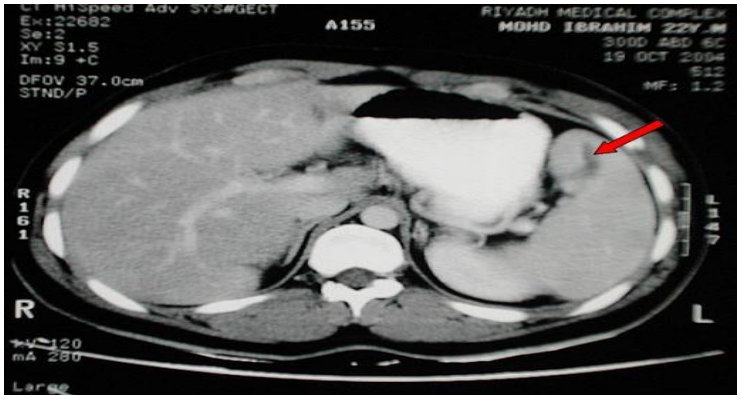
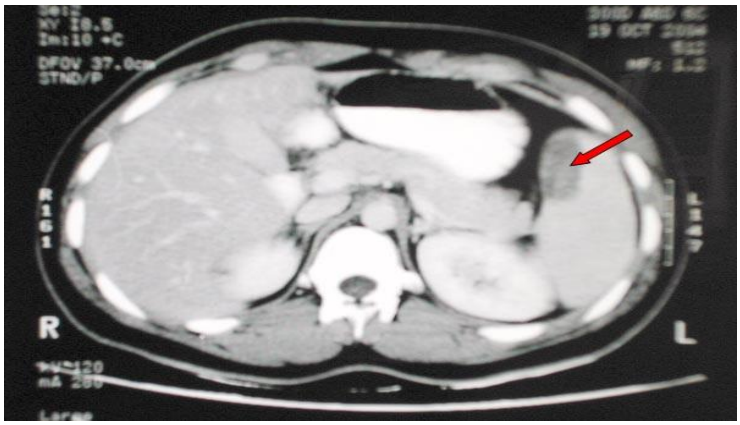


Image showing a grade 2 – laceration but the wall is not disrupted. Go with conservative therapy.



4.2.5 TREATMENT

- ABCDE
- Non-operative approach:
  - Widely practiced in pediatric trauma
  - criteria for non-operative approach :
    - Haemodynamically stable children / adult (not in a shock)
    - Those patients who do not have any peritoneal findings at any time (no rigidity, no tenderness, just bruising).
    - Those who did not need more than two units of blood (more than 2 go to OR)
- Operative approach:
  - Decision to perform splenctomy or splenorraphy is usually made after assessment & grading the splenic injury
  - Contra indication for splenic salvage: (perform splenoctomy)
    - The patient has protracted hypotension (Everything is done but there is no response and the patient is still bleeding)
    - Undue delay is anticipated in attempting repair the spleen (if we put a needle patient will bleed)
    - The patient has other severe injuries (in the liver, bowel, or bladder)

4.2.6 COMPLICATIONS OF SURGERY

- Early complications occur such as:
  - Bleeding.
  - Acute gastric distention.
  - Gastric necrosis (short gastric vessels are close to each other so when you ligate them, it might lead to necrosis.)
  - Recurrent splenic bed bleeding.
  - Pancreatitis (remember the tail of pancreas ends at the pelvis of the spleen)
  - Subpherinic abscess
- Late complications occur such as:
  - Thrombocytosis
  - OPSS (1 – 6 Week)
  - DVT

4.3 RENAL TRAUMA

4.3.1 GENERAL CONSIDERATIONS

- The commonest organ prone to injury in the urinary system.
- If contusion occurs, it can be treated by conservative therapy.
- If hematuria is present, it means there is a poor indicator of severe renal injury (complete or partial kidney damage)

4.3.2 DIAGNOSIS: MEASUREMENT OF MEAN ARTERIAL PRESSURE

- Symptoms and signs (3 Fs) :
  - Flank abrasion
  - Fracture of the ribs
  - Fracture vertebral transverse process
- Investigation: Intravenous urography (IVU) + CT scan.

4.3.3 MANAGEMENT

- For minor injuries such as hematoma: US scan, percutanous drainage, antibiotic usage.
- For severe injuries: partial nephroctomy or total nephroctomy.

5 MCQS

1. **In abdominal injuries, the most informative initial investigation is:**
   A. CT
   B. Ultrasound
   C. <u>Diagnostic peritoneal lavage</u>
   D. Abdominal x-ray

2. Blunt trauma to the abdomen most commonly injures which of the following organs?

A. Liver
B. Kidney
C. <u>Spleen</u>
D. Intestine
E. Pancreas

Explanation: The diagnosis of injuries resulting from blunt abdominal trauma is difficult; injuries are often masked by associated injuries. Thus, trauma to the head or chest, together with fractures, frequently conceals intraabdominal injury. Apparently trivial injuries may rupture abdominal viscera in spite of the protection offered by the rib cage. The structures most likely to be damaged in blunt abdominal trauma are, in order of frequency, the spleen, kidney, intestine, liver, abdominal wall, mesentery, pancreas, and diaphragm.
Abdominal paracentesis is a rapid, sensitive diagnostic test for patients with suspected intraabdominal injury and may be extremely helpful in the management of patients with associated head, thoracic, or pelvic trauma in whom signs and symptoms of the abdominal injuries may be masked or overlooked. Abdominal CT scans, which should be done promptly and rapidly, are being used more frequently to evaluate these injuries.

3. Which of the following conditions is most likely to follow a compression-type abdominal injury?

A. Renal vascular injury
B. Superior mesenteric thrombosis
C. Mesenteric vascular injury
D. Avulsion of the splenic pedicle
E. <u>Diaphragmatic hernia</u>

Explanation: In the rapid deceleration injury associated with automobile crashes, the abdominal viscera tend to continue moving anteriorly after the body wall has been stopped. These organs exert great stress upon the structures anchoring them to the retroperitoneum. Intestinal loops stretch and may tear their mesenteric attachments, injuring and thrombosing the superior mesenteric artery; kidneys and spleen may similarly shear their vascular pedicles. In these injuries, however, ordinarily the intraabdominal pressure does not rise excessively and diaphragmatic hernia is not likely. Diaphragmatic hernia is primarily associated with compression-type abdominal or thoracic injuries that increase intraabdominal or intrathoracic pressure sufficiently to tear the central portion of the diaphragm.

RAISED INTRACRANIAL PRESSURE

1 INTRODUCTION

- The skull is like a rigid box that doesn't allow any expansions.
  - It contains: the brain (volume measured is 1400 ml), meninges, vessels, blood (volume measured is 75 ml), and CSF (volume measured 75 – 100ml). Water, blood, and CSF are incompressible.
- The pressure in the skull is called the Intra-cranial pressure (ICP). The ICP must stay balanced in order for the survival of the brain.

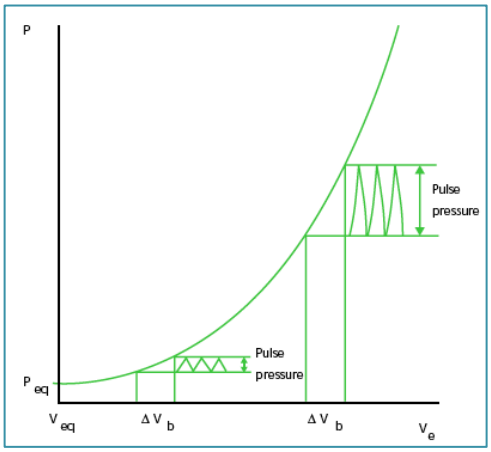
2 BASIC PRINCIPLES OF ICP

2.1 MONRO-KELLIE DOCTRINE

Doctrine means a particulate system of principles taught of advocated.

- This principle states that any change in the brain's volume is associated with a change in CSF or blood volume.
- When the volume of the brain increases, the other components will have to compensate. CSF will decrease and vasoconstriction will occur in order to decrease the blood volume.

2.2 VOLUME – PRESSURE CURVE



- Increase in the volume of one compartment will lead to change in the volume of the other ones.
- When the blood volume increases, the ICP will increase but the cranium's components' accommodation will keep it balanced until a certain level where even a small increase in the volume can take the curve over and will no longer be able to accommodate leaving the ICP with a sudden increase. At this point, symptoms such as headache, nausea, vomiting, numbness and weakness will start to occur.
- Assume that brain develops a tumor. Depending on how fast the tumor grows, the pressure will either increase slowly and be tolerated, or massively (ex, sudden hemorrhage) and lead to comatose.
- Example 1: A patient came to the ER complaining from headaches. He was conscious when the doctor talked to him and examined him. Few minutes later he collapsed and went into a coma. In this case, the patient was at the at edge of the pressure - volume curve

- Example 2: A patient with a brain tumor that grows 1 mm yearly, his brain will have enough time to accommodate, CSF will get a lot of time to change its absorption and production pattern, and the blood flow will change and has a lot of time to accommodate. Unlike a sudden change which isn't tolerated well.

2.3 ICP WAVEFORM

- Any pressure has a waveform.
- The ICP waveform corresponds to the cardiac waveform. So when systole occurs, there will be a rise in the ICP waveform. It gives the brain pulsation and this pulsation is what forms the ICP. Pulsation of heart is transmitted into the great vessel then into the internal carotid artery and into the brain.

2.4 NORMAL ICP VALUES

Because children have softer bones, their ICP value is lower. Infants have lesser ICP = 1.5 – 6 because their bones haven't united yet. up)

Table 1 Normal intracranial pressure values

Age group	Normal range (mm Hg)
Adults	<10–15
Children	3–7
Term infants	1.5–6

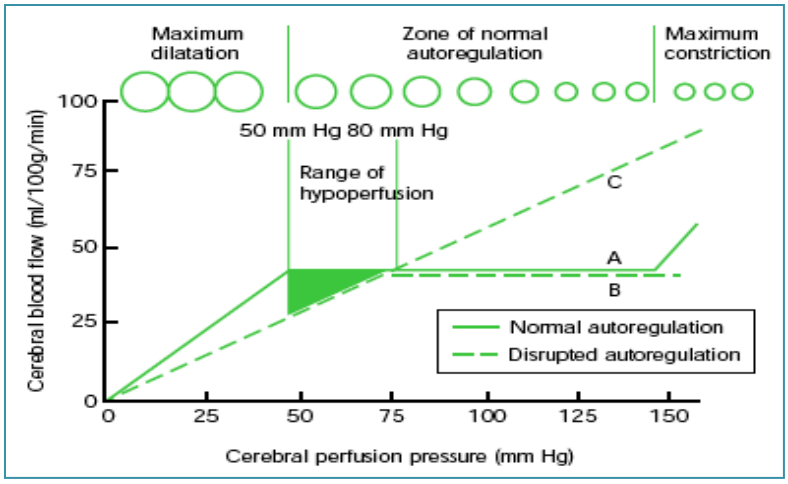
3 CEREBRAL PERFUSION PRESSURE

- Cerebral perfusion pressure is the pressure coming from the blood rushing into the brain.
- It has a very wide range between 50 and 140 mmHg
- In trauma cases, it is preferred to keep cerebral perfusion pressure around 70.
- The brain only dysfunctions at a very high extreme pressure or very low extreme pressure.

3.1 CEREBRAL AUTOREGULATION

- It is the ability of cerebral vessels to maintain cerebral perfusion within strictly determined limits.
- Mechanisms of cerebral auto-regulation:
  - A rise in the systolic blood pressure will cause constriction of cerebral arteries.
  - A drop in the systolic blood pressure will cause cerebral vessels to dilate for accommodation.
  - This will help keep the person conscious and able to judge.
  - Example: someone got dehydrated; s/he will still be able to go drink water. But if the brain collapsed, one won't be able to protect him/her self.

- If there is a repetitive increase in the pressure such as hypertension, the brain vessels will start to develop small aneurisms. They're very tiny aneurisms that develop on the small arterioles and at some point they rupture and cause hypertensive hemorrhage to the brain.
  - Loss of auto regulation will cause changes in cerebral blood flow with changes in the BP levels.
    - Increase of pressure will constantly increase the blood flow pressure. When it reaches to an extreme abnormal state, the auto regulation will then fail.
    - Example:. 25:25 , 50:50. 75: blood flow pressure remains constant.
- Disrupted auto-regulation: BBB or vessels badly affected like bad hematoma or bad contusion of brain. In that area, increased pressure will increase flow and this area can bleed inside.



3.2 MEASUREMENT OF CPP

MAP = mean arterial pressure and it's the blood coming from the body to the brain.

- The heart pumps the blood with pressure into the brain. The brain has its own pressure. The pressure that goes into the brain has to be the average pressure in the head subtracted from the average of the blood's pressure.

  CPP = MAP – ICP

3.2.1 MEASUREMENT OF MEAN ARTERIAL PRESSURE

- The systolic heart beats over the diastolic heart beats divided by 3.
- You can find out by using the blood pressure cuff and connecting it to a monitor in the ICU where it will show the results there.

3.2.2 MEASUREMENT OF ICP

- It is measured by inserting a catheter into the head.
- ICP can be adjusted by giving fluids or medications that can increase the pressure and by this the cerebral perfusion pressure can be maintained.
- Example: if a MAP of someone was = 85. And the ICP was = 15

Measure the CPP = 85 -15 = 70.
(Recommended to keep it above 70 in head injuries)

- In a case of trauma with bad head injury:
  ICP and MAP must be measured, CPP must be around 70. If it was around 40 then BP must be increased or ICP must be decreased.
- If ICP goes up, how does the brain get perfusion? Process of auto-regulation.

4 RAISED ICP

Any abnormal contents such as masses, tumors or hematoma will cause an increase in the pressure which will affect the brain.

4.1 CAUSES OF RAISED ICP

- Vitamin D → is an Acronym to remember the causes of raised ICP:
  Vascular, **I**nfection, **T**rauma, **A**utoimmune, **M**etabolic, **E**ndocrine, **N**eoplastic, **D**rugs
- ICP causes can be classified in several ways. It can be classified according to the structures over there. If the problem was in the brain causes can be tumor, traumatic contusion, CSF obstruction, obstructive hydrocephalus, thrombosis… etc
- Or classified according to major pathological criteria: infection, trauma, and tumor.

Table 2 Examples of causes of raised intracranial pressure

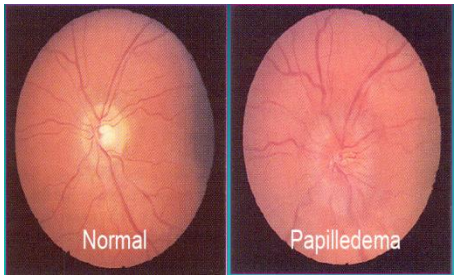
Pathological process	Examples
Localised mass lesions	Traumatic haematomas (extradural, subdural, intracerebral) Neoplasms (glioma, meningioma, metastasis) Abscess Focal oedema secondary to trauma, infarction, tumour
Disturbance of CSF circulation	Obstructive hydrocephalus Communicating hydrocephalus
Obstruction to major venous sinuses	Depressed fractures overlying major venous sinuses Cerebral venous thrombosis
Diffuse brain oedema or swelling	Encephalitis, meningitis, diffuse head injury, subarachnoid haemorrhage, Reye's syndrome, lead encephalopathy, water intoxication, near drowning
Idiopathic	Benign intracranial hypertension

4.2 SIGNS AND SYMPTOMS

- Vomiting
- Headaches:
  - Characteristics of the headache:
    - Early morning headaches. It is very characteristic. Once the patient wakes up with a really bad headache, when he's in his best situation. What happens? Patient was laying flat during sleeping which will increase venous return and the amount of

blood reaching the brain will increase. But when the patient is sitting upright, gravity will take blood down and ICP will decrease.
    - Throbbing / Bursting
    - It increases with sneezing and coughing. Coughing and sneezing will increase intrathroacic pressure which will keep the blood from coming down and this will increase the ICP
- Papilledema:
    - It's important to examine the fundus in patients with raised ICP.
    - This symptom is reliable but may take several days to develop.
        - Therefore when a patient comes to the ER with raised ICP, he is not examined for papilleodema first.
    - It can be associated with fundal hemorrhage and this indicates acute and severe rise in ICP.
    - It happens only with chronic problems like with growing brain tumor.

    - When you look into the eye you'll find a blurred optic disk because the venous return from the eye that's supposed to go to the head, wants to go but it's finding very high pressure so it becomes congested.
    - Thick congested veins cause edema.



    - Increase pressure in brain → veins within optic nerve become congested → whole optic nerve head becomes congested.
    - Very large tortures veins , elevated and floored optic disk margin can be seen.

- In Neurological exam the most common signs:
    - Hemiplegia (any weakness)
    - Cranial nerve deficit
    - Pupillary dilation:
        - It is one of the earliest signs that occur and it is very characteristic.
        - The pupil dilates or constricts based on the Occulomotor nerve that comes from the midbrain in the brainstem (figure -1-), just next to the temporal lobe.
        - So if the temporal lobe is pushed, it compresses the nerve. In the beginning of herniation, this nerve will be affected.

- If there is a mass compressing the 3rd nerve → ipsilateral pupil dilation and contralateral hemiplasia ”weakness” will occur.

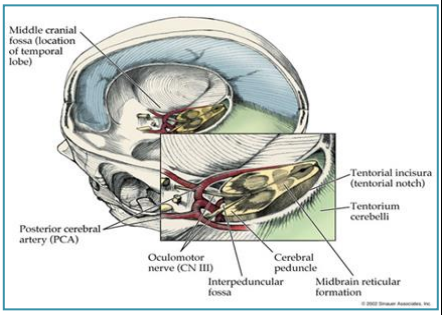


Figure 1: Coronal cut MRI shows u the same thing

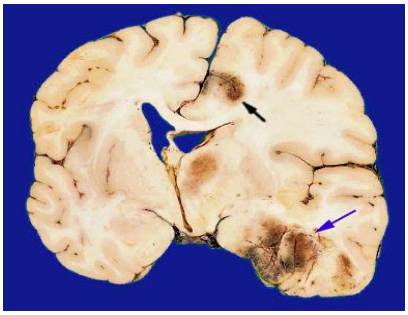


Figure 2: This pathological picture shows Cingulate herniation (subfalcine herniation), central herniation, and some Uncal herniation. Blood in temporal area, basal ganglia, frontal area.

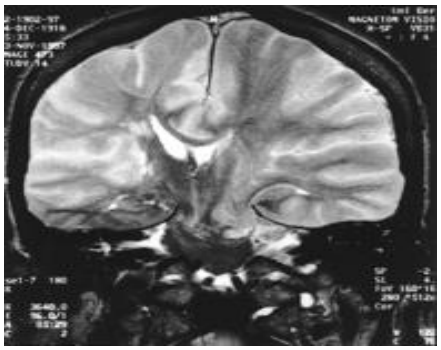


Figure 3

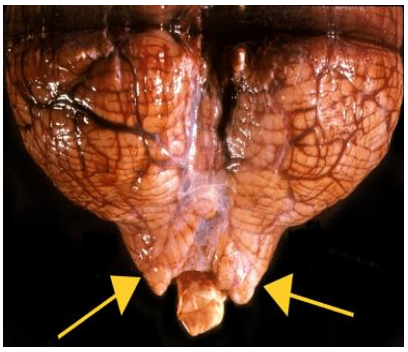


Figure 4: Tonsillar herniation, tonsil comes down from foramen magnum and bone compress it makes it flat

- Systemic: reaction to increased ICP:
  - Raised BP (recall: CPP=MAP-ICP) blood is pumping so high to compensate (you rise MAP by rising systolic BP) if you drop his Bp you Kill him.
  - When you have a raised ICP, if you increase the ICP how to maintain a good CPP? By increasing MAP.
  - Respiratory change:
    - Cheyne-Stokes breathing: not seen in every case
    - Oscillating periods of apnea-tachypnea.
    - A lot of pressure on the brainstem (stop breathing → suddenly breathing fast → again suddenly stop breathing...Etc).

- Raised ICP in infants results in: skull here isn't fully developed yet so it can accommodate:
    - Widened sutures
    - Increased Head circumference
    - Dilated head veins
    - "Sun set" eyes "his eyes always looking down" pushed down
    - Tense and bulging fontanels (normally flat and sunken except if he cries it bulge and come flat again)
    - Head is to large
    - A lots of Dilated veins

4.2.1 KERNOHAN'S NOTCH

- Simply, when there is a huge growing right side hematoma it will push the whole brain stem to the opposite side (it will push the whole brainstem against the contralateral tentorium) and that may cause ipsilateral weakness and contra-lateral dilated pupil.
- Don't take any patient to the OR room unless we do a CT to make sure.
- This sign is used to clinically estimate the side of bleeding.
- CT scan is done to know the exact location of the bleeding.

4.3 GLASGOW COMA SCORE

- It is very important for the assessment of the severity of coma.(so will be easier to estimate prognosis)
- It relies on 3 things: the ability to open the eyes, verbal responses and motor responses.
- If a patient's GCS was 3 (which is the lowest), he might die within days.
- If a patient's was GCS 14, he should be admitted to the hospital for 2 days then leave.
- When it comes to head injury there is a classification of GCS:
    - Mild GCS= 13 – 15
    - Moderate GCS= 9 – 12
    - Severe GCS= 3 – 8
    - The lowest number in GCS is 3 and the highest number is 15

Glasgow Coma Score		
Eye Opening (E)	**Verbal Response (V)**	**Motor Response (M)**
4=Spontaneous 3=To voice 2=To pain 1=None	5=Normal conversation 4=Disoriented conversation 3=Words, but not coherent 2=No words.....only sounds 1=None	6=Normal 5=Localizes to pain 4=Withdraws to pain 3=Decorticate posture 2=Decerebrate 1=None
		Total = E+V+M

4.4 RAISED ICP AND BRAIN HERNIATIONS

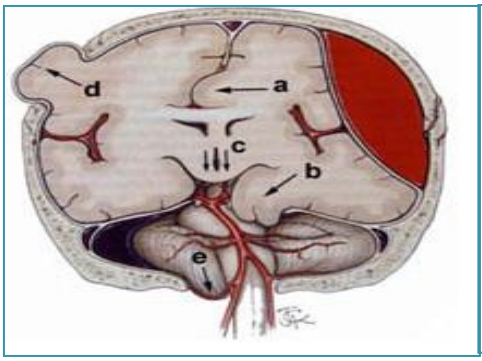
- Most of our body organs contain water. If you try to compress an organ that is full of water, this organ is going to shift. And this is what happens in case of raised ICP.
- When ICP increases, the brain goes under so much pressure that it will go to least resistance part in the brain and go out through it → brain herniations.
- If there was a severe brain compression, the brain is going to shift and go through an opening such as foramen magnum which will compress the respiratory center in the brain stem and cause a fatal problem.

Hemiplegia means total paralysis of the arm, leg, trunk of the same side of the body.

Tentorium is a membranous cover of cerebellum the process of the dura mater supporting the occipital lobes and covering the cerebellum

4.4.1 TYPES OF BRAIN HERNIATIONS

- **Uncal herniation**: It is the <u>most common</u> clinically seen type of brain hernias. Uncus is the most medial part of the temporal lobe. It's the part that is going to be herniated. If there's an increased ICP, the uncus goes above the tentorium and compresses the brainstem, causing dilated pupil "3rd cranial nerve affected", coma state and hemiaplagia.
- **Central herniation**: If there was a hematoma or mass that compresses the upper part, it will push the whole brain down through the tentorial opening.
- **Tonsillar herniation**: This type is fatal. If there was massive increase in the ICP especially that around the cerebellum, the tonsil will come down through the foramen magnum and will compress the lower medulla where the centre of respiration lays and the patient will stop breathing.
- **Cingulate herniation**: subfalcine herniation, it's when the left side of the brain is compressed and pushes the right side then goes under the falx cerebri to other side.
- **Outside herniation**: If there was a skull fracture and the pressure inside was so huge so the brain will look for the easiest way to be out.



A. Cingulate herniation
B. Uncal herniation
C. Central herniation
D. Outside herniation
E. Tonsillar herniation

4.5 INVESTIGATIONS

- If a patient came with headache and vomiting, check for Papilledema and do an urgent CT to the head.
- Lumbar Puncture is contraindicated until you do at least the CT (because if you take the CSF from the back and there was high pressure in the brain, it will

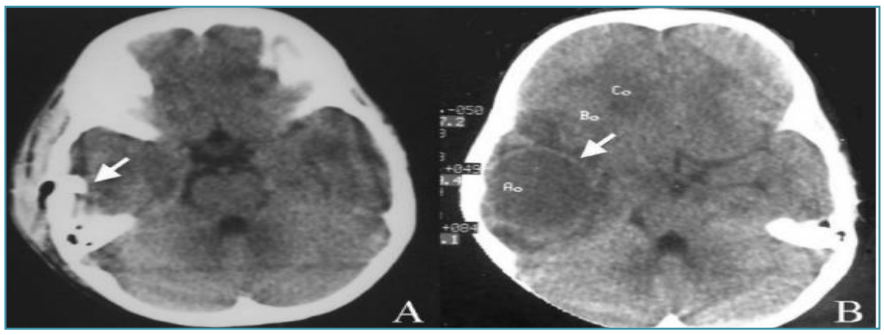
cause tonsil herniation which will kill the patient because he won't be able to breathe.)

- If also has fever, you start by checking for Papilledema and do a CT before doing Lumbar puncture to rule out meningitis.

4.6 TREATMENT

Mannitol is an osmotic diuretic, goes into the blood, makes the blood very light and will suck fluids

- General measures:
    - To increase the venous return, elevate the Head (30 degrees) to help with VR
    - No neck compression to relief veins
    - Give Mannitol for patients who have decreased LOC (or Furosemide) it will increase osmotic pressure in vessel & suck fluid from intracellular.
    - Steroids (Dexamethazone) only for tumors (a lot of edema around of tumor, it can be controlled by giving dexamethazone).
    - Hyperventilation: controlled to PCO2 35-40 mmHg a lot of hyperventilation → wash out CO2 → shrink blood vessels (decrease the amount of blood reaching brain) a CO2 is a very potent vasodilator so you want to decrease the amount CO2 so the blood vessels will go down and ICP will go down, a controlled hyperventilation.
    - Sedation, muscle relaxants decrease metabolic rate
    - Hypothermia decrease metabolic rate
    - Barbiturates: terminal option → you put the brain in complete relaxation.
- Specific treatment: Depends on the cause of raised ICP:
    - Vascular - SAH / ICH
    - Infection/Abscess:
      Rounded space
      In IV drug abusers or immune suppressed patients, with sinusitis.
      Sustained infection, that when you give contrast → enhanced picture (big collection of pus)

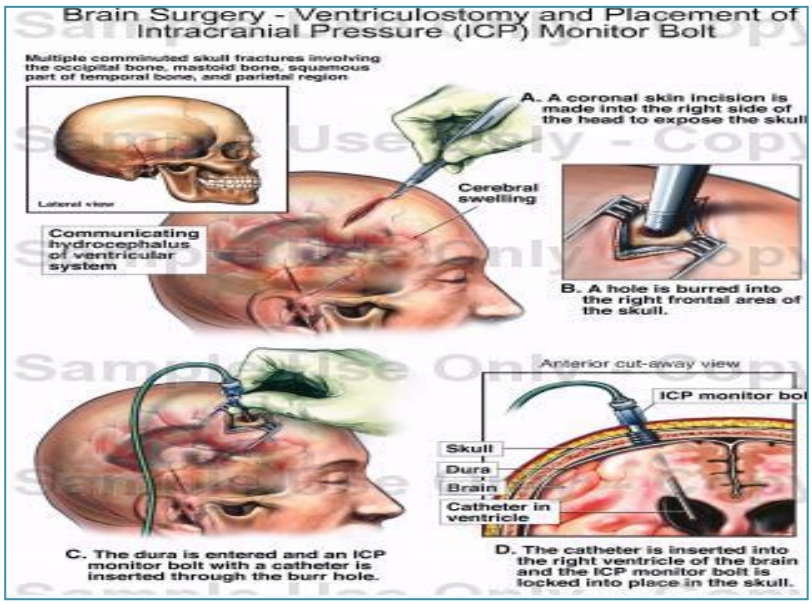


    - Trauma:

Localized: * Epidural Hematoma * Subdural Hematoma, compress brain, subfalcine herniation + temporal herniation.
Diffuse: Severe shaking of head → cut of Fx → Salt and pepper appearance of blood scattered around in brain

- Tumor: midline shift to other side, edema around it (Meningioma, Glioblastoma Multiformi). (you dissect the tumor)
- Hydrocephalus: Treated with shunt, ventricles enlarged, diffusion of CSF into brain substance.

- ICP can be monitored by inserting a catheter in the right ventricle of the brain substance to give pressure and suck fluid.



Case:

A 20 year old man presented to the ER unconscious with BP of 75/30, HR of 125 bpm, and with a right hemiplegia caused by a motor vehicle crash as unrestrained driver.

1. What are the differential diagnoses?
   a. Intracranial bleeding (he's unconscious, with right hemiplegia)
   b. Hematoma in the brain (that's why he is with hemiplegia and he is bleeding somewhere in the body and because of that he is hypotensive and unconscious and he has high HR)
2. How to deal with him in the emergency?
   a. 1st ABC:
      A. Check airway → endotracheal intubation (the first thing done for unconscious patient, because if his airway was blocked he will die within seconds)
      B. Breathing → chest tube (if tube was inserted and the patient's lungs were not inflated, he might have pneumothorax, and this will kill him in a minute)
      C. Circulation → stop the external bleeding but after giving I.V fluids. (Brain needs fluids so fluids must be replaced)
   b. 2nd insert 2 large I.V lines to start fluid, blood.
   c. 3rd C.T: to find out why he is unconscious.

5 MCQS

1. **The Glasgow coma scale (GCS) is dependent upon the following except:**
   A. Response to speech
   B. Response to pain
   C. <u>Response of the pupils</u>
   D. Best response
   E. Response of the patient

2. **Severe head injury is defined as Glasgow coma score of:**
   A. 3
   B. <u>3 - 9</u>
   C. 10
   D. 11-12

3. **Which of the following statements regarding the Glasgow coma scale is true?**
   A. It serves as a scale to assess the long-term sequelae of head trauma
   B. A high score correlates with a high mortality
   C. It includes measurement of intracranial pressure
   D. It includes measurement of papillary reflexes
   E. <u>It includes measurement of verbal response</u>
   Explanation:The Glasgow coma scale was developed to enable an initial assessment of the severity of head trauma. It is now also used to standardize serial neurologic examinations in the early postinjury period. It measures the level of consciousness using three parameters: verbal response (5 points), motor response (6 points), and eye opening (4 points). The score is the sum of the highest number achieved in each category. The fully oriented and alert patient will receive a maximum score of 15. A score of less than 5 is associated with a mortality of over 50%.

4. **An acute increase in intracranial pressure is characterized by which of the following clinical findings?**
   A. <u>Respiratory irregularities</u>
   B. Decreased blood pressure
   C. Tachycardia
   D. Papilledema
   E. Compression of the fifth cranial nerve.
   Explanation: The onset of irregular respirations, bradycardia, and finally increased blood pressure with increasing intracranial pressure (ICP) is termed the *Cushing response.* These physiologic alterations are caused by brainstem compression. Slow rises in ICP are, by contrast,

autoregulated by the brain's compensatory mechanisms and lead to a late onset of neurologic sequelae. A mass lesion is more apt to compromise local cerebral blood flow and increase cerebral edema and ICP. The vector of the mass effect may lead to herniation of brain parenchyma through the tentorial incisura or foramen magnum with resultant brainstem compression. Herniation usually causes compression of the third cranial nerve and thus leads to a fixed and dilated pupil on that side. Papilledema is a finding with chronic increases in ICP.

COMMON CONGENITAL NEUROSURGICAL DISEASES

1 HYDROCEPHALUS

1.1 DEFINITION

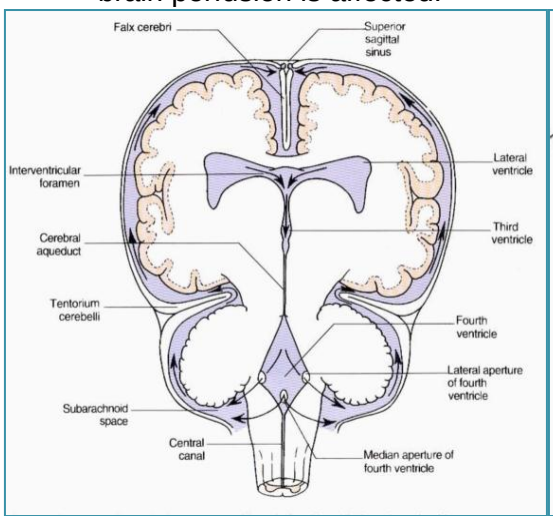
- Hydro; water and cephalus; head
- Hydrocephalus is an increase in the CSF, associated with increased ICP.
- Ventriculomegaly secondary to cortical atrophy; aka hydrocephalus exvacuo

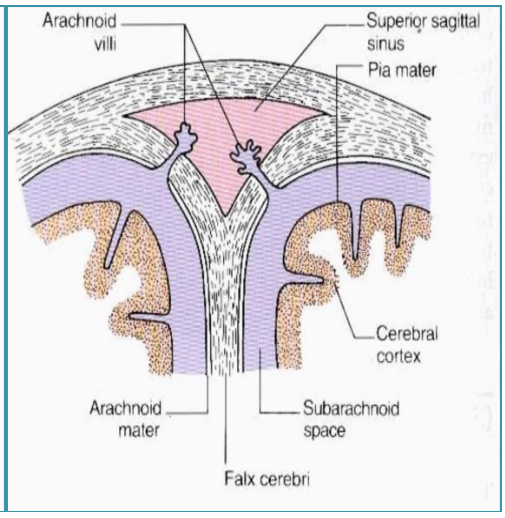
1.2 CAUSES

- Overproduction of CSF *(Tumor of the choroid plexus)*
- Obstruction of CSF flow
- Under absorption of CSF into the blood stream *(leads to accumulation of CSF) (When the way to the arachnid granulations is obstructed → Examples: Post meningitic lesions and post hemorrhagic lesions.*

1.3 PHYSIOLOGY

- Total volume of CSF in the ventricles varies from 5-15 ml in neonates to 150 ml in adults. *(Depends on age and weight)*
- **Produced by choroid plexus and the extracellular fluid of the brain.**
- Rate of production is 0.3-0.4 ml/minute.
- Only very high ICP will reduce CSF production; usually at the point when brain perfusion is affected.





Hydrocephalus

- It is an accumulation of CSF within the cerebral ventricle and is usually associated with altered ICP
- The pressure is usually high, and sometimes normal, but rarely low (negative pressure hydrocephalus)
- When the ventricles are large but the patient is asymptomatic, that is not hydrocephalus; it's

just hydrocephalus ex vacuo "old name" or ventriculomegaly. So when you see large ventricles, it does not indicate hydrocephalus UNLESS there are symptoms of pressure changes of the brain.

CSF is produced by the choroid plexus of the ventricles

A tumor of the plexus can increase CSF production

Everyday the plexus produces 500ml of CSF

Figure 1: Coronal view of the brain
1. Both lateral ventricles will drain CSF into 3rd ventricle through the foramen of Monro **IMP**
2. From 3rd ventricle the CSF will pass to the 4th ventricle in the posterior fossa through the aqueduct of Sylvius **IMP**
3. From 4th ventricle CSF circulates around the brain then passes through the three apertures (median foramen of Magendie & 2 lateral foramina of Luschka) to circulate around the brain, spinal cord and in central canal of spinal cord.
Figure 2: This small cut section at the level of superior sagittal sinus (which is one of the dural venous sinuses of brain) shows the arachnoid granulations, which are an extension of the arachnoid. CSF drains into the arachnoid granulations (villi), then through the core of the villi into the venous circulation.
So CSF circulates within ventricles then around the brain then it is reabsorbed in the cerebral ventricles.

Rate of CSF production is 0.3-0.4 ml/minute → 500 ml CSF/day – 150 ml in the CNS → 350 CSF absorbed every day.
- It is a process of active formation and it does not stop it
- It may be affected by ↑ ICP BUT it does not stop it
- If there is any problem affecting absorption or the pathway of CSF → accumulation of CSF and ↑ ICP (which decreases CSF production)

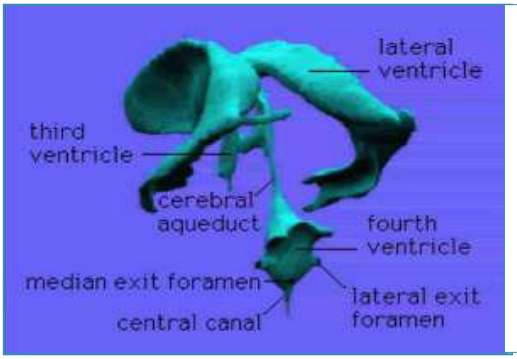


Figure 3:
Shows the communication between the ventricles
2 lateral ventricles inside cerebral hemisphere - they have a frontal horn, occipital horn & temporal horn

Both communicate through foramen of Monro> 3rd ventricle> cerebral aqueduct> 4th ventricle

1.4 EPIDEMIOLOGY

- The overall incidence of infantile hydrocephalus is 1/1000 live birth and 2/1000 in some countries like Africa and India ** It is not a common disease*

1.5 TYPES OF HYDROCEPHALUS

1.5.1 COMMUNICATING:

- All ventricles are dilated
- Overproduction or under absorption of CSF
- No obstruction in the pathway of CSF within the ventricles *(the ventricles can communicate with each other)*

1.5.2 NON-COMMUNICATING:

- Partial dilatation
- Blockage of the flow of CSF
- Obstruction within ventricles or the pathway of CSF *(obstruction to the CSF flow at the foramen of Monro, the third ventricle, the aqueduct of Sylvius, the fourth ventricle, or the foramina of Magendie or Luschka.)*
- Congenital, since birth
- Acquired, develops after birth as a result of injury, tumors or meningitis.

1.6 ETIOLOGY

1.6.1 CONGENITAL (PRIMARY)

- Aqueductal anomalies *Most common cause of congenital hydrocephalus*
- Dandy Walker malformation *associated with meningomyelocele*
  - *Atreisa of the foramen of Magendie or Luschka*
- Chiari II malformation
- Myleomeningocele
- Intrauterine viral infection *(CMV, mumps, rubella, varicella)*
- Toxoplasmosis
- Congenital tumors
- Vein of Galen aneurysms
- Chromosomal anomalies *(Trisomy 13 and 18)*
- Congenital or primary hydrocephalus*. (idiopathic; no known reason)*

Important 3 causes of communicating hydrocephalus:
- Post-hemorrhagic
- Post-meningitic
- Post-traumatic

3 posts for acquired causes!

Remember that the cause of acquired hydrocephalus is usually outside the ventricles.

1.6.1.1 AQUEDUCTAL ANOMALIES

- Aqueduct is the passage of CSF between the 3rd & 4th ventricles-passes in the midbrain
- It is still know as aqueductal stenosis, however by definition, the correct term is
- “aqueductal atresia“ because no aqueduct is found in most of cases - but in some cases
- (who develop hydrocephalus late in their childhood) it allows a small amount of CSF to
- pass through so it is usually called stenosis

1.6.1.2 DANDY WALKER MALFORMATION (LARGE DANDY WALKER CYST)

- By definition it is congenital hypoplasia or even aplasia of cerebellum associated with formation of a large CSF cavity within the posterior fossa due to the obstruction of CSF flow by a large cyst (which doesn’t allow CSF to pass from the 4th ventricle and circulate around the brain)
- There are different types of Dandy Walker cyst according to the volume of cerebellum that’s involved
- Most of cases of Dandy Walker malformation are associated with hydrocephalus
- In short: large CSF cyst on posterior fossa due to agenesis of the cerebellum that communicates with the 4th ventricle, and causes hydrocephalus.

Dandy-Walker malformation is characterized by agenesis or hypoplasia of the cerebellar vermis, cystic dilatation of the fourth ventricle, and enlargement of the posterior fossa

Presentation: Incoordination, ataxia, nystagmus

1.6.1.3 VEIN OF GALEN ANEURYSMS

- A large vascular malformation where there is a direct communication between the arterial system and venous system (shunting), leading to dilatation of the Vein of Galen (one of the deep venous structures in the brain) and to obstructive hydrocephalus.
- What is the clinical manifestation for such cases in neonates?
  - 1st and most important is heart failure (the size of the arteriovenous shunt that can steal 80% or more of the cardiac output), then

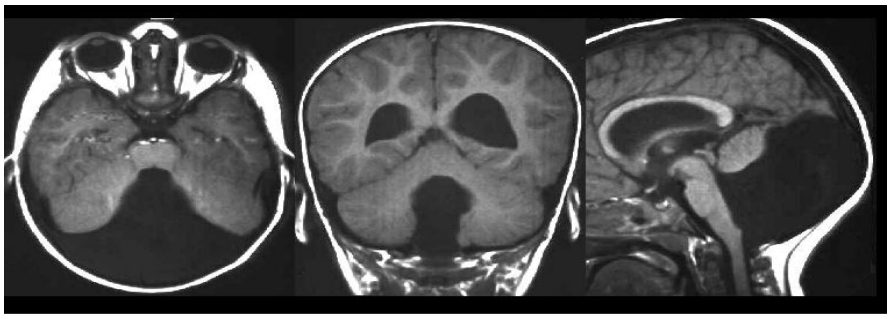
symptoms of hydrocephalus (developmental delay, seizures, headaches)

- Rx: treat the cause, which is the aneurysm (stop passage of blood from artery to vein by embolization) - and no need for shunt to treat the hydrocephalus
  - Also shunts can burst the aneurysms and cause fatal hemorrhage

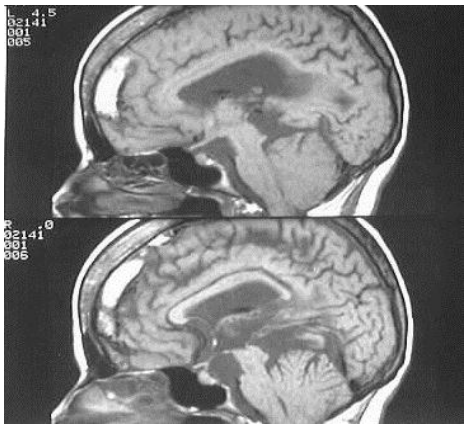
1.6.2 ACQUIRED (SECONDARY)

- Most cases
- Germinal plate hemorrhage: *"immature blood vessel walls"*
  - Leads to intracranial hemorrhage in premature babies <1500 gm (30%-40%)
- Subarachnoid hemorrhage:
  - *Due to trauma*
- Post-meningitis:
  - 1% of survivors of bacterial meningitis
  - More in neonates
  - Especially G-ve organisms (i.e. E. coli).
  - Rare but important, postnatal cysticercosis.
- Tumors. *(Most common cause)*
- SAH. (Subaeachnoid Hemorrhage)
- Severe TBI (Traumatic Brain Injury)

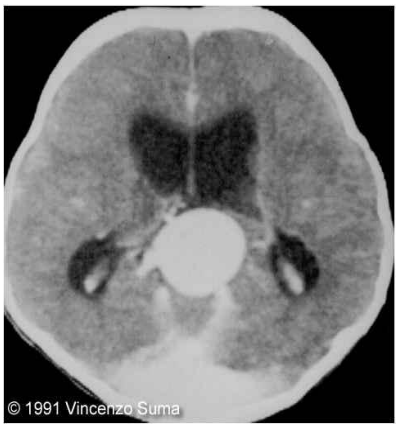
Normal pressure hydrocephalus (NPH)
-Usually in elderly,
-Presents with dementia-like symptoms
-Patient's family complain of patient's memory loss
-It is treatable
-Investigate with CT and MRI → you'll see ventricular dilatation more than cortical atrophy



Dandy Walker malformation



Congenital or primary hydrocephalus



Vein of Galen aneurysms

1.7 CLINICAL FEATURES

1.7.1 INFANTS & YOUNG CHILDREN:

- Increasing head circumference. *(Scalp bones are still soft. So the head circumference increases abnormally – not according to curve of growth)*
- Irritability, lethargy, poor feeding, and vomiting. *(Leads to delayed development)*
- Bulging anterior fontanelle. *(Wide, full and tense)*
- Widened cranial sutures.
- McEwen's cracked pot sign with cranial percussion. ⓘ *(sounds like you're tapping on a cracked pot)*
- Scalp vein dilation (collateral venous drainage).
- Sunset sign (downward deviation of the eyes). ⓘ
  - *not only infants but also in children and sometimes adults*
- Epidsodic bradycardia and apnea. *(If hydrocephalus is left untreated – the increased intracranial pressure will press on the brain stem –where the respiratory centers are located- which will lead to this)*
- *Irritability, drowsiness, vomiting*

1.7.2 JUVENILE & ADULT:

- Headaches *(Most common symptom) (Usually in the early morning. Why? Because during sleep, the patient will hypo ventilate, which will lead to ↑ CO2 → ↑ vasodilation → blood stasis & ↑ intracranial pressure)*
- Projectile Vomiting. *(The symptoms are usually relived after vomiting. Why? Because vomiting leads to hyperventilation which will reverse the CO2 retention and cause vasoconstriction of the blood vessels → ↓ Intracranial pressure)*
- Seizures. *(Acute manifestation)*
- Decreased level of consciousness.
- Focal neurological deficit. *(Depending on the cause of the hydrocephalus)*
- Collection of CSF around previous shunt site
- Progressive visual disturbances
- Papilledema *(When the hydrocephalus has been there for days, not in the acute onset)*

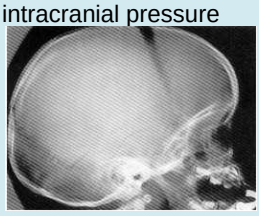
1.8 INVESTIGATIONS

- CT scan: *easy, available and you can do it in minutes (Method of choice)*
  - The pattern of ventricular enlargement can help delineate the cause:
  - *Allows you to see any lesions*
  - *Suitable for acute hydrocephalus*
    - Lateral & 3rd ventricle dilatation
      - normal 4th ventricle: suggests aqueduct stenosis
      - deviated or absent 4th ventricle: suggests posterior fossa tumor *"obstructive hydrocephalus"*
    - Generalized dilatation: suggests a communicating hydrocephalus.

Night (sleep) → hypoventilation→ increased Co2 retention → vasodilatation → symptoms are worse
Vomiting (on waking up) → hyperventilation→ washing out of Co2 → vasoconstriction and less dilations → symptoms improve

X-ray is not a diagnostic procedure for hydrocephalus now

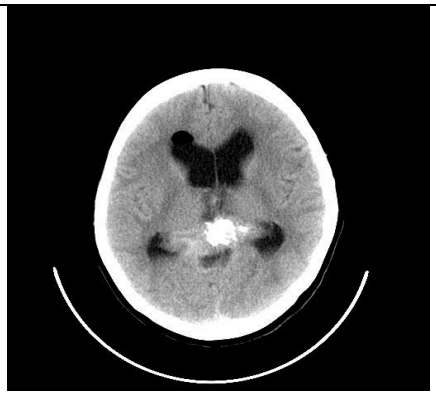
You will see widening of the suture of skull secondary to increased intracranial pressure



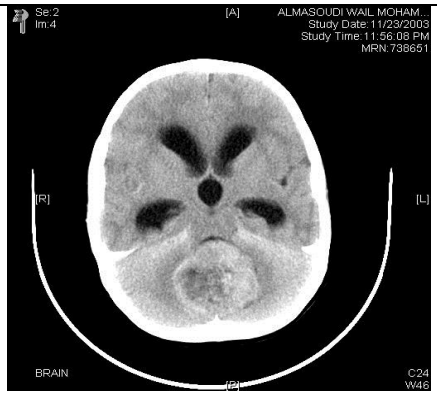
ⓘ **Classical Radiological Sign:**
-Separation of sutures
-Pressure of the gyri on the bone

- MRI:
  - Shows more anatomical details
  - Allows better visualization → Example: Aqueductal stenosis
  - Not available in the ER, takes longer time than a CT

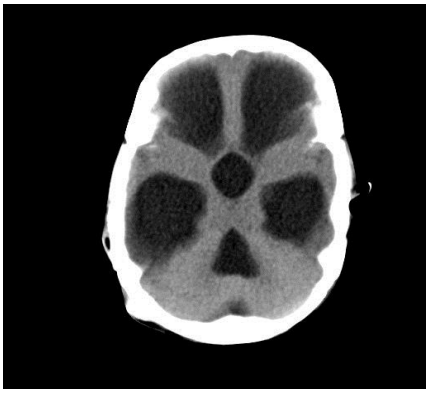
CT Scan



Lateral ventricles dilated but 3rd ventricle not very dilated > lesion in 3rd ventricle that prevents CSF from entering the 3rd ventricle at level of foramen of **Monro**



Posterior fossa tumor



Communicating hydrocephalus

1.9 TREATMENT

1.9.1 SURGICAL TREATMENT

- Endoscopic choroid plexectomy was tried but found unsuccessful. *Not done now because all children die during the operation*
- Replaced now by Choroid plexus coagulation *We use endoscope, introduce telescope in the ventricle, and it helps in reduction of CSF formation*

There's no medical treatment, you can use some medication to reduce the volume of CSF and reduce

ICP, but CSF is actively produced by the choroid plexus so it's difficult to get rid of the problem

1.9.2 INTRACRANIAL SHUNTS

- In obstructive hydrocephalus where the subarachnoid spaces are still patent

1.9.2.1 ENDOSCOPIC THIRD VENTRICULOSTOMY

- The endoscope is passed through a burr hole to the third ventricle where the floor is fenestrated just anterior to mamillary bodies. *(So we bypass the obstruction in the aqueduct or posterior fossa).*The hole is enlarged by introducing the endoscope or an inflatable balloon.

ⓘ Endoscopic third ventriculostomy: **1st line treatment** now

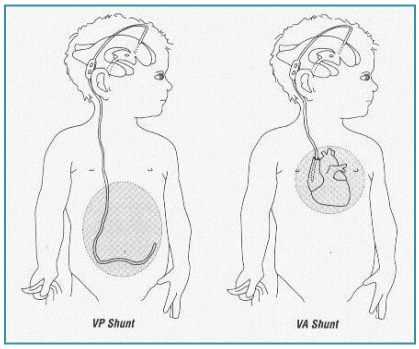
1.9.2.2 VENTRICULOCISTERNOSTOMY:

- Shunt between lateral ventricle and the cisterna magna.
- It has high morbidity and mortality*. (Not done any more)*

1.9.3 EXTRACRANIAL SHUNT:

Extra-cranial Shunt: if you have communicating hydrocephalus or if you don't have the facility or capability to do endoscopic third ventriculostomy

- From the ventricular system, usually the lateral into another body cavity; the peritoneal cavity (VP Shunt), right atrium (VA Shunt) and occasionally pleural cavity.
- Aim is to normalize the intracranial pressure
- Specially designed shunt valve with the appropriate rate of flow and pressure. Regulate the CSF flow in a unidirectional way.



- Shunts are made of silicon which is well tolerated by the body. It causes minimal or no tissue reaction or intravascular thrombosis

1.9.4 COMPLICATIONS OF VP SHUNTS

1.9.4.1 MECHANICAL FAILURE

- Under drainage or over drainage
- Blockage (Obstruction) *(Most common complication)*
- Improper placement
- Migration of the shunt system
- 25 to 40% in the first few months after surgery, later on 4 to 5 %.

1.9.4.2 SHUNT BLOCKAGE (OBSTRUCTION)

- 50% of mechanical failure, highest in the immediate post operative period.
- Proximal occlusion (In brain)
  - By brain debris or choroid plexus
  - Ideally the catheter should lie away from the choroid plexus
- Shunt valve blockage
  - By brain debris or blood clots or due to failure of the valve system
- Distal obstruction (In abdomen)
  - Less frequently than the proximal obstruction
  - Accumulation of particles
  - Catheter encysted or isolated in one area of the peritoneal cavity *Following infection or adhesion*
- Catheter migration, disconnection , or fracture *Sometimes catheter pass through stool or coming out through the anus*

1.9.4.3 SHUNT INFECTION

- Most dreadful complication

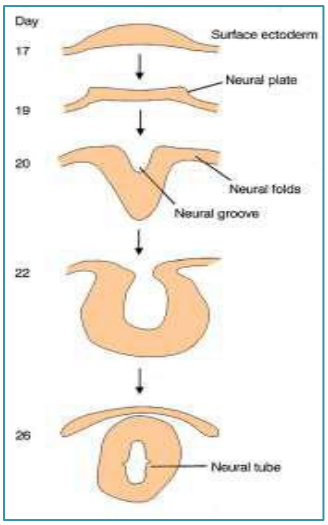
- About 5%, and may result in further risk of intellectual impairment.
- Organisms
  - Staphylococcus epidermidis about 40% *Commonest cause. It's normally found in the skin and it easily reaches the shunt, causing CSF infection "meaning meningitis leading to disability "*
  - Staph aureus about 20% by.
  - Streptococci and gram negative organisms are less frequent.
- Clinical features *infection leads to increase CSF volume so patient suffer from hydrocephalus symptoms again*
  - Early, within 8-10 weeks.
  - Fever, malaise, headache & irritability, neck stiffness.
  - Peritonitis is less common.
  - Patients with Staph epidermis may look remarkably well with only intermittent fever or irritability.
  - Diagnosed by blood culture, routine blood examination and CSF examination. *(CSF sample should be taken from the shunt)*
- Treatment of shunt infection:
  - Removal of shunt and the external ventricular drainage plus antibiotics.
    - *Take the shunt out → put temporally external drainage → treat the infection by antibiotics till the CSF cleared of infection → then replace the external drainage by internal shunt*
    - *External drainage very useful b/c we can drain the CSF, take sample of CSF for culture and to inject antibiotics intra ventricular to treat infection or speed up the clearance of infection*
- Treatment with antibiotics alone. For patients who have very low grade of infection & Clear CSF
  - Antibiotic prophylaxis is controversial however it was found that intra-operative antibiotics or antibiotics for the first 24 hours give the best results.

If you press valve and there is no fluid passing through shunt > distal obstruction

If you press shunt & valve collapse and doesn't refill again > proximal obstruction

1.9.5 LESS COMMON COMPLICATIONS:

- Subdural collection from over drainage
- Slit ventricle syndrome, (over drainage) *> cause collapse of ventricles*
- Disconnection or fracture of shunt tubing
- Seizures



2 DEVELOPMENTAL SEQUENCE

2.1 DEVELOPMENTAL SEQUENCE

- Neural plate invaginates as neural folds push up
- Neural folds eventually form neural groove
- Cells of neural fold eventually meet
- Form the neural tube
- Neural tube runs anterior – posterior along embryo
- Surrounding ectoderm eventually encloses neural tube

Types:
Open → occurs when the brain and/or spinal cord are exposed at birth through a defect in the skull or vertebrae.

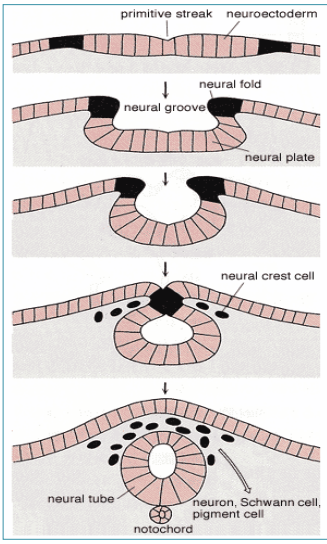
Closed → occurs when the spinal defect is covered by skin.

- When neural tube closes off brain and spinal cord are formed and this process is completed on day 28 of pregnancy begin > *so if the spina bifida formed at 28 day it's formed!*
- Spinal cord covered posteriorly by spinal process and laminae

3 NEURAL TUBE DEFECT

3.1.1 SPINAL DYSRAPHISM

- Spina Bifida
- Failure of closure of posterior neural arch
- Open or Closed
- 80% in lumbosacral region *(Occasionally in the head)*



CNS:

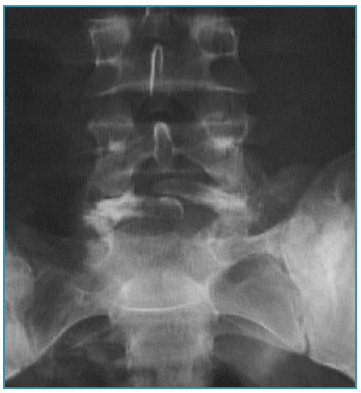
- Neuroectoderm in origin > neural plate > neural groove > neural fold > more folding > ends try to proximate and touch each other > forming neural tube > migrate rostral forming brain & caudally > spinal cord
- Then it'll be covered by mesoderm > forms the bones "bodies vertebra" & lamina & spinal process (posteriorly) > failure of that is called spina bifida
- Myelomeningocele: herniation of S.C > if S.C outside the canal & will affect the lower limb of baby b/c it's not functioning
- Meningocele: only contains CSF covered by meninges **Commonly in lumber area**

3.2 TYPES OF MYELODYSPLASIA

- Spina bifida occulta
- Lipomeningocele *"lump of fat in spinal canal"*
- Meningocele
- Myelomeningocele = Spina Bifida

3.2.1 SPINA BIFIDA OCCULTA

- Found incidentally *when the patient does x-ray for any reasons.*
- 5-10% of population *so it's common.*
- Not clinically significant *"asymptomatic"*
- Tuft of hair, dimple sinus or port wine stain
- High incidence of underlying defect
- No treatment required, U/S or MR



Spina Bifida Occulta: The two laminae try to reach each other but are not touching each other and do not form

If there's associated coetaneous lesions that alert us to a bigger problem
Hair tuft, Dimples. Sinus, Pigmintation, Lypoma, Focal abnormalities
→Example: Intraspinal tumors

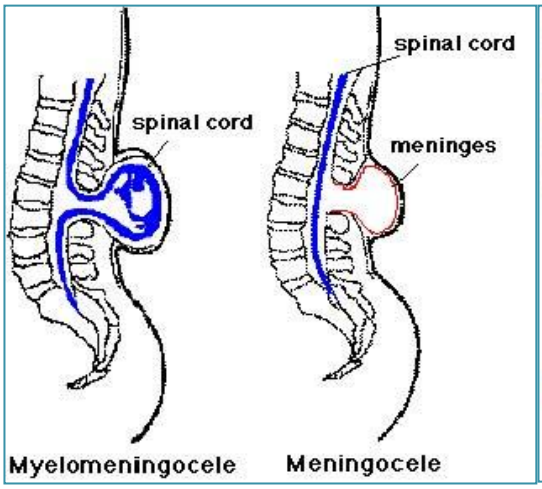
One of the causes or recurrent meningitis in children (You'll find signs of an underlying problem like a tuft of hair or dimpling)

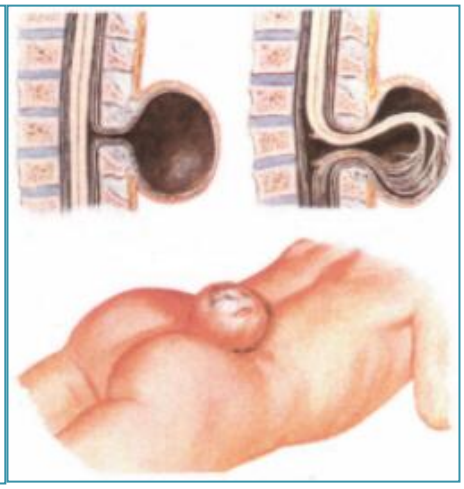
Split cord syndrome
Early life →
Asymptomatic →As they get older they might have sphincter control problems, weakness in the lower limbs

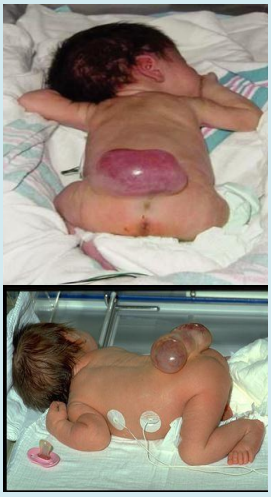
3.2.2 MENINGOCELE

(Sac filled with CSF but no spinal cord)

- Cystic CSF-filled cavity lined by meninges →*so no neural abnormalities "motor or sensory deficit in lower limbs" but some have some autonomic deficits like sphincters; Incontinent or nocturnal enuresis.*
- No neural tissue
- Communicates with spinal canal
- Look for other cong. Anomalies
- Seldom any neurological deficit
- Usually discovered incidentally
- Dx: U/S or MRI
- **Urgent** excision if CSF leak (If ruptures), otherwise deferred
- Perhaps indefinitely if small







Examination

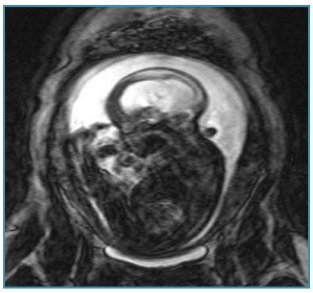
-Locally → Size, content
-Assess skin quality (for surgery)
-If there's CSF leak → it is a surgical emergency → Risk of Infection
-Translumination test to know the content
-Neurological examination
-Examine the sphincter to see if there's any problem with the anus (incontinence)
-Check for other associated anomalies (E.g.: Kyphosis), size of the head.
-Get the family collaborations from other specialties depending on the child's problem

3.2.3 MENINGOMYELOCELE

- *Most common*
- *Risk increases with each pregnancy 5%*
- *Female predominance*
- *High association with other anomalies*
- Spinal cord and roots protrude through the bony defect, lie within cystic cavity, if ruptured, CSF will leak trans-illumination *(emergency case).*
- *Because the S.C outside >> the pt will have autonomic, sensory and motor deficit in the lower limbs and they are born with paraplegia.*
- Observe limb movements (degree & level of neurological damage)
- Note dilated bladder & patulous annual sphincter
- Dx: U/S or MRI *(MRI is more accurate)*
- If ruptured; immediate closure & replacement of neural tissues into spinal canal
- Gross hydrocephalus, multiple serious cong. anomalies; many adopt thoughtful conservative treatment
- Look for other cong. Anomalies : gross hydrocephalus , Chiari malformation and other multiple serious cong. Anomalies
- Many adopt thoughtful conservative treatment.

- *Ruptured meningiocele/ meningiomyocele is a surgical ER*

3.3 ANTENATAL DIAGNOSIS



- Maternal U/S,
- MRI
- Serum/amniotic fluid for alpha-fetoprotein & acetylcholinesterase
- Contrast enhancing amniography
- Possibility of therapeutic abortion

3.4 PREVENTION

- Give folic acid supply during pregnancy

3.5 TREATMENT

3.5.1 SHORT TERM TREATMENT

- If the defect has normal skin covering>>> Treatment in elective basis (no needs to emergency surgery)
- Thin or no skin covering at all, ruptured sac >>>early surgery as possible to prevent meningitis.

3.5.2 LONG TERM TREATMENT

- Early treatment of hydrocephalus
- Regular follow up in spina bifida clinic
- Urological, orthopedic, paediatrics, and physical therapy
- Uological; urinary incontinence, vesicoureteric reflux, repeated UTI, renal impairment, hypertension and stunted growth
- Orthopedic; feet deformity, and tendon transfer, pelvic and spine deformities
- Neurosurgical; tethered cord, chiari II malformation, shunted hydrocephalus
- *You have to see them at least once or twice a year*

3.6 ANENCEPHALY

(No brain)

- Defective closure of the rostral neural tube
- results in anencephaly or encephalocele
- Neonates with anencephaly have a rudimentary brainstem , or midrain , no cortex or cranium
- Rapidly fatal condition if born alive
-

3.7 CLINICAL MANIFESTATIONS

According to the level of spinal bifida (Meningomyelocele)

- Lower Lumbar (L5,S1) → Distal weakness in the feet and sphincters incontinent
- Upper lumbar (L1) → Complete paraplegia of the lower limbs.

Myeloschisis or rachischisis**:** where there is defect in the bone and the spinal cord exposed outside no skin covering > type of Meningomyelocele Sever neurological deficit in the lower limbs.

3.8 INCIDENCE

- 2/1000 birth .*2-.3/1000 in Scandinavia b/c they put folic acid in flour and they do therapeutic abortion once baby diagnosed with spina bifida.*
- Risk increase to 5% if a sibling is affected
- Teratogens; Sodium Valporate
- Associated with Hydrocephalus, Chiari II and aqueduct forking

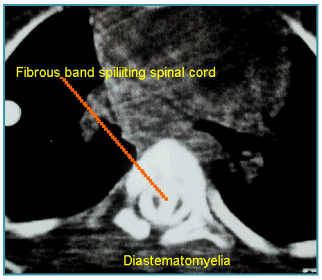
3.9 CAUSES

- The main cause is Folic acid deficiency; *since the development of the nervous system end in day of 28 of pregnancy. The woman should take folic acid when they plan to get pregnant.*
- Teratogens; Sodium Valporate *(antiepileptic drug , if you start giving this medication, once she becomes pregnant she may develop baby with spina bifida > so you need to change it before pregnancy)*

3.10 OTHER CONGENITAL ANOMALIES

- Tethered spinal cord
- Diastematomyelia
- Lipomeningocele *Definition: fat in the meningocele.*
- Congenital dermal sinus: *The baby is born with back dermal sinus with tuft of hair, check for spina bifida in this baby.*

3.10.1 DIASTEMAMYELIA



- A bone or fibrous band divides spinal cord in two longitudinal sections ⓘ
- Associated lipoma may be present, which tethers cord to vertebra
- Signs &Symptoms include weakness, numbness in feet, urinary incontinence, decreased or absent reflexes in feet
- **Dx: CT**
- Rx - surgery to free cord

3.10.2 ENCEPHALOCELE

Large cyst in the skull contains CSF or brain

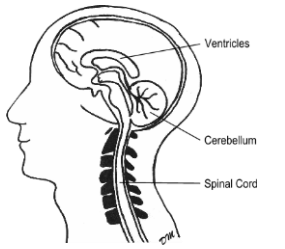
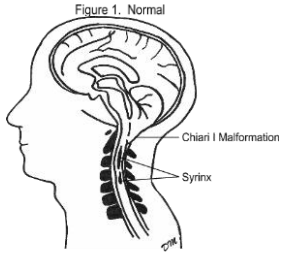
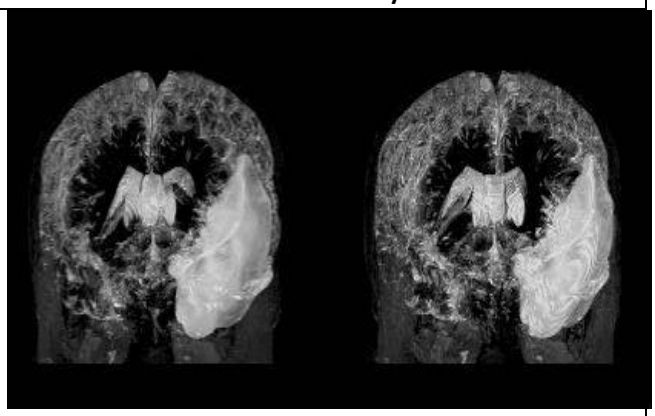
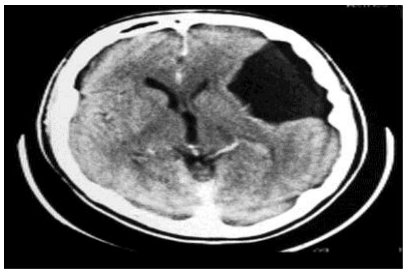
- Usually occipital
- May contain occipital lobe, or cerebellum
- Often associated with hydrocephalus
- Immediate treatment if ruptured
- Outcome depends upon contents

3.10.3 CHIARI I MALFORMATION

- Cerebellum herniation *through foramen magnum >> crowding of the area >> affect the*
- *CSF circulation >> dilation of the CSF within the S.C; this is called syringomyelia*
- *Two types:*
  - Type A → no spinal bifida (Meningomyelocele)
  - Type B → with spinal bifida

3.10.4 CLINICAL MANIFESTATION OF CHIARI MALFORMATION

- Babies have clinical manifestation called foramen magnum syndrome, which include symptoms like:
  - Pressure of the S.C in that area.
  - Stretching of lower cranial nerves, associated syringomyelia
  - Leads to neck pain, Headache, upper limbs symptoms, weakness of hands, loss of fine touch,loss in temperature sensation in the tip of the fingers ,upper motor neurons manifestations of the lower limb.

Chiari I malformation	Arachnoid cyst
 Figure 1. Normal  Figure 2. Chiari I Malformation Downward herniation of cerebellar tonsil through foramen magnum into cervical spinal canal	 

Encephalocele

Emergency if it ruptures

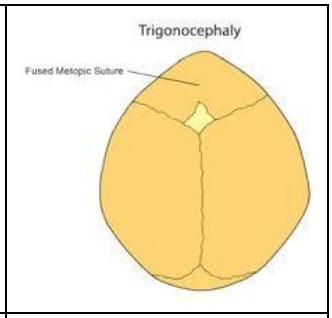
3.10.5 ARACHNOID CYSTS

- Developmental anomaly → Intra-arachnoid cavity filled with CSF → Benign
- Treatable → Good prognosis
- Majority in the sylvian fissure
- Space occupying lesion symptoms → Convulsions, raised ICP
- In supracellar space → Can produce endocrine dysfunction
- Diagnosis
  - CT scan (Not the best) Can be confused with a tumor
  - MRI is more precise
- Treatment → Shunting

3.10.6 CRANIAL SYNOSTOSIS

- Early fusion of sutures → Scull deformity
- Coronal → Normally allows the head to enlarge in the anterior-posterior direction → Early closure leads to Brachycephaly
- Sagittal → Normally allows the head to enlarge in the lateral direction →Early closure leads to Scaphocephaly
- Metopic → Early closure leads to Trigonocephaly (Triangular shape to the head)
- Cosmetic problems → Must be fixed
- Global → All sutures involved → Must be fixed because the brain doesn't have room to grow

Brachycephaly | Scaphonocephaly



Trigonocephaly

4 MCQS

1. **Which statement is true:**
   a. Spina bifida occulta is a neurosurgical emergency.
   b. Meningocele contains spinal cord.
   c. Spina dysraphism occurs most commonly in lumbosacral region.
   d. None of the above
2. **In arachnoid cyst, what is false?**
   a. Occurs commonly in the temporal area.
   b. All cases should be treated urgently to avoid complications
   c. May present with seizures
   d. May be asymptomatic

3. **The most common type of cerebral herniation is:**
    a. Central
    b. Cingulate
    c. Transtentorial
    d. Tonsillar
4. **The investigation of choice in increased ICP is:**
    a. Skull x-ray
    b. MRI
    c. Lumbar puncture
    d. CT scan
5. **Hydrocephalus is defined as:**
    a. Accumulation of CSF with ICP
    b. Soft tissue contusion
    c. Intracranial hemorrhage
    d. Enlargement of the head
6. **Obstructive hydrocephalus is best treated by:**
    a. Surgery
    b. Drainage
    c. Craniotomy
    d. Endoscopic third ventriculostomy
7. **In Spina Bifida, the following is correct except:**
    a. It is a failure of closure of posterior neural arch
    b. Contains spinal cord in menengeocele
    c. There are two types, Open and Closed
    d. 80% in lumbosacral region

🗝 **Answers: 1:C, 2:B, 3:C, 4:D, 5:A, 6:D, 7:B**

COMMON NECK SWELLINGS

1 PARATHYROID GLAND

- General characteristics
  - We have four parathyroid glands in the posterior aspect of the thy thyroid gland
  - Both the superior and the inferior parathyroid glands receive blood supply from the inferior thyroid artery
- Embryology of The parathyroid glands:
  - The upper parathyroid glands originate from the 4th pharyngeal pouch
  - The lower parathyroid glands originate from the 3rd pharyngeal pouch
- Physiology of the Parathyroid:
  - Ca2+ homeostasis: release of Parathormone/Parathyroid hormone (PTH) to raise Ca2+ levels in the blood
  - Vitamin D regulation: PTH induces Vit.D hydroxylation in the kidney, and this process is necessary for Vit.D activation.
  - Calcitonin: is released from the c-cells of the thyroid gland decrease Ca2+ levels. These are not of physiological significance.

1.1 HYPERPARATHYROIDISM:

- Definition: Is an increase secretion of PTH from the parathyroid glands that leads to increase serum calcium, decreased serum phosphate. Hyperparathyroidism can be either primary or secondary.

1.1.1 PRIMARY AND SECONDARY HYPERPARATHYROIDISM

- Primary is more common and is due to increase secretion from the any of the glands due to hyperplasia, adenoma or carcinoma.
- Secondary hyperparathyroidism is due disordered metabolism (chronic kidney disease or Vit.D metabolism disorders) that causes hypocalcaemia for prolonged times and secondary enlargement of the parathyroid glands.
- Serum levels of PTH are increased along with Ca2+ (because PTH increases Ca2+ levels)
- Hyperparathyroidism is the most common cause of hypercalcaemia in society ⓘ
- The most common cause of hypercalcaemia in the hospital is malignancy ⓘ

1.1.2 EPIDEMIOLOGY:

- Statistics from Western countries indicate a 0.1-0.5% prevalence rate for PHP.
- No evidence for geographical variation
- 1200- 6000 cases were expected in Aseer area alone, but when Prof.Shehri investigated the prevalence of cases, they only found 30!
- Uncommon in children
- 2-3 times in females

1.1.3 CAUSES:

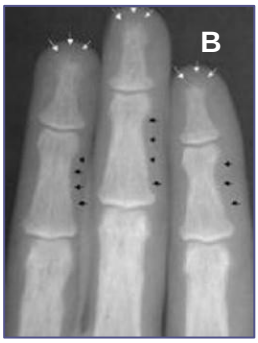
1. **Adenoma**: most common cause of 1ry hyperparathyroidism is: (84% of cases)
    a. Usually NONE is palpable
    b. Affects one gland ⓘ
2. **Hyperplasia**: 15% of cases
    a. Usually NONE palpable
    b. Usually affects all four glands ⓘ
3. **Carcinoma**: 1% of cases
    a. Presents with palpable swelling (unlike adenoma and hyperplasia) ⓘ

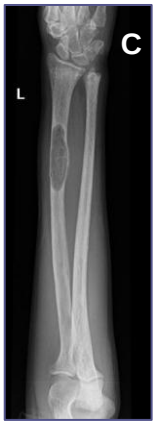
If a patient presents with hypercalcemia, elevated serum PTH a neck lump, it is either one of two things: carcinoma of the parathyroid gland

1.1.4 SIGNS AND SYMPTOMS:

Signs and symptoms are related to increased serum Ca2+ which affects multiple organs and systems:

1. **Bone**: high levels of PTH activate bone resorption and cause bone matrix depletion. Bone involvement on x-rays can be seen as:
    a. Osteopenia is most common sign of hyperparathyroidism, can be generalized or local. This bone loss that may lead to fractures, bone & joint pain.
    b. Subperiosteal erosion (picture): is an early and virtually pathognomonic sign of hyperparathyroidism.Most commonly in the middle phalanges of the index and middle fingers, primarily on the radial aspect
    c. Brown tumor (picture): which is only a radiological description and not an actual tumor.
    d. Cyst formation
2. **Kidney**: stones and glomerular calcification
3. **Abdomen**: abdominal pains where some patients may develop peptic ulcer disease, pancreatitis.
4. **Neuropsychiatric** symptoms: depression, mood changes
5. **General** symptoms: Fatigue
6. The symptoms range from: No symptoms → mild, general symptoms like fatigue and depression→ renal symptoms → bone symptoms





Mnemonic for symptoms: “Painful bones, renal stones, abdominal groans, and psychic moans”

A brown tumor is a rare manifestation of hyperparathyroidism. Reparative granulation tissue and active, vascular, proliferating fibrous tissue may replace the normal marrow contents, resulting in a brown tumor. Hemosiderin imparts the brown color (hence the name of the lesions)

1.1.5 PRESENTATION:

- In the west:
    - 60 - 70% detected by routine screening.
    - Many are asymptomatic
- In KSA:
    - Age 30 – 77 (median 40)
    - Majority are Females
    - Almost all patients in KSA present with bone symptoms
    - 54% have also renal manifestations.

1.1.6 CASES FROM THE DOCTOR:

Case 1:

40 year old lady that presented with left humerus fracture, past medical history is significant of bilateral ureteric stones that have been removed and a non-functional left kidney. Serum Ca2+ was 11.2 mg/dl and PO4 2.2mg/dl. Bone symptoms, kidney symptoms (failure and colic), high calcium and low phosphorus.

Case 2:

30 year old lady that presented with long history of generalized bone ache, heart burn,easy fatigability and right humeral fracture, past medical history is significant of left ureteric stone. Serum Ca2+ was 14.3 mg/dl and po4 2.4 mg/dl. Bone, GI and renal symptoms present, and high calcium and low phosphorus.

Case 3:

45 y old lady ESRF, Advanced bone disease (usually pt with renal failure has secondary hyperparathyroidism b/c of low calcium and phosphate and can transform to tertiary hyperparathyroidism) , But in this patient with Hx it turns that she has primary hyperthyroidism b/c of adenoma and for many years she had recurrent renal stones until she reached ESRF!

1.1.7 INVESTIGATIONS:

- ↑ Serum Ca2+
- ↑ PTH
- ↓ Phosphorus
- ↑ Chloride, PTH effects on kidney leads to Ca retention
- Imaging X-Ray: Hand X-Ray ⓘ you may see brown tumors.
- Other imaging: U/S can show you Adenoma , CT can sometimes showadenoma but not always , Last thing is nuclear scan" Sestamibi Scan"

1.1.8 MANAGEMENT:

- All symptomatic patients should be treated: A) Adenoma: surgical removal or B) Hyperplasia: remove 3 and a half parathyroid glands (subtotal parathyroidectomy)
- Asymptomatic patients: There is debate on wither asymptomatic patients should be treated or only followed up
- Postoperative management: Be careful of bone hunger syndrome which might cause tetany.

1.1.9 CONCLUSION:

- PHP is a very under diagnosed disease in Saudi Arabia.
- Patients are not diagnosed early
- Complications could be serious and these are avoidable.

1.1.10 RECOMMENDATIONS

- The medical community needs to be more aware of the disease.
- The diagnosis should be especially considered in the following cases:

- Patients with bilateral or recurrent renal stones ⓘ
- Patients with suggestive radiological bone changes
- Patients with high serum calcium level

2 THYROID DISEASES

2.1.1 GENERAL CONSIDERATIONS

- Q: Thyrotoxicosis vs. Hyperthyroidism?
  - A: Thyrotoxicosis is the clinical condition of presence of high levels of thyroid hormones in the Blood.
  - Hyperthyroidism is over activity of the thyroid gland, thus it causes thyrotoxicosis
  - Thyroid disease can present as:
    - Lump “goiter”
    - Change in function (hypo or hyper)
- Q: If we see a lump, how can we tell if it is a thyroid lump?
  - A: Ask the patient to swallow. If it doesn’t move with swallowing then it is not thyroid disease (could be dermoid cyst, lipoma, lymph Node). If it moves then it is one of two:
    - Thyroid lump “goiter”
    - Thryroglossal cysts
- Then you ask the patient to stick his tongue out and if the lump moves then it is a thyroglossal cyst. Because Thryroglossal cysts extend to the tongue.

2.1.2 EXAMINATION:

- A suspected thyroid nodule should be treated as a lump anywhere in the body, but the fluctuation test can not be done due to the presence of pretracheal fascia which fixes the thyroid in position.
- Ultrasound and FNA (Fine Needle Aspiration) are used to differentiate between different conditions. In the thyroid gland, usually what feels like cyst turns out to be solid and what feels solid turnsout to be a cyst.
- Other Important examination points for the thyroid gland:
- A-Neurological Examination:Reflexes are brisk and exaggerated in hyperthyroidism, Reflexes are delayed in hypothyroidism
- **Eye Examination** ⓘ: There are three
  1) Exophthalmos: the eye ball is pushed forwards by the increase in retro-orbital fat, edema, and cellular infiltration.
  2) Lid lag (ask the patient to look down andfollow your finger or a pen and you will seehis eye lid moving slower than his cornea)
  3) Lid retraction
- Hand: Moist, sweaty, pulse is high in hyperthyroidism

2.1.3 CAUSES OF THYROID SWELLING:

1. Thyroid cyst:
   a. Benign
   b. Diagnosed by U/S and FNA
   c. Treated by aspirating the cyst
   d. If it reoccurs up to two times aspirate it again but in the 3rd time surgery should be done

2. Multinodular goiter:
    a. Can present as:
        i. Incidentally
        ii. With or without symptoms of hyper or hypothyroidism

> **Toxic Goiter:**
>
> A goiter that is associated with hyperthyroidism is described as a toxic goiter. Examples of toxic goiters include diffuse toxic goiter (Graves's disease), toxic multinodular goiter, and toxic adenoma (Plummer disease).Nontoxic goiter: A goiter without hyperthyroidism or hypothyroidism is described as a nontoxic goiter. It may be diffuse or multinodular.

        iii. Local compression causing dysphagia, dyspnea, stridor, plethora or hoarseness
        iv. Solid
    b. Diagnosed by US, FNA and then nuclear scan
        i. Warm scan is normal like the lobe on the rightside of the picture
        ii. Hot: abnormal
        iii. Cold (circled area): abnormal. And it means that area is not up taking iodine hence it is no longer thyroid tissue, indicative of malignancies in 15% of patients.

3. Inflammatory (thyroiditis)
    a. Acute, very rare
    b. Sub-acute, very rare
    c. Hashimoto "chronic": most common and usually presents with hypothyroidism> dx by serological markers, on biopsy lymphocytes, monocytes, etc
4. Benign tumor: Follicular adenoma
5. Malignant tumor

2.2 THYROTOXICOSIS

- Thyrotoxicosis can be a manifestation of a number of thyroid conditions, but the most common are:
    1) Grave's disease (usually affects the young)
    2) Toxic multinodular goiter
    3) Toxic nodule

2.2.1 SIGNS AND SYMPTOMS

1. Nervousness
2. Weight loss with Increased appetite
3. Warm moist skin
4. Heat intolerance
5. Sweating
6. Muscular weakness
7. Menstrual irregularities
8. Tachycardia +/- Arrhythmias (especially in elderly where they may present with atrial fibrillation)
9. Goiter

10. **Systolic Bruit & thrill (a bruit maybe heard when applying the stethoscope of the swelling)** ⓘ
11. Eye signs (mentioned earlier)

2.2.2 LAB TESTS

- Increases T4, T3
- Decreased TSH (due to inhibition by high levels of T4 and T3)

2.2.3 MANAGEMENT

1. Medical
2. Radio-nuclear iodine
3. Surgery

Case:

Aisha is a 55-year old lady that presented to your clinic. Her main complaint is related to some recent difficulty in hearing. The family noticed that she started to have difficulty in understanding that she gained weight, and her voice started to be coarse.

Q: How to diagnose?
A: Decreases T4, T3, Increased TSH

2.3 THYROID CANCER

2.3.1 GENERAL CONSIDERATIONS:

- Thyroid cancers are usually non-functional, meaning they do not produce symptoms
- Cancers can appear as solitary nodules or diffusely enlarged glands.
- **A young patient " younger than 20" with a single thyroid nodule should be considered as a case of thyroid cancer (papillary carcinoma is the most common) until proven otherwise** ⓘ
- Lymphatic spread of the cancer does not affect the prognosis

2.3.2 TYPES OF THYROID CANCER:

1. Papillary carcinoma:
    a. Accounts for 85%
    b. Overall most common endocrine cancer
    c. Appears in early adult life
    d. Lymphatic spread
    e. Good prognosis, 5 year survival is >95%
2. Follicular carcinoma:
    a. Accounts for about 10%
    b. Differentiation between benign and malignant is not easy
    c. Blood spread
    d. Doesn't spread to lymph but spreads to bone and blood ⓘ
    e. Prognosis not as good
3. Medullary carcinoma:
    a. Accounts for about 7%
    b. Arises from C-Cells
    c. C-cells secrete calcitonin

MEN IIa: medullary carcinoma, hyperparathyroidisim, pheochormocytoma

MEN IIb: Medullary carcinoma, mucosal neuromas, pheochromocytoma and marfanoid shape

   d. Familial medullary carcinoma accounts for 25% of medullary carcinomas the other 75% are sporadic
   e. Associated with MEN IIa/IIb syndrome (multiple endocrine neoplasia)
   f. Prognosis is not good, especially if it's part of MEN that's why we screen family and we remove thyroid before age of 6 years.
   g. Produces amyloid
4. Undifferentiated (anaplastic):
   a. Usually in Old patients
   b. Accounts for about 1%
   c. Rapidly growing
   d. Locally invasive
   e. Rarely curable
5. Lymphoma
   a. More common in our part of the world
   b. Usually diagnosed post op
   c. Treated by Chemo/radiotherapy.

3 MCQS:

1. **The third postoperative day following thyroidectomy a patient c/o tingling of her finger tips and is found to have serum calcium of 1mmol/l/. The next step in treatment should be**:
   a. Careful observation until the Calcium level increases
   b. Administration of Vit. D
   c. Administration of dihydrotachysterol
   d. Administration of 1,25(OH) 2D (Calcitriol)
   e. Administration of calcium gluconate by slow intravenous drip
2. **Medullary Thyroid Carcinoma**:
   a. Is a tumor of Para follicular C cells
   b. Produces thyroxin as the principle hormone
   c. Are TSH dependent
   d. Can be treated by radio-iodine ablation
   e. T3 act as a tumor marker
3. **What is the least likely cause of Hypercalcemia?**
   a. Metastatic tumor
   b. Sarcoidosis
   c. Acute pancreatitis
   d. Hyperparathyroidism
   e. Vit. D deficiency
4. **The approach to patient with thyroid nodule includes the following except:**
   a. Thyroid scan.
   b. Fine needle aspiration.
   c. Ultrasonography.
   d. TSH T4 T3.
   e. Calcitonin level.

🗝 **Answer key: 1:E 2:A 3:E 4:E**

BREAST DISEASES

1 INTRODUCTION

Overview of the structure and function of the breast.

1.1 ANATOMY OF THE BREAST

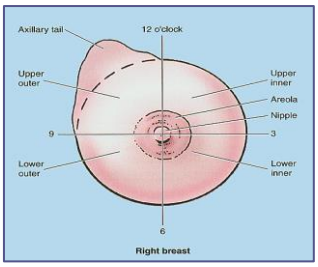
- Breasts (mammary glands) are modified sebaceous glands.
- The breast extends from the 2nd to the 6th ribs and transversely from the lateral border of the sternum to the mid-axillary line.

1.1.1 BREAST BORDERS:

- Upper border: collar bone.
- Lower border: 6th or 7th rib.
- Inner border: edge of sternum.
- Outer border: mid-axillary line.

1.1.2 BREAST DIVISIONS:

Each breast is divided into 5 segments.



- **Four quadrants:**
  By horizontal and vertical lines intersecting at the nipple (upper outer quadrant, upper inner quadrant, lower outer quadrant, and lower inner quadrant). Majority of benign or malignant tumors lie in the upper outer quadrant ⓘ
- **Tail of Spence** (the axillary tail): an additional lateral extension of the breast tissue toward the axilla.

1.1.3 EXTERNAL ANATOMY OF THE BREAST:

- Nipple: pigmented and cylindrical, at the 4th intercostal space (at age 18)
- Areola: pigmented area surrounding the nipple.
- Glands of Montgomery (Montgomery's Tubercles): sebaceous glands within the areola, which act to lubricate the nipple during lactation

Glands of Montgomery can get obstructed (blocked) and inflamed which could raise concerns to the female of a serious pathology, even though it's a simple occlusion.

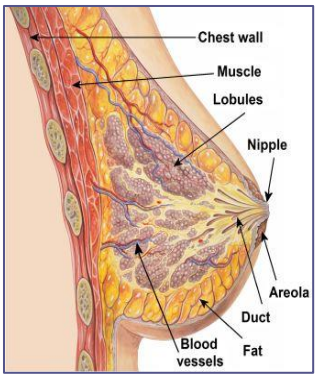
1.1.4 MUSCULATURE RELATED TO THE BREAST:

- The breast lies over the muscles that encases the chest wall. The muscles involved include the pectoralis major (60%), pectoralis minor, serratus anterior (30%), external oblique, latissimus dorsi, subscapularis, and rectus abdominis fascia (10%).

1.1.5 INTERNAL ANATOMY OF THE BREAST:

The breast is composed of 3 different types of tissue:

1. Glandular tissue: It is the milk-producing tissue. Each mammary gland consists of 15-20 lobes. Each lobe is further divided into 20-40 lobules composed of clusters of milk-secreting glands (alveoli/acini) and is drained by a lactiferous duct that opens onto the nipple.
2. Fibrous (supporting) tissue: Strands of connective tissue called the suspensory ligaments of the breast (Cooper's ligaments) extend through the breast to the underlying muscle separating the breast's lobes
3. Fatty tissue: Subcutaneous and retro-mammary fat. It gives the bulk of breast. No fat beneath areola and nipple.



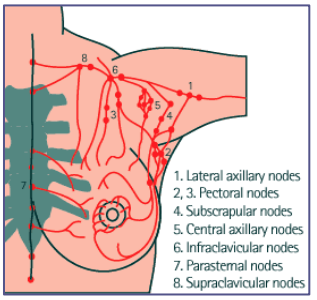
1.1.6 LYMPHATIC DRAINAGE OF THE BREAST:

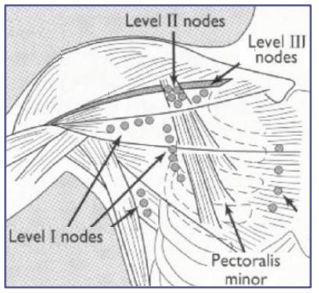
- Superficial lymphatic nodes drain the skin and deep lymphatic nodes drain the mammary lobules.
  - Axillary, infraclavicular, supraclavicular, parasternal (internal mammary)
- Lymphatic drainage of the breast:
  - The medial portion of the breast → to the internal mammary nodes
  - The central and lateral portions 75-80% → drain to the axillary lymph nodes
  - **Axillary lymph nodes**:
    Axillary lymph nodes can be classified anatomically into 5 groups and clinically into 3 levels.

- **Anatomical classification of axillary lymph nodes:**
  1. Anterior (pectoral) group: deep to pectoralis major.
  2. Posterior (subscapular) group: along subscapular vessels.
  3. Lateral group: along the axillary vein.
  4. Central group: within the axillary pad of fat.
  5. Apical group: which drains all of the other groups, lies behind the clavicle at the apex of axilla.

- **Clinical/surgical classification of axillary lymph nodes:**
  - This surgical classification is used in axillary dissection. It is based on the relationship of the lymph nodes to pectoralis minor. There are 3 levels of axillary lymph nodes and options for dissection: ⓘ
    1. Level 1: any lymph node *below* pectoralis minor (first group involved in malignancy), account for 80% of lymph nodes.
    2. Level 2: any lymph node *behind* pectoralis minor.
    3. Level 3: any lymph node *above* pectoralis minor.

Internal mammary nodes most of the time they can't be felt on palpitation, they're only seen on imaging.





1.2 PHYSIOLOGY OF THE BREAST

Normal physiological breast changes in females.

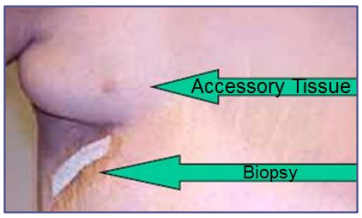
- Puberty: need estrogen and progesterone.
  - Estrogen: growth and appearance, milk-producing system.
  - Progesterone: lobes & alveoli, alveolar cells become secretory.

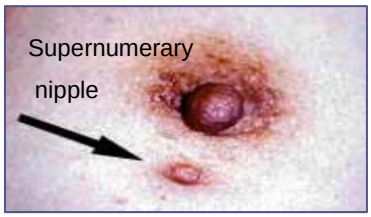
- Asymmetry is common ⓘ
- Menses:
  - Progesterone: 3-7 days prior to menses, engorgement.
  - Physiologic nodularity: retained fluid.
  - Mastalgia.
- Pregnancy and lactation:
  - Glandular tissue displaces connective tissue.
  - Increases in size.
  - Nipples prominent and darker.
  - Mammary vascularization increases.
  - Colostrum present.
  - Attain Tanner stage V with birth.
- Aging:
  - Perimenopause: decrease in glandular tissue, loss of lobular and alveolar tissue.
  - Fatten, elongate, pendulous.
  - Infra-mammary ridge thickens.
  - Suspensory ligaments relax.
  - Nipples flatten.
  - Tissue feels “grainy”.

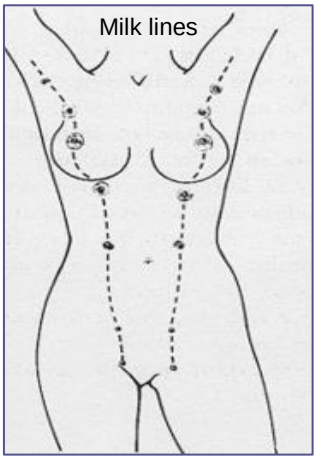
ⓘ Asymmetry is a common concern among female adolescents. Typically, the asymmetry is more noticeable during puberty and eventually breast size evens out during development. If it was a major and persistent asymmetry a breast augmentation or reduction surgical procedure may be considered **AFTER** breast development/puberty is complete (NEVER interfere surgically during puberty).

1.3 NORMAL VARIATIONS OF THE BREAST

- Accessory breast tissue and supernumerary nipples, which develop along the milk lines (sites of accessory breast tissue and nipples).
- Hair.
- Lifelong asymmetry.







2 CLINICAL APPROACH

- History
- Clinical examination
- Imaging
- Cytology and tissue diagnosis.

Management of a patient with a breast **lump**:

- History and examination
- Ultrasound and mammogram if above 35 years old.
- FNAC or core biopsy or excision biopsy
- Definitive treatment which is either:
  - *Observation*
  - *Excision*
  - *If malignant, along the lines of cancer cases*

2.1 SPECTRUM OF COMPLAINTS

- Women come to see a breast surgeons for the following reasons:
  1. Breast lump (painful/painless) - 60% ⓘ
  2. Anxiety – 20%
  3. Breast pain without a lump – 10%
  4. Nipple discharge – 5%
  5. Change in breast contour – 2%

6. Nipple-areolar complex disorder – 1%
7. Axillary mass – 1%
8. Screen detected lesion – 1%

- Triple assessment of a patient with a **lump**:ⓘ
  - History and examination
  - Mammogram (99%) if above 35 years old
  - F.N.A

2.2 HISTORY

Full and complete history should be taken, particular attention should be paid to:

- Age of the patient (e.g. 45 y/o lady has a higher risk than 16 y/o)
- Breast development stating from childhood to present.
- Endocrine status of patient mainly menstruation and OCP use.
- Size of lump in relation to menses.
- Pattern of pain in relation to menses.
- How regular the cycle is and quantity of blood.
- Changes in breast during previous pregnancies e.g. abscess, nipple discharge, retraction of nipple.
- Number of pregnancies.
- Breast feeding
- Abnormalities which took place during previous lactation period e.g. abscesses, nipple retraction, milk retention.
- Family history of breast diseases especially cancer and particularly in near relatives.
- Nipple discharge.
- Age at menarche.
- Age at 1st birth.
- L.M.P.
- For post-menopausal women: H.R.T (hormonal replacement therapy) and date of menopause.

Breast presentation:

-Skin dimpling: carcinoma, aging, breast infection, previous breast surgery

-Changes in nipple/areola: Duct ectasia, carcinoma, paget's disease, eczema

-Painless lump: carcinoma, cyst, fibroadenoma, fibroadenosis

-Painful lump: cyst, periductal mastitis, abscess, sometimes carcinoma

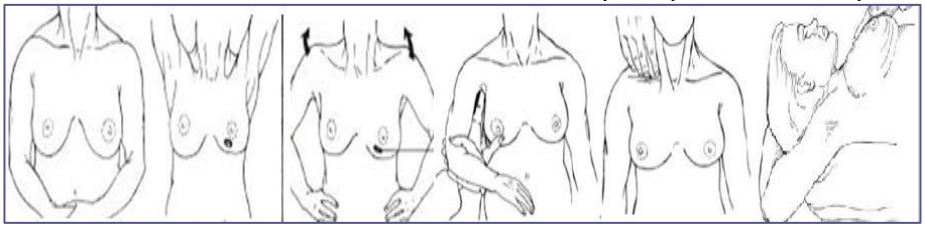
-Pain and tenderness (no lump): cyclical, non-cyclical, very rarely a carcinoma

-The cardinal signs of a late cancer of the breast: hard, non-tender, irregular lump, tethering or fixation, palpable axillary lymph nodes.

2.3 CLINICAL EXAMINAITON

- **Exposure**: from the waist and above.
- **Position**: sitting, supine and 45°
- **Inspection**
  - Inspect both breasts by having the patient perform the following maneuvers while sitting:
  - Patient's arms by her side.
  - Patient's arms above her head.
  - Patient's arms on her hips with valsalva (pectoral contraction maneuver).
  - Leaning forward while sitting.
  - Note for size, symmetry, skin changes (dimpling or tethering), nipple complex (inversion or retraction), color, contour, and scars.
  - Inspect axillae with the patient's arms over her head.
- **Palpation:**
  - Patient should be lying supine.
  - Place pad under shoulder to flatten breast.

- Raise arm over her head.
- Abnormal finding? Check the other breast.
- Palpate both breasts
  - Palpate Sitting
  - Rest arm in your hand and palpate axilla. Her arm should be relaxed.
  - Palpate supra-clavicular and infra-clavicular nodes
- Using preferred pattern
- Palpate with pads of three fingers
- Note for any nipple discharge
- Palpate all *lymph nodes* (must examine ALL)
  - From distal arm to under arm with deep palpation
  - Axillary (pectoral, medial, lateral, posterior, central)
  - Supraclavicular
  - Infra-clavicular
  - Nodes deep in the chest or abdomen
  - Infra-mammary ridge: shelf in the lower curve of each breast (Usually missed during clinical examination)
- Examine normal side first.
- Examine abdomen and the back/lumbar spine (for metastasis)



Breast self-examination and mammography:

-BSE should be done once a month, preferably just after period. If the women is post-menopausal, she should choose a day that she will remember each month

2.4 IMAGING

- When to image?
  - Investigation of a palpable lump or nipple discharge.
  - Screening in appropriate groups (asymptomatic 40 y/o)
  - Metastatic adenocarcinoma with an unknown primary.

2.4.1 MAMMOGRAPHY

2.4.1.1 DIAGNOSTIC VS. SCREENING MAMMOGRAPHY:

- **Diagnostic mammography** → performed in order to evaluate a breast complaint or abnormality detected by clinical examination.
- **Screening mammography** → performed for asymptomatic 'well' women to detect unsuspected lesions. E.g. routine screening for women who are 40 years or older.

2.4.1.2 CARDINAL MAMMOGRAPHIC FEATURES OF MALIGNANCY

- Speculated mass (stellate lesions) → check for the presence of a surgical scar. All other stellates are presumed invasive carcinoma that requires work up and biopsy. If unexplained, don't be seduced by stability.
- Architectural distortion without mass→ should be treated as stellate lesion.
- MICRO-calcifications with casting or irregularity→ 60% of localization biopsies are for calcifications, but only 25% of these yield malignancy.

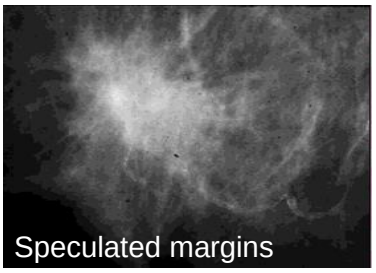
Imaging features which can be associated with ductal carcinoma in situ (DCIS):

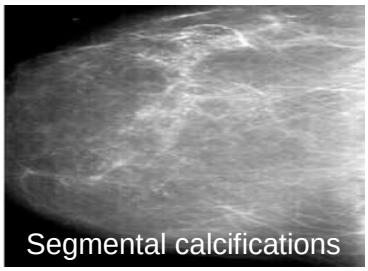
- Micro-calcifications linear (75-90%)
- Circumscribed mass
- Ill-defined mass
- Prominent duct or nodule
- Architectural distortion
- Asymmetry
- Sub-areolar mass

Benign Vs. Malignant calc.:

Based on size → Macrocalc. are always benign. Microcalc. mostly benign but can be malignant.
Based on shape → Benign: punctate, linear, spherical, popcorn, vascular, smoothly dense.
Malignant: mostly ductal, segmental and clustered.
Based on distribution → widespread bilateral distribution is suggestive of a benign process

- Distribution (casting, linear, segmental, clustered) → segmental and clustered indicate malignancy.
- Morphology (pleomorphism).
- Relationship to parenchyma.
- Circumscribed density with distinct margins.
- Asymmetric density


Speculated margins


Segmental calcifications

2.4.2 BENIGN VS. MALIGNANT IMAGING CHARACTERISTICS IN BREAST CANCER:

Benign	Malignant
Circumscribed mass	Spiculated mass
Fat-containing lesion	Architectural distortion with no history of prior surgery
Microcalcifications	Microcalcifications
Round, uniform density, large, coarse	Linear, branching, pleomorphic, casting
Widely scattered	Tightly clustered
Long axis of the lesion is along the normal tissue planes	Lesion is taller than it is wide
Homogeneous internal echotexture	Decreased hyperechogenicity
Hyperechogenicity	Marked acoustical shadowing
Smoothly marginated	Spiculation

2.4.3 ULTRASONOGRAPHY

2.4.3.1 ROLE OF ULTRASOUND:

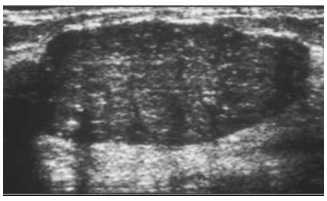
- Characterize a mammographic abnormality.
- Characterize a mammographically occult clinical abnormality.
- Initial examination in the younger women.
- Imaging guided biopsies.
- Some utility in distinguishing benign from malignant lesions.
- Still no role in screening even in the mammographically dense breast
- Developing role in monitoring neo-adjuvant therapy?
- What does ultrasound look for?
  - Location of a lesion.
  - Solid Vs. Cystic.
  - Margins.
  - Surrounding structures.

2.4.3.2 ADVANTAGES AND DISAVDVANTAGES OF ULTRASOUND

- **Advantages**:
  - Painless
  - Does not use ionizing radiation
  - Very good at detecting cysts
  - Can “see through” mammographically dense breasts (done at any age)
- **Disadvantages**:
  - Not good for screening the breast
  - Cannot always characterize lesions precisely
  - More operator-dependent than mammography

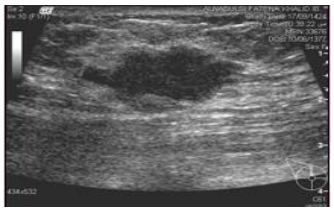
2.4.3.3 ULTRASONOGRAPHIC FEATURES

- Cysts:
  - Contain no or few echoes
  - Have smooth margins
  - Often compressible with ID
  - Have posterior enhancement (increased echoes=whiter)



U/S Fibroadenoma

- Benign masses:
  - Have smooth margins
  - Have relatively uniform internal appearance
  - Don’t disturb surrounding tissues
  - Are usually wider than tall
- Malignant masses:
  - Have irregular or indistinct margins
  - Have heterogeneous internal appearance
  - Often cut across surrounding tissue planes
  - Are often taller than wide or rounded (special types)
  - The normal breast tissue appear as symmetrical waves under U/S. Malignant lesions disturb that pattern but benign lesions follow that pattern.



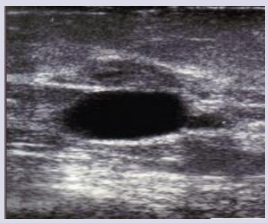
Speculated margins (suggestive of malignancy)

2.4.3.4 ULTRASOUND CORRELATION

Ultrasound/clinical correlation is as important as ultrasound/mammographic correlation! Ultrasound can be considered as an *extension of palpation*.

- **Challenges for ultrasound correlation:**
  - Small lesions in larger breasts.
  - Small lesions deep within echogenic parenchyma.
  - Dense parenchyma interspersed with fatty lobules.
  - Surgically scarred breasts.
  - Multiple mammographic lesions.
  - Complicated cysts.
  - Cellular malignancies.
- **Fundamentals of mammographic/ultrasound correlation:**
  - Correlate lesion location
  - Correlate lesion size
  - Correlate lesion margin

Cysts can present as a palpable mass or a focal tender area within the breast. A majority of cysts are found in asymptomatic women on their screening mammogram.
-On mammography: they appear as a mass and may have associated benign rim/eggshell calcifications.
-On ultrasound: it is the confirmatory diagnostic test, demonstrates a well-defined mass devoid of internal echotexture (if any internal echoes are present, U/S guided FNA is recommended to fully exclude malignancy)



Benign mass: a simple cyst

  - Don’t assume that previous imaging assessment was correct (pull out all the films if necessary).
  - Take account of both mammographic & ultrasonography appearances.
  - Most probably benign lesions are benign. Out of 543 probably benign lesions in 5514 screening mammograms, only 1 was malignant (0.2%), and 21% regressed or disappeared.
- **Key points:**
  - Meticulous imaging technique.
  - Careful correlation of mammogram with ultrasound, and imaging with clinical findings.
  - Clear communication reduces errors.

2.5 CYTOLOGY AND BIOPSY

- **Fine-needle aspiration cytology**
  - Procedure description: a thin needle is inserted into the mass for sampling of cells that are later on examined under a microscope.
  - Clinical, U/S guided, mammotomes.
  - Sensitivity 80-98%
  - False negative 2-10%
  - Scoring of result code 9 → code 5
- **Core biopsy**
  - Tissue diagnosis
  - Painful
  - Costly
  - Receptor status
- **Open biopsy**

2.6 NIPPLE DISCHARGE

- 5% of women coming to the clinic complain of nipple discharge.
- 95% of these complaints are benign.

2.6.1 CAUSES OF NIPPLE DISCHARGE

Commonest causes in non-pregnant women:

- Carcinoma
- Intra-ductal papilloma (**most common** cause) ⓘ
- Fibrocystic changes
- Duct ectasia
- Hypothyroid
- Pituitary adenoma (prolactin secreting adenoma, can present with galactorrhea)

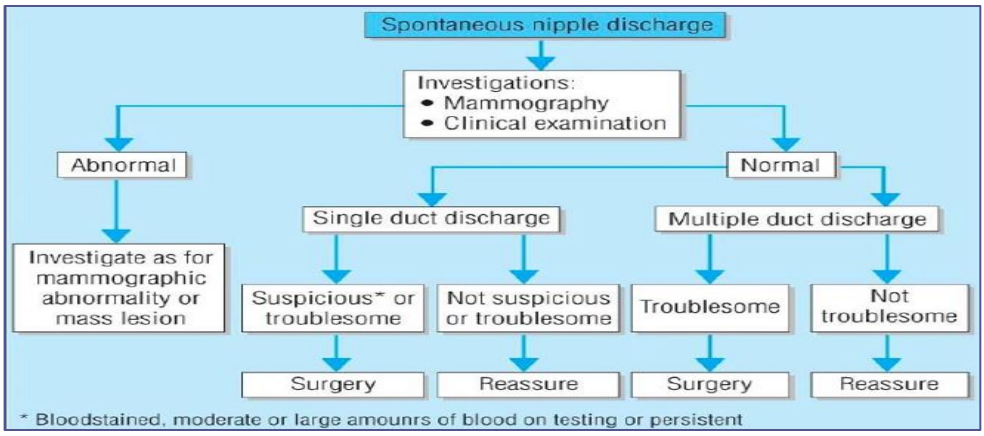
2.6.2 CLINICAL CHARACTERISTICS:

- **Physiologic** discharge (e.g. lactation) → usually bilateral, multiple ducts, non-spontaneous, screen for phenothiazine use (antipsychotic)
- **Pathologic** discharge → Unilateral, spontaneous (without squeezing the nipple), single duct, discolored discharge

2.6.3 CLINICAL EVALUATION

For further evaluation of spontaneous nipple discharge a ductography can be performed. Ductography is useful in detecting the location of the lesion within the duct and the extent of involvement. This information can be extremely helpful in pre-surgical planning.

- Most important points in **history** of nipple discharge are ⚐
  - *Is it spontaneous or on pressure? Is it coming from single or multiple?*
  - Colors: *Serous, serosanguinous, bloody, clear, milky, green, blue-black.*
- R/O mass by clinical examination and mammogram.
- Identify source of discharge and test for presence of blood in discharge
- Consider ductography
- Lab tests: thyroid, prolactin.



2.6.4 MANAGEMENT:

- Physiologic
  - Treat cause if present
  - Follow-up 6 months (observation)
- Pathologic
  - Biopsy and excise (single duct excision or total duct excision)

3 COMMON BENIGN BREAST DISORDERS

- Fibrocystic changes
- Fibroadenoma
- Intraductal papilloma
- Mammary duct ectasia
- Mastitis
- Fat necrosis
- Phylloides tumor
- Male gynecomastia
- Galactocele

3.1 FIBROCYSTIC CHANGES

3.1.1 CHARACTERISTICS

- Most common breast pathology
- Lumpy, bumpy breasts
- 50-80% of all menstruating women
- Commonest incidence among age 30-50
  - 10% in women less than 21
- Caused by hormonal changes prior to menses

📄 Galactocele:

Def.: Cyst containing milk.

CP: dull aching pain with a well formed lump

Diagnosis: clinically or U/S

Management: aspiration both therapeutic and diagnostic under full aseptic technique to prevent infection. If it appeared small on U/S there's no need to aspirate just reassure the patient. If it accumulates again then aspirate again while reassuring the patient that it'll resolve after lactation period.

📄 ⓘ **Additional notes**:

• Big cyst >2 cm→ must aspirate

• Atypia or hyperplasia → if atypia / hyperplasia / dysplasia changes were present must EXCISE, if simple then just reassure the patient & conservative management.

• Complicated cyst (i.e. both solid and cystic components) →

Biopsy is needed from solid component to exclude malignancy.

• Constant cyst (i.e. doesn't change with multiple imaging in different times) → must biopsy

- Relationship to breast cancer doubtful

3.1.2 SIGNS AND SYMPTOMS

- Mobile cysts with well-defined margins
- Singular or multiple
- May be symmetrical
- Upper outer quadrant or lower breast border
- Pain, discomfort and tenderness
- Cysts may appear quickly and decrease in size
- Lasts half of a menstrual cycle
- Subside after menopause, if no HRT.

3.1.3 INVESTIGATIONS

- Aspirate cyst fluid ⓘ
  - If bloody → go for surgical biopsy.
  - If non-bloody and disappear completely → observe.
  - If non-bloody and doesn't resolve → surgical biopsy.
- Imaging for questionable cysts
  - In young patients only U/S is performed show multiple cysts
  - In 40 and above patients both U/S and mammogram are performed to exclude any underlying malignant pathologies.

3.1.4 MANAGEMENT

- Treatment based on symptoms
- Reassure patient
- ''Atypical Hyperplasia'' on pathology report indicates increased risk of breast cancer→ must excise
- ***Comfort measures:***
  - Eliminate Methylxantines (coffee, chocolate): may take 6 months for relief.
  - Local heat/cold
  - Wear a good supporting bra
  - Low-Sodium diet
  - Vitamin E: Antioxidant but do not take more than 1200/day
- ***Medications for mastalgia:***
  - NSAIDS (simple analgesia)
  - Monophasic oral contraceptive pills (to stabilize hormonal levels)
  - Spironolactone
  - Dopamine Agonists: Bromocriptine
  - Rare or former use: Danazol (for severe cases, side effects include acne and hirsutism, only 50% respond to it, mostly not used), Tamoxifen, GnRH agonist (Luprolide)

ⓘ Fibroadenoma: To leave alone or to excise?

• EXCISE if →

- >3-4 cm or giant fibroadenoma

-localized

-painful

-rapidly growing

-a family history of malignancy (does NOT mean that fibroadenoma is pre-malignant but done only to relieve the patient's worries)

-patient's preference

-indeterminate diagnosis

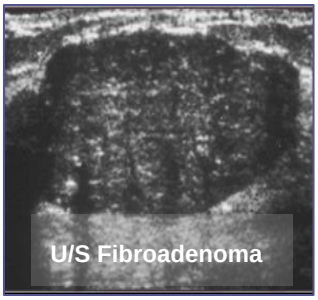
-if 35 y/o and older recommended

•If left alone → it'll either remain the same or regress (some patients during pregnancy it regresses) or increase in size or calcify

3.2 FIBROADENOMA

3.2.1 CHARACTERISTICS

- Second most common breast condition (**most common lump**)
- Most common in black women
- Late teens to early adulthood (15-30 years old of age)


U/S Fibroadenoma

- Rare after menopause
- Totally benign, and **NO** malignancy potential

3.2.2 SIGNS AND SYMPTOMS


Mammogram showing multiple calcified fibroadenomas

- Firm, rubbery, round, mobile mass
- Painless, non-tender
- Solitary, 15-20% are multiple
- Well circumscribed
- Mostly located in upper-outer quadrant of the breast
- 1-5 cm or larger (if more than 5 cm it is called a giant fibroadenoma)

3.2.3 INVESTIGATIONS AND TREATMENT

- Triple assessment
- Imaging: U/S mostly used because its more common in young and mammogram
- Biopsy
- Excision and close follow-up

3.3 INTRADUCTAL PAPILLOMA

3.3.1 CHARACTERISTICS

- Slow-growing
- Overgrowth of ductal epithelial tissue
- Usually not palpable
- Cauliflower-like lesion
- Length of involved duct
- **Most common cause of persistent bloody nipple discharge** (**IMPT)**
- 40-50 years of age

Papilloma: To leave or to excise?

- If single papilloma → can observe and see if it disappears
- If it doesn't resolve → excise
- If presented with intraductal PAPILLAMATOSIS (appears as multiple filling defects on ductogram) considered a pre-malignant condition → must excise
- Exclude malignancy in young by US or ductogram (filling defect), if 40 and above by U/S, ductogram and mammogram

3.3.2 SIGNS AND SYMPTOMS

- Watery, serous, serosanguinous, or bloody discharge
- Spontaneous discharge
- Usually unilateral
- Often from single duct → pressure elicits discharge from single duct
- 50% no mass palpated

3.3.3 INVESTIGATIONS AND TREATMENT

- Test for occult blood
- Ductogram
- Biopsy
- Excision of involved duct

3.4 MAMMARY DUCT ECTASIA

3.4.1 CHARACTERISTICS

- Inflammation and dilation of sub-areolar ducts behind nipples, completely benign

- May result in palpable mass because of ductal rupture
- Greatest incidence after menopause
- Etiology Unclear → Ducts become distended with cellular debris causing obstruction

3.4.2 SIGNS AND SYMPTOMS

- Multi-colored discharge
  - Thick, pasty (like toothpaste)
  - White, green, greenish-brown or serosanguinous
- Intermittent, no pattern
- Bilaterally from multiple ducts
- Nipple itching with drawing or pulling (burning) sensation

3.4.3 INVESTIGATIONS AND TREATMENT

- Test for occult blood
- Imaging → Mammogram and sonogram
- Biopsy → Excision of ducts if mass present
- Antibiotics
- Close follow-up

3.5 MASTITIS

3.5.1 CHARACTERISTICS

- Breast infection when bacteria enter the breast via the nipple
- Ducts infected
- Fluid stagnates in lobules
- Usually during lactation
- Penicillin resistant staphylococcus common cause

3.5.2 SIGNS AND SYMPTOMS

- Pain and tenderness
- Nipple discharge: -Pus -Serum -Blood
- Localized induration
- Fever and rigor
- Abscess: localized tenderness, severe fever and rigor

3.5.3 TREATMENT

- Antibiotics
  - ''Oxacillins'' for PP mastitis (PP=postpartum=after childbirth)
  - Cephalosporin for other abscesses → cephalexin, Keflex
- Empty breast if PP
- Incision and drainage of abscess

3.6 FAT NECROSIS

3.6.1 CHARACTERISTICS

- Cause

The problems behind this condition:

• Infection:

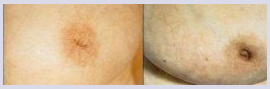
Duct ectasia→stasis→risk of infection→higher chance of abscess (periductal mastitis) caused by mixed organisms→broad spectrum antibiotics and abscess drainage.

•Similar presentation to malignancy:

Inflammation→can retract nipple→ patient is worried about malignancy→ U/S and mammogram according to age→reassure the patient, if 40 and above take aspiration for cytology

-left picture: slit-like nipple characteristic of duct ectasia

-right picture: nipple retraction from carcinoma



ⓘ Lactating women presented with fever, painful and tender breasts → most likely mastitis →broad-spectrum antibiotics (cephalosporin 1st generation IV), warm sponges, if abscess must drain it.

  - **Trauma** to breast (e.g. seat belt trauma in car accidents)
  - Surgery
- Necrosis of adipose tissue

3.6.2 SIGNS AND SYMPTOMS

- Pain or mass → usually non-mobile mass

3.6.3 TREATMENT

- Resolves over time without treatment but may be excised

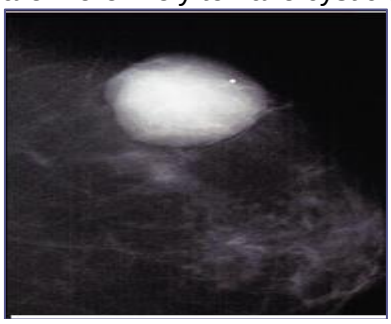
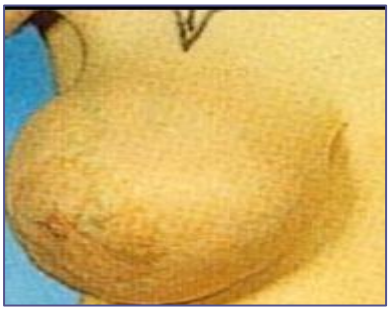
3.7 PHYLLOIDES TUMOR (CYSTOSARCOMA)

3.7.1 CHARACTERISTICS

- Giant fibroadenoma (a variant of fibroadenoma) with rapid growth (patient presents with a history of a rapidly growing mass)
- **Malignant** potential, lesions > 3 cm are more likely to be malignant
- Most are benign, 25% recur locally if incompletely excised
- The malignant form of this lesion mostly locally malignant (about 10%) can metastasize hematogenously to the lungs and not to the axillary lymph nodes
- Often occurs in women aged 40+

3.7.2 INVESTIGATIONS AND TREATMENT

- Imaging: both mammography and ultrasound, they present as well-defined masses that are very similar to a benign fibroadenoma. The malignant forms are more likely to have cystic spaces on U/S

- Treatment → excision is the **only** treatment! Chemotherapy and radiotherapy are not effective.

3.8 MALE GYNECOMASTIA

3.8.1 CHARACTERISTICS

- Diffuse hypertrophy of breast
- 30-40% of male population
- Adolescence and older men
- Caused by imbalance of estrogen/testosterone
- Medical conditions (hepatitis, COPD, hyperthyroidism, TB)
- May be associated with genetic cancer families

Inflammatory carcinoma vs. mastitis:

-may have similar *appearance*, but completely **different history**

-inflammatory carcinoma →non-lactating elderly, peau d'orange, must perform U/S, mammogram and biopsy.

Can a lactating female with abscess still breast feed her newborn? YES, except if her baby appeared to be sensitive to antibiotic administered

Carcinoma vs. fat necrosis:

-fat necrosis is important because both clinically and radiologically can appear very similar to malignancy. In order to exclude cancer a biopsy should be performed.

- Must exclude testicular and adrenal malignancies (hormone producing tumors)
- Medications associated with gynecomastia:
  - Marijuana
  - Narcotics
  - Phenothiazines
  - Diazepams
  - Anything that affects the CNS

3.8.2 TREATMENT

- If pre-puberty → wait to see if it resolves
- Change medication
- Treat underlying illness
- Occurs in families with genetic mutation
  - Colon, prostate cancer

4 BREAST CANCER

4.1 FAST FACTS

- Killer of women:
  - USA 1:8
  - KSA? 1:15
  - 187000 cases of cancer breast in one year (USA)
  - 45000 deaths due to it in one year (USA)
- Breast cancer is the most common cause of death from cancer in western women
- Every day in Australia, over 30 women discover they have breast cancer
- In Australia 11,400 people (11,314 women and 86 men) were diagnosed with breast cancer in 2000.
- 9 out of 10 women who get breast cancer do not have a family history of the disease
- Age is the biggest risk factor in developing breast cancer – over 70% of cases occur in women over 50 years
- Women aged 50–69 who have a breast screen every two years can reduce their chance of dying from breast cancer by at least 30%
- Breast cancer is the most common cancer in women aged over 35 years - 25% of all cancers diagnosed
- The average age of diagnosis of breast cancer in women is 45 - 55 years
- During the period 1994 to 1998, the five year survival rate for women diagnosed with breast cancer was 85 %
- Although we know of many factors that contribute to the risk of women getting breast cancer, the cause remains unknown
- Most common type of breast cancer is ductal carcinoma.
- Five year survival rates

Stage at diagnosis	Survival rates (%)
Localized	96.8
Regional	75.9
Distant	20.6

*based on U.S. statistics from 1986 to 1993, reprinted with permission from American Cancer Society.

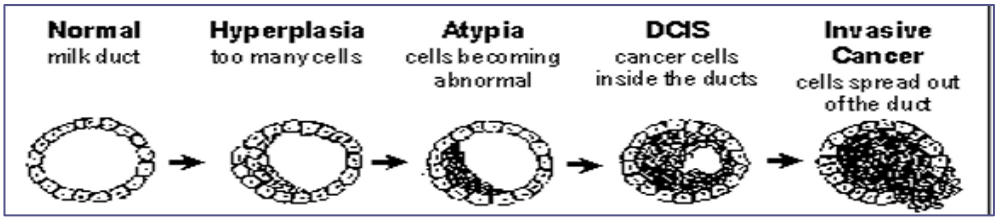
Established risk factors for breast cancer in women:

Relative risk >4	- Age (older age group higher risk) - Country of birth (North America, Northern Europe) - Mother and sister with history of breast cancer, especially at an early age - Biopsy confirmed atypical hyperplasia and a history of breast cancer in a first degree relative
Relative risk 2.1	- Nodular densities on mammogram occupying >75% of breast volume - History of cancer in one breast - Radiation to chest - Mother or sister with history of breast cancer, diagnosed at an early age - Biopsy-confirmed atypical hyperplasia without a family history of breast cancer
Relative risk 1.1	- Socioeconomic status (high) - Place of residence (Urban - Race/ethnicity (White >45 and Black <45) - Religion (Jewish) - Nulliparity, breast cancer >40 years of age - Age at first full-term pregnancy, age at menarche, age at menopause - History of primary cancer in endometrium, ovary - Obesity (Obese breast cancer > 50 years, Thin breast cancer <50 years)

4.2 STAGING CLASSIFICATION OF BREAST TUMORS

Stage 0	Stage 1	Stage 2	Stage 3	Stage 4
Neither palpable tumor nor axillary lymph nodes.	Tumor less than 2 cm, no lymph node involvement	Tumor more than 2 cm but less than 5 cm, 1 ipsilateral axillary lymph node involvement (movable)	Tumor more than 5 cm, with skin involvement or fixation, and involvement of fixed lymph node	Tumor of any size with distant metastases such as bone, liver, lungs, brain and including supraclavicular node involvement

50 y/o female with a 2 cm tumor and liver metastasis → stage 4



4.3 HISTOPATHOLOGICAL TYPES OF BREAST CANCER

- **Infiltrating (or invasive) Ductal Carcinoma (IDC)**
  - Starting in a milk passage, or duct, of the breast, this cancer breaks through the wall of the duct and invades the breast's fatty tissue. It can spread to other parts of the body through the lymphatic system and through the bloodstream. Infiltrating or invasive ductal carcinoma accounts for about 80 percent of all breast cancers. Most common type.
- **Infiltrating (or invasive) Lobular Carcinoma (ILC)**
  - This type of cancer starts in the milk-producing glands. About 10 to 15 percent of invasive breast cancers are invasive lobular carcinomas. These are multicenteric, and they can appear in the other breast as well (bilateral).
- **Medullary Carcinoma**
  - This type of invasive breast cancer has a relatively well-defined distinct boundary between tumor tissue and normal breast tissue. It accounts for about 5 percent of all breast cancers. The prognosis for medullary carcinoma is better than that for invasive lobular or invasive ductal cancer
- **Colloid Carcinoma**
  - This rare type of invasive disease, also called mucinous carcinoma, is formed by mucus-producing cancer cells. Prognosis for colloid carcinoma is better than for invasive lobular or invasive ductal cancer.
- **Tubular Carcinoma**
  - Accounting for about two percent of all breast cancers, tubular carcinomas are a special type of invasive breast carcinoma. They have a better prognosis than invasive ductal or lobular carcinomas and are often detected through breast screening.
- **Adenoid Cystic Carcinoma**
  - This type of cancer rarely develops in the breast; it is more usually found in the salivary glands. Adenoid cystic carcinomas of the breast have a better prognosis than invasive lobular or ductal carcinoma.

Surgical treatment of breast cancer depending on stage:

Stage 1 and 2 → WLE or mastectomy, axillary nodes <u>then</u> radiotherapy and chemotherapy

Stage 3 → neo-adjuvant chemotherapy <u>then</u> surgery

Stage 4 → <u>no</u> role of surgery

4.4 PROGNOSTIC FACTORS

- Size of tumor
- Grade of tumor
- Lymph nodes involvement

Mammogram of DCIS with malignant microcalcifications. Note the fine, linear, heterogeneous clustered calcifications associated with an ill-defined mass lesion. Although the hallmark imaging feature for DCIS is the presence of microcalcifications, DCS can also present less frequently without them.

5 BREAST CANCER: Treatment

5.1 DUCTAL CARCINOMA IN SITU TREATMENT

- Depending on the degree of DCIS the options of treatment are
  - Total mastectomy
  - Lumpectomy
  - Lumpectomy and radiation therapy
- DCIS does not spread to the axillary lymph nodes so these are usually not removed.

5.2 LINES OF TREATMENT

- Surgery:
  - For Stage I and II WLE or mastectomy + axillary nodes.
  - Surgical Intervention: 1. Mastectomy 2. W.L.E (wide local excision)
- Radiotherapy.
- Chemotherapy.
- Hormonal therapy.
- Ovarian ablation.
- Reconstruction

5.2.1 CHEMOTHERAPY

- Chemotherapy for breast cancer is usually given in cycles every 3 or 4 weeks.
- The common schedules include:
  - CMF (Cyclophosphamide, Methotrexate and 5-Flurouracil)
  - AC (Adriamycin, Cyclophosphamide)
  - Taxol or Taxotere
- Chemotherapy side effects:
  - Fatigue
  - Anorexia
  - Nausea and vomiting
  - Hair loss
  - Effects on the blood.
  - Mouth problems
  - Skin problems
  - Fertility
  - Bowel problems

5.2.2 RADIOTHERAPY

- Side effects

	Common reactions	Uncommon reactions
During the course of treatment	✔ skin reddening and irritation ✔ Fatigue ✔ loss of hair ✔ sore throat	✔ skin blistering ✔ nausea ✔ rib fractures (less than 1 in every 100)
After the course of treatment	✔ Discomfort and sensitivity in the treated area. ✔ increased firmness ✔ swelling of the treated breast	✔ Pneumonitis and scarring (about 1 or 2 women in every 100 women between 6 weeks and 6 months after therapy

5.2.3 TAMOXIFEN

- Tamoxifen is a drug that has been used for the treatment of breast cancer. It can increase survival for some women with breast cancer and reduce their risk of developing cancer in the opposite breast. Tamoxifen is sometimes used whose breast cancer recurs.
- It is also being tested to see if it can prevent the development of breast cancer in unaffected women who are at an increased risk because of a strong family history of the disease.
- Tamoxifen is taken by mouth. Tablets are either 10 mg or 20 mg.
- It is usually started after surgery or after the completion of radiation Rx
- Tamoxifen should take it at the same time each day.
- Currently the recommended length of Tamoxifen therapy is five years.

Common side effects	Uncommon side effects
✔ **Hot flushes or sweats** ✔ **Irregular menstrual periods (in women who have not gone through the menopause)** ✔ **Vaginal irritation, including vaginal dryness or discharge** ✔ **Fluid retention and weight gain**	✔ Light-headedness, dizziness, headache or tiredness ✔ Rash ✔ Nausea

5.3 LYMPHOEDEMA

- Definition: Lymphedema is long-term swelling of the arm after axillary surgery or radiotherapy to the axilla.
- Symptoms: include a general heaviness of the arm, a swelling of the fingers or sometimes difficulty putting on a long sleeve.
- The earlier treatment is started the easier it is to achieve good results.
- Less than 1 in 10 women who have had either lymph glands removed or radiation to the armpit will develop noticeable lymphedema. This risk increases to 1 in 3 if the pt. had both of these treatments.
- It can occur any time after the operation, even up to 10 years.

5.4 POST-OPERATIVE BREAST RECONSTRUCTIONS

- The aim of breast reconstruction is to rebuild the breast shape and, if desired, the nipple and the areola.
- Benefits:
  - Reconstruction usually doesn't restrict any later treatments, nor does it usually interfere with radiotherapy, chemotherapy or hormone therapy.
  - The patient will not need to wear an external prosthesis.
  - Follow-up after the operation is no more difficult and any recurrence of cancer in the area can still be detected.
  - Some women feel more self-confident and feminine after reconstruction
- There are two main types of breast reconstruction:
  - tissue or skin expander with breast implants
  - flap reconstruction

6 MCQS

1. Ductal carcinoma in situ (breast):
   a. In the great majority of cases presents as a palpable mass
   b. Usually present as mammographic finding of micro-calcification
   c. Mastectomy is the treatment of choice in all cases
   d. Axillary dissection is an integral part of its surgical treatment

2. It is advisable to remove a fibroadenoma if:
   a. It is painful
   b. It is more than 3 cm in size
   c. There is a positive family history of breast cancer
   d. All of the above

3. All the following are mammographic features of breast carcinoma except?
   a. Skin and nipple discharge
   b. Diagnostic for women below 20
   c. Speculated mass
   d. Micro-calcification

4. All true for fibroadenma except:
   a. Microscopically have both epithelial and stromal components
   b. During pregnancy and lactation may undergo partial/total infarction
   c. Affect old females
   d. Are pseudocapsulated

5. Regarding intraductal papilloma. All true except?
   a. Characterized by papillary configuration
   b. Solitary intraductal papilloma's are lesion of large duct
   c. May present with bloody nipple discharge
   d. Does not require surgical excision

6. Which of the following factors increases the risk of breast cancer among women?
   a. Obesity and nulliparity
   b. Age at menarche
   c. Multiple pregnancies
   d. Low-fiber diet

🗝 Answer Key: 1;B , 2;D , 3;B , 4;C , 5;D , 6;A

ADRENAL GLAND DISEASE

1 INTRODUCTION

1.1 HISTORY

- 1563: Anatomy
- 1855: Addison described clinical features of the syndrome named after him (primary adrenal insufficiency)
- 1912: Cushing described hyper-cortisolism [Cushing's disease vs. syndrome: Disease, problem in pituitary but syndrome is anything else apart from pituitary]
- 1934: The role of adrenal tumors in hypercortisolism understood
- 1955: Pheochromocytoma was first described by Frankel (before that all patients with this disease died b/c of crisis and there was no treatment)
- 2003: First robotic adrenalectomy was performed.

1.2 EMBRYOLOGY

- Paired gland (almost as big as dates)
  - Cortex (coelomic epithelium)
    - Outermost layer: Zona glomerulosa → Mineralocorticoids
    - Intermediate layer: Zona fasciculata → Glucocorticoids
    - Innermost layer: Zona reticularis (3rd year) → Sex hormones
  - Medulla (ectoderm: neural crest)
    - Medulla: secrete 20% nor epinephrine, and 80% epinephrine
- Ectopic tissues: spread in the neck & torso (chest, abdomen & pelvis). That's why pheochromocytoma maybe thoracic in origin.

The adrenals start producing hormones during childhood but in very low levels > not enough to differentiate b/w male and female, but between the ages of 10-12 it starts functioning, finally at the age of 13 differences appear.

1.3 ANATOMY

- The adrenals cannot be palpated normally; if it was palpable it's most likely cancer.
- What is clipped in surgeries (adrenalectomy) is the veins! Any tear may cause severe hemorrhage..
- The adrenal vein returns the blood from the medullary venous plexus and receives branch from the cortex. It emerges from the hilum and on the right side and opens into the inferior vena cava, and on the left side into the renal vein.
- That's why the right side is shorter (more prone to bleed), while the left side is longer.
- When sampling the right adrenal we take from Right adrenal vein and for left adrenal we take from Left renal vein.
- Each gland has 3 arteries and one vein. Arteries comes from:
  - Inferior phrenic > superior suprarenal artery
  - Abdominal aorta > middle suprarenal artery
  - Renal artery > inferior suprarenal artery

The adrenals are located deeply (retroperitoneal organs), which makes it difficult to remove them.
One approach: from the back below 12th rib, enter fascia surrounding adrenal gland > not applicable to all adrenals e.g. big adrenals

1.4 PHYSIOLOGY

The adrenal glands secrete different hormones base on the layer that is secreting.

- Adrenal cortex
  - Aldosterone
  - Cortisol
  - Sex steroids
- Adrenal medulla:
  - Noradrenaline (20%)
  - Adrenaline (80%)

📄 The precursor for all these hormones is cholesterol and in each layer there is an enzyme converting it to the appropriate hormone & there is a limiting step > once a disease affects it, it leads to over-secretion in one way and problem in the other way.

1.5 ADRENAL IMAGING

- **CT scan**ⓘ: to differentiate between benign and malignant📄
  - Benign
    - Intensity, texture similar to liver
    - Low attenuation
    - Homogeneous (one color, there is no hypo & hyper density at the same time)
    - Smooth border
    - Smooth contour
    - < 4 cm in greatest dimension (the first criteria to be checked and then the others, size should be less than 4)
  - Malignant lesions:
    - High attenuation (>30 HU) [hyper-vascular – more white]
    - Heterogeneous
    - Irregular borders
    - Local/ vascular invasion
    - Lymphadenopathy
    - Metastases.
    - Large size (>6cm)) (black size 6cm-12cm →malignant .. remove it black size 2cm - 4cm → cyst)
- **MRI**. (CT still gold standard but if MRI is available, it'll give a better picture)
- **Nuclear scan**. (If you suspect pheochromocytoma - to look at the function of the mass > see uptake of gland)
- **PET scan**. (If you are looking for cancer > hot spots)

ⓘ CT scan is the gold standard

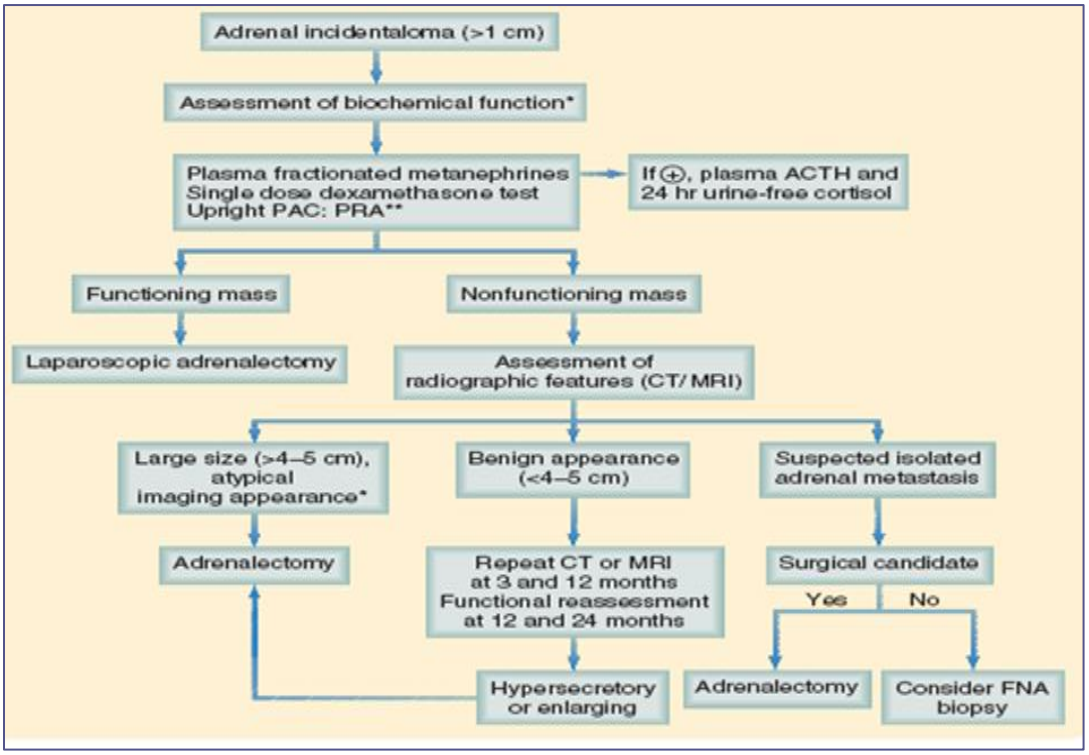
📄 Tissue biopsy will give you a definite diagnosis (if it's a benign or malignant mass) - but CT can give you an indication

2 ADRENAL DISEAES

2.1 INCIDENTALOMA (MOST COMMON)

- Found in 1-4 % of CT scans
- Incidence increases with age
- Causes:
  - **Small nonfunctioning adrenal tumors**: MOST COMMON (>80%)
  - Some with subclinical secretions of hormones (usually missed by the patient and physician)
  - Subclinical Cushing 5%
  - Pheochromocytoma>if functioning → interfere regardless of size 5%
  - Adrenocortical cancer 5%
  - Metastatic carcinoma 2%
  - Conn's

2.1.1 CLINICAL PATHWAY



PAC: Plasma aldosterone concentration
PRA: Plasma renin activity
PAC: PRA for Conn's. Metanephrine: look for pheochromocytoma. Single dose dexamethasone: for Cushing's.

- If functioning mass → Surgery.
- If non-functioning mass → CT scan
- CT may show:
- Benign appearance (< 4 cm) you repeat the scan after 3-12 months.
- If it secretes or has a malignant appearance we perform surgery.
- Handled based on the Size and function.

2.2 HYPERALDOSTIRONISM (CONN'S)

2.2.1 CAUSES:

1. Primary hypertension + metabolic alkalosis + with or without hypokalemia → primary hyperaldosteronism
    a. Adenoma (Most common)
    b. Idiopathic bilateral adrenal hyperplasia.
    c. Unilateral adrenal hyperplasia.
    d. Adrenocortical carcinoma.
    e. Familial (rare)
2. Secondary to any decrease in renal perfusion → Causes secondary hyperaldosteronism
    a. Renal artery stenosis
    b. CHF (congestive heart failure)

c. Liver cirrhosis
d. Pregnancy
e. Primary hyperaldosteronism

High aldosterone

- Na and water retention + K+ loss → ECF volume expansion & HTN
- Hypokalemia → myopathy; muscle weakness
- Acid excretion (H+ secretion from tubules) → metabolic alkalosis
- **Renin angiotensin system controls aldosterone secretion:**
  - Renal stenosis → ↓ blood flow to kidney → juxtaglomerular apparatus senses decreased flow (decreased volume) → retain Na+ and water → ECF expansion

∴ Anything that causes a decrease in renal perfusion can cause secondary hyperaldosteronism

2.2.1.1 PRIMARY HYPERALDOSTERONISM

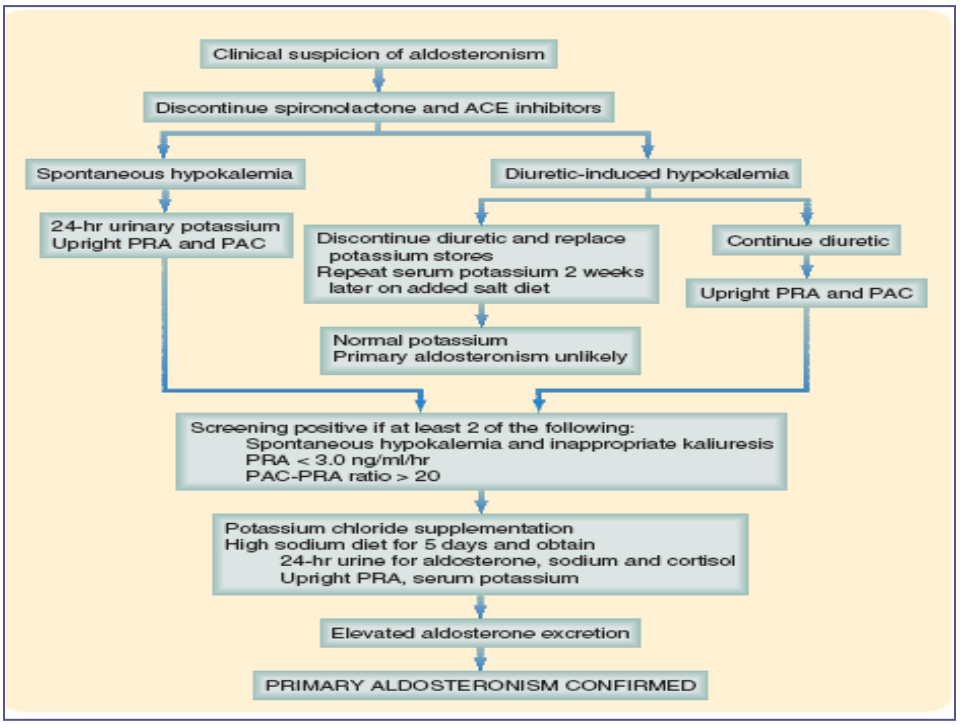
- Excessive production of aldosterone by the adrenal glands independent of any regulation by the renin-angiotensin system
- Age 30-50 years (middle age group)
- Female > male, 2:1 (females usually more prone to endocrine diseases and cancers)
- Prevalence 5-13%
- **Clinical features**: "patients will mostly present with fatigue (weakness b/c of ↓K+) & persistent HTN (uncontrolled, even w/3-4 medications)"
  - HTN (hypertension) with or without hypokalemia ⓘ
  - Weakness, polyuria, parasthesias, tetany, cramps
  - Metabolic alkalosis, relative hypernatremia
  - Elevated aldosterone secretion and suppressed plasma renin activity (b\c of aldosterone hypersecretion)
- **Diagnosis of 1ry hyperaldosteronism**
  a. Screening tests:
     i. PAC (ng/dl) / PRA (ng /ml) >20 (ordered by the endocrinologist not the surgeon)
     ii. Plasma aldosterone > 15 ng/dl (could mean it's Conn's)
  b. Confirmatory tests:
     i. Sodium suppression test (to differentiate b\w primary & secondary)
        1. In primary secretion is not affected ≠ in secondary secretion is suppressed
     ii. Urinary aldosterone excretion >14 ug/ 24hr

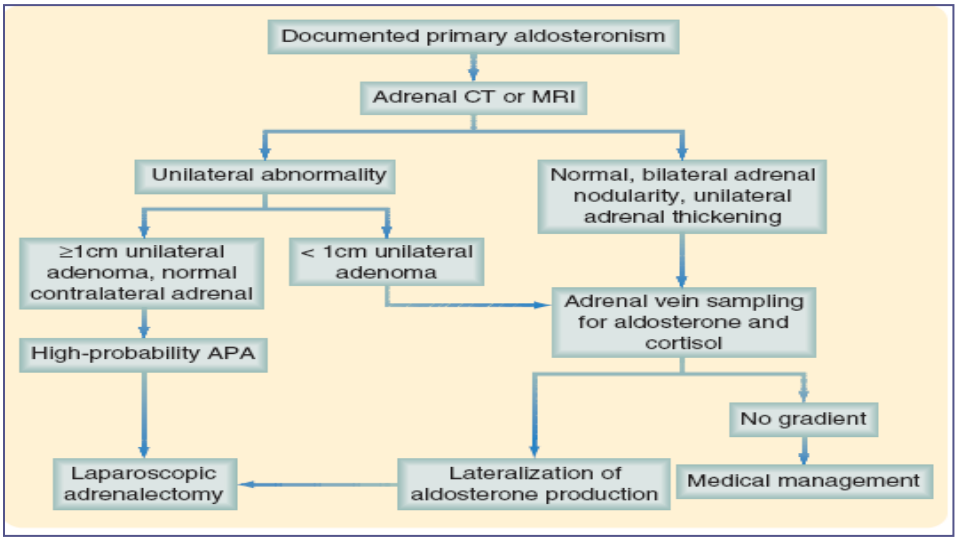
2.2.2 TREATMENT AND MANAGEMENT

1. Pre-operative preparation:
   a. Spironolactone: competitive aldosterone antagonist (corrects pathology caused by aldosterone by binding to its receptors)
      i. Promotes K retention (K+ sparing diuretic)
      ii. Reduces extracellular volume
      iii. Reactivates the renin-angiotensin-aldosterone system
   b. Amiloride: not as common as spironolactone
   c. Other K+ sparing diuretics
2. Surgery:

   a. Laparoscopic adrenalectomy
   b. Open surgery
3. Medical treatment (if surgery is not possible) due to:
   a. Unfit patients.
   b. Bilateral gland pathology. (Bilateral adenoma or hyperplasia)
4. Prognosis
   a. 1/3 persistent hypertension: patients should know that 1/3 of patient won't be cured from hypertension
   b. K levels will be restored (Normal daily activity is restored).
      i. Usually after treatment they get back to their normal lives with no complaints of HTN

2.2.3 CLINICAL PATHWAY





- Some medication induce aldosteronism → stop them and then start screening and confirmatory tests
- If you confirm hyperaldosteronism → do CT
- Venous sampling: catheters all the way to IVC from right & left side → see the
- gradient
- Lateralization (hormones higher in right than left) → this is the diseased gland → right adrenalectomy
- If no gradient → give medical treatment b\c you can't take out both adrenals, otherwise you have to replace adrenal hormones i.e. glucocorticoids and mineralocorticoids

2.3 PHEOCHROMOCYTOMA

"When there is a patient with pheochromocytoma in the ward everyone is ready; the physician, surgeon, anesthetist and ICU physician- to arrange an ICU bed, administer α- & β-blockers, and prepare the OR. Why? B/c they might lose him if BP shoots up >bleeding > death."

2.3.1 EPIDEMIOLOGY

If you suspect pheochromocytoma, DO NOT treat it as outpatient. The patient could die in crisis.

- Less than < 0.1% of patients with hypertension
- Not common in our community
- 5% of tumors discovered incidentally on CT scan (less than 4-5 cm but functioning)
- Most occur sporadically (no genetic predisposition)
- Associated with familial syndromes, such as:
  - Multiple endocrine neoplasia type2 (MEN2A) 40%
  - MEN 2B
  - Reckling hausen disease (Neurofibromatosis type I)
  - VonHippel-Lindau disease
- 90% of patients with pheochromocytoma are hypertensive (Once you diagnose the patient for the first time with HTN you HAVE to perform an US to the abdomen)
  - Hypertension is less common in children
- In children, 50% of patients have multiple (bilateral) or extra-adrenal tumors

2.3.2 SIGNS & SYMPTOMS

Clinical findings are variable

- Episodic or sustained hypertension
- Triad of **palpitation, headache, and diaphoresis**ⓘ
- Anxiety, tremors and weight loss
- Dizziness, nausea, and vomiting
- Abdominal discomfort, constipation, diarrhea
- Visual blurring
- Tachycardia, postural hypotension
- Hypertensive retinopathy (in short period of HTN history, few years only!)

2.3.3 ESSENTIAL FEATURES

- Episodic **HEADACHE** (due to HTN), excessive **SWEATING**, **PALPITATIONS**, and **VISUAL BLURRING**
- **HYPERTENSION**, frequently sustained, with or without paroxysms ⓘ

- Postural tachycardia and hypotension
- Elevated urinary catecholamines or their metabolites, hyper-metabolism, hyperglycemia
- **Rule of 10s**: ⓘ
  - 10% malignant
  - 10% familial
  - 10% bilateral
  - 10% multiple tumors
  - 10% extra-adrenal ("all places including head and neck" but the commonest is the abdomen, that's why we have to U/S the abdomen)

2.3.4 EXTRA ADRENAL PHEOCHROMOCYTOMA

Very RARE

- Abdomen (75%) [Closest to normal site so it is the most common]
- Bladder (10%)
- Chest (10%)
- Pelvis (2%)
- Head and neck (3%)

2.3.5 WORK UP

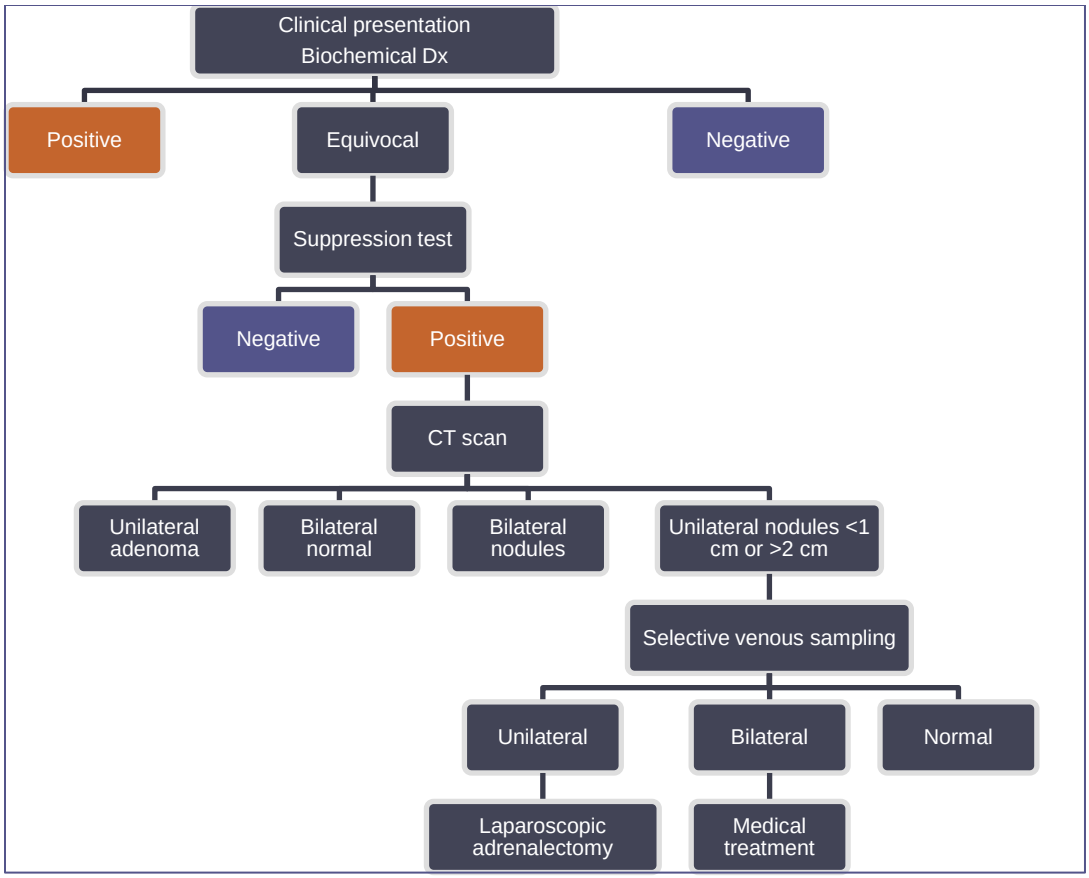
- History and physical exam
- Suspect pheochromocytoma based on symptoms
- CT, MRI, or other scans
- Plasma and urine studies (metanephrines, catecholamines, VMA)
- Begin treatment with a-blockers
- Possible MIBG scan
- Operative excision of tumor

2.3.5.1 LAB FINDINGS

- Hyperglycemia "and they cannot maintain fasting; they become hypoglycemic – we can differentiate it from Cushing"
- Elevated plasma metanephrines
- Elevated 24-hour urine metanephrines and free catecholamines
- **Elevated urinary vanillylmandelic acid (VMA)** ⓘ
- Elevated plasma catecholamines

2.3.5.2 IMAGING

- Adrenal mass seen on CT or MRI
- Characteristic bright appearance on T2-weighted MRI
- Asymmetric uptake on MIBG nuclear scan
  - Particularly useful for extra-adrenal, multiple, or malignant pheochromocytoma
  - Not useful for sporadic biochemical syndrome with unilateral mass



2.3.6 CONSIDERATIONS

- **Avoid arteriography** or fine-needle aspiration as they can precipitate a hypertensive crisis
- Early recognition during **pregnancy** is important because if left untreated, half of fetuses and nearly half of the mothers will die.
- Patients with pheochromocytoma usually die of high blood pressure, as the adrenal gland itself is very sensitive if it were to be touched in surgery there will be a surge of secretions, **which leads to a severe increase in blood pressure leading to a BP of 250 leading to intracranial hemorrhage!**

RULE OUT:

- Other causes of hypertension
- Hyperthyroidism
- Anxiety disorder
- Carcinoid syndrome

2.3.7 TREATMENT

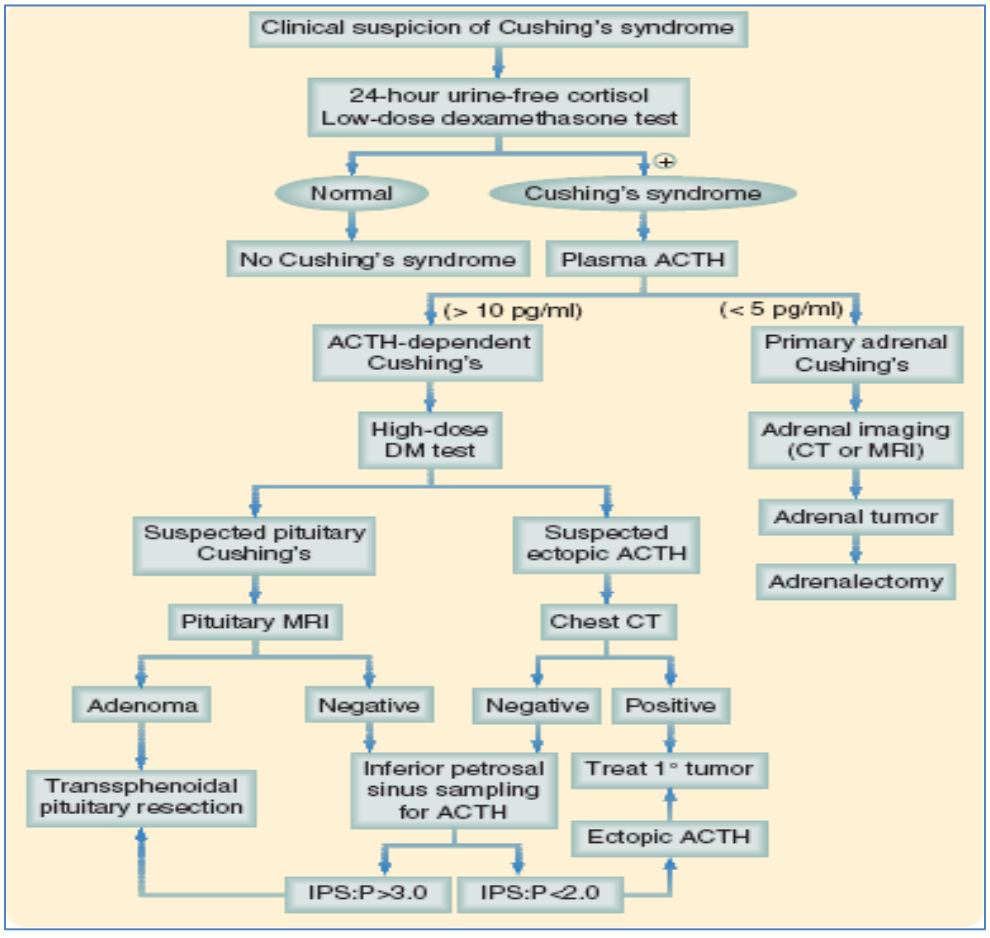
- **All patients with suspected Pheochromocytoma should be ADMITTED and treated as an in-patient.**
- HYPERTENSIVE CRISIS (can develop multisystem organ failure, mimicking severe sepsis)
- Some patients can present w/septic shock, if you can't explain it → suspect pheochromocytoma

- **Medical**:
  - α-Adrenergic blocking agents should be started (ATLEAST 2 weeks before the surgery) as soon as the biochemical diagnosis is established to restore blood volume, **to prevent a severe crisis, and to allow recovery from the cardiomyopathy**.
  - *Good alpha and beta-blocker control → smooth anesthesia, smooth surgery, and smooth recovery after.*
- **Surgical**:
  - Indications: **ALL** pheochromocytoma should be excised
  - Contraindications to surgery:
    - Metastatic disease (because we cannot control it)
    - Inadequate medical preparation (α-blockage) without proper α-blockade surgery is contraindicated.

2.4 CUSHING'S

- Disease vs. Syndrome?
  - Cushing's disease: secondary to pituitary adenoma
  - Cushing's syndrome: secondary to anything else

2.4.1 CLINICAL PATHWAY



- If we suspect Cushing first we do simple **low dose** (screening test) → suppression = normal patient
- If "+ve" (there is no suppression) = Cushing's
- Next step: ACTH dependent or not?
- In **high dose** test: if not suppressed it's either ectopic e.g. pulmonary carcinoma **OR** adrenal tumor (difference is in ACTH level: undetectable in adrenal & very high in ectopic), if pituitary it will be suppressed by high but not low dose dexamethasone

  "All will be suppressed by high dose but there is a difference in the reading – so we do CT chest (for ectopic) & MRI for brain (Cushing's disease) to confirm the diagnosis"

2.5 ADRENOCORTICAL CARCINOMA

- **Essential features:**
  - Variety of clinical symptoms through excess production of adrenal hormones
  - Complete surgical removal of the primary lesion and any respectable metastatic sites has been the mainstay of treatment.
- **Epidemiology:**
  - These tumors are rare; 1—2 cases per million persons in the US
  - Less than 0.05% of newly diagnosed cancers per year
  - Bimodal occurrence "in the very young and the very old", with tumors developing in children< 5 years of age and in adults in their fifth through seventh decade of life
  - **Male** to female ratio is 2:1, with functional tumors being more common in women
  - **Left** adrenal involved slightly more often than the right (53% vs. 47%); bilateral tumors are rare (2%)
  - 50—60% of patients have symptoms related to hypersecretion of hormones (most commonly Cushing's syndrome and virilization)
  - Feminizing and purely aldosterone-secreting carcinomas are rare
  - **50% of patients have metastases** at the time of diagnosis
  - It is very difficult to diagnose, because the symptoms appear when it is too late (*just like pancreatic cancer*)
- **Signs & symptoms:**
  - Symptoms of specific hormone excess (cortisol excess, virilization, feminization)
  - Palpable abdominal mass "b\c the mass very big"
  - Abdominal pain
  - Fatigue, weight loss, fever, hematuria
- **Lab findings:**
  - All laboratory abnormalities depend on hormonal status of tumor
  - Elevated urinary free cortisol or steroid precursors
  - Loss of normal circadian rhythm for serum cortisol
  - Low serum adrenocorticotropic hormone (ACTH)
  - Abnormal dexamethasone suppression test
  - Elevated serum testosterone, estradiol, or aldosterone levels
- **Imaging:**
  - Evaluation of adrenal glands with CT or MRI (adrenocortical carcinomas are typically iso-dense to liver on T1-weighted MRI, and hyper-dense relative to liver on T2-weighted MRI images)

- MRI more accurately gauges the extent of any intracaval tumor thrombus.

- **Considerations**
  - Mean diameter of adrenal carcinoma at diagnosis is 12 cm (black size 6cm to 12cm → malignant .. remove it , black size 2cm to 4cm → cyst)
  - Radiographic evaluation of suspected metastatic sites for purposes of staging should be undertaken prior to thought of any surgery.
  - We should rule out: Pheochromocytoma ⓘ

Right sided surgeries are always more difficult than left sided ones. Right tumors go to the IVC creating big thrombi and that makes the surgery more difficult. (restrict part of IVC→ remove the thrombus → anastomose it again)

All tissues involved should be removed, such as part of kidney or part of liver or maybe lymph nodes.

3 SUMMARY

Remember:

- Any **functioning** tumor or disease should be removed unless the patient is unfit or it is bilateral go with medical treatment.
- Any nonfunctional, small in size we follow up.
- The gold standard for adrenalectomy is **laparoscopy**. ⓘ

4 OPERATIVE APPROACHES

- In surgeries they clip “ligate” the adrenal vein not the artery, they are usually very small branches and very fragile
- We remove splenorenal ligament then push kidney away to remove adenoma alone.

Adenomas are usually brown in color: We open the splenorenal ligament then push the liver, pancreas, or stomach to the right side and isolate left kidney with the adrenal and clip “ligate” the adrenal vein - we do not bother with the artery; usually very small branches.

5 MCQS

1. A 30-year-old primigravida complains of headaches, restlessness, sweating, and tachycardia. She is 18 wk pregnant and her blood pressure is 200/120 mm Hg. Appropriate workup might include:
   a) Exploratory laparotomy
   b) Mesenteric angiography
   c) Head CT scan
   d) Abdominal CT scan
   e) Abdominal ultrasonogram
2. The most likely diagnosis in a patient with hypertension, hypokalemia, and a 7-cm suprarenal mass is:
   a) Hypernephroma
   b) Cushing’s disease
   c) Adrenocortical carcinoma
   d) Pheochromocytoma
   e) Carcinoid

In laparoscopy we use 5 ports:
5 ports .5-1 cm
2 for the surgeon
One for camera
2 for assistance

🗝 Answer Key: 1;E , 2;C

VENOUS DISEASE

1 INTRODUCTION

1.1 ANATOMY

- Venous blood flow of the Lower Limb is divided into 3 components: the superficial, communicating, and deep veins. (Figure 3,4)
- The superficial system comprises both the greater and lesser Saphenous veins and their tributaries. (Figure1,2)
- The superficial venous system is connected to the deep venous system through smaller communicating or perforator veins. (Figure 3)
- Veins have valves (varying in number). Their job is to prevent blood from refluxing. (figure 5,6)

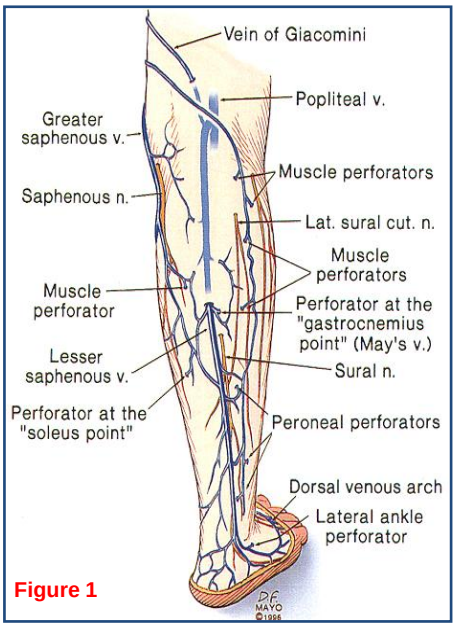


Figure 1

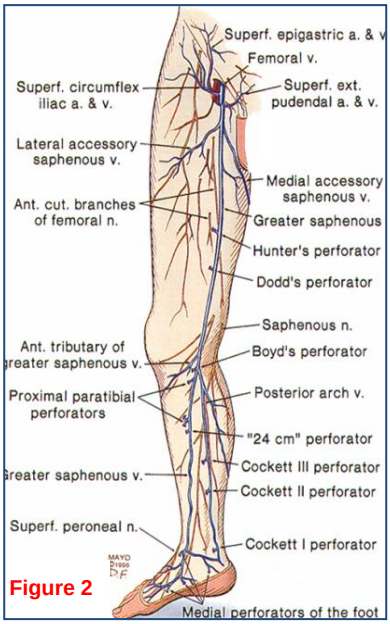


Figure 2

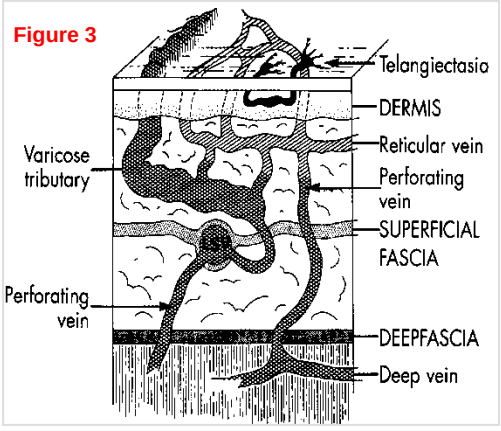


Figure 3

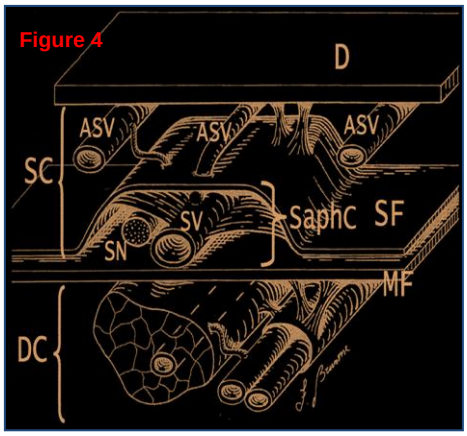


Figure 4

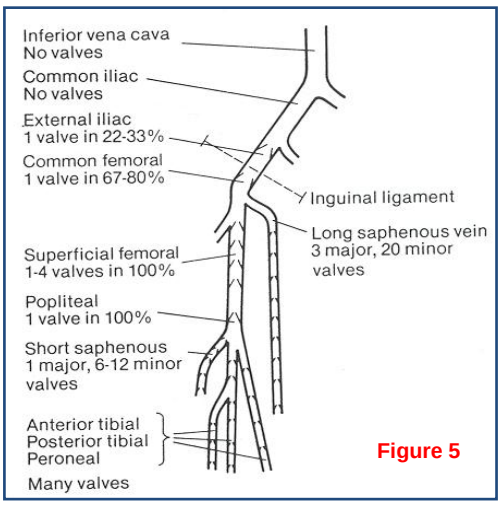


Figure 5

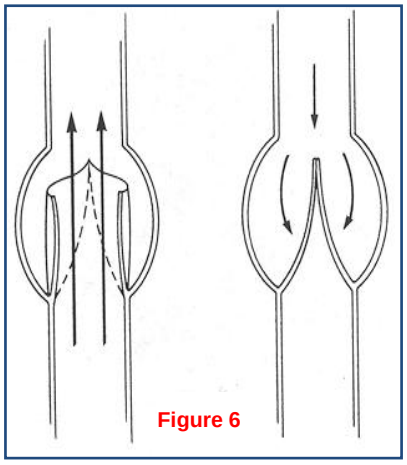


Figure 6

1.2 PHYSIOLOGY

1.2.1 THE FLOW OF THE VENOUS

Depends on many factors:

- Hydrostatic pressure (figure 9)
  - The hydrostatic pressure is (+)ve in LL and (–)ve in UL compared to the heart.
  - total pressure of the LL is higher That's why problems mainly will be in lower limb
- Alternating pressures of the chest and abdomen during breathing: (figure 8)
  - Chest is always negative in pressure.
  - But abdomen:
    - In expiration diaphragm will go up creating a (–) ve pressure, which will lead to blood sucking to heart, and subsequent blood sucking from the legs.
    - On inspiration diaphragm will go down, creating a (+)ve pressure which will lead to closure of veins.
    - That cycle will continue and form a valve like function.
- Calf muscle pump: (figure 7)
  - When calf muscles are at rest, deep veins expand and blood is drawn in from the superficial veins.
  - With calf-muscle contraction, blood is forced up the deep veins (opening the valves) towards the heart.
  - **Movement of blood (Normal):**
    - From superficial to deep.
    - From down to up.

Figure 7

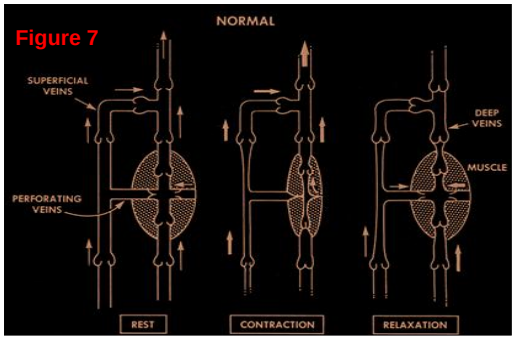


Figure 8

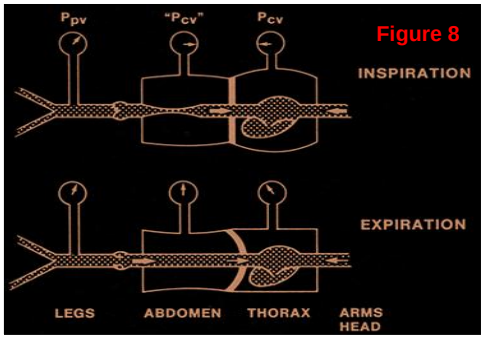
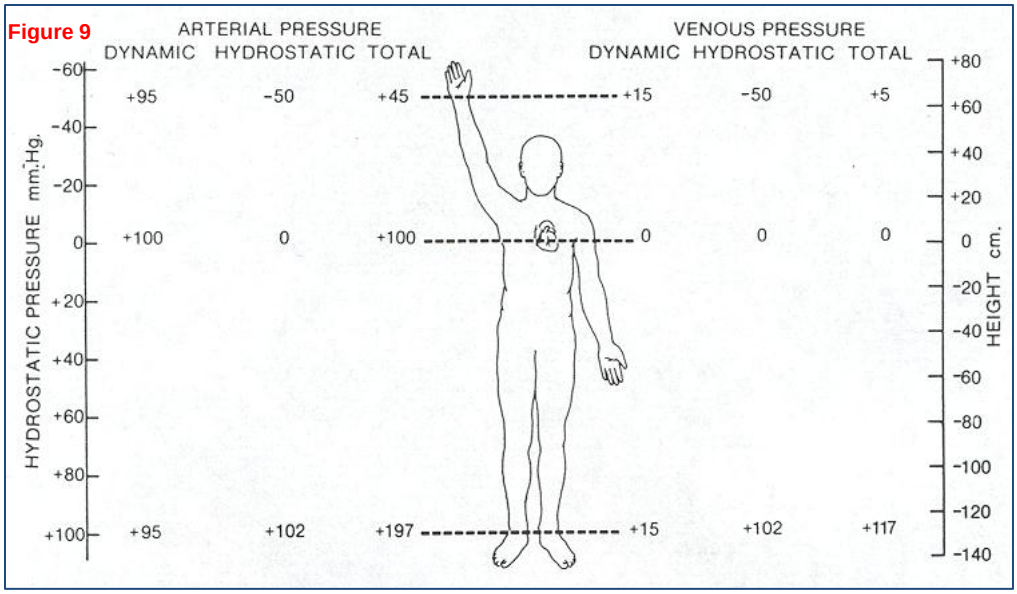


Figure 9



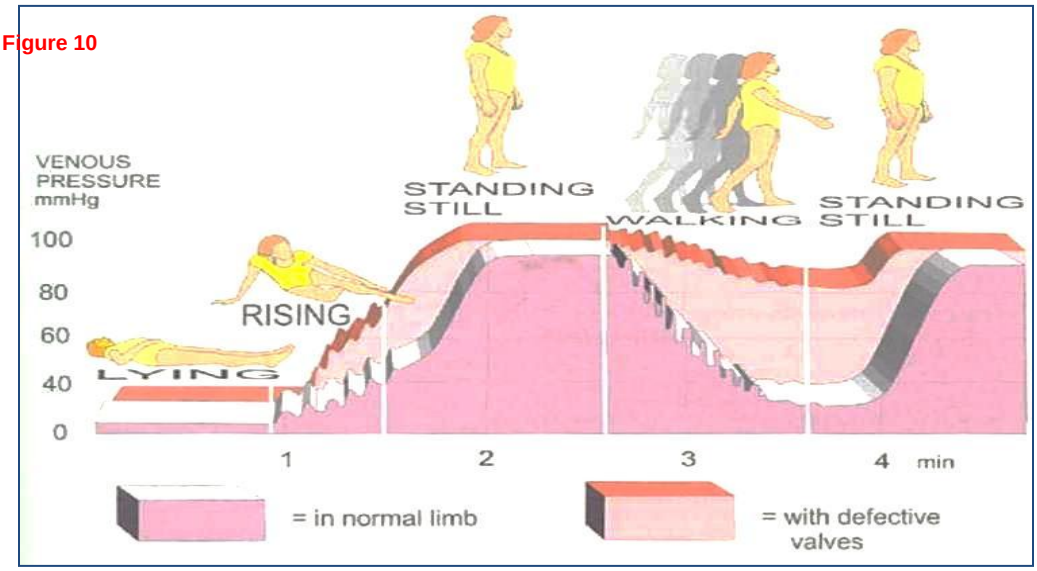
1.2.2 AMBULATORY VENOUS PRESSURE:

- NORMALLY: (figure 10)
  - **When lying supine**: pressure in lower limbs is low (10 mmHg)
  - **When rising**: deep veins start pulling blood from superficial veins slowly→ so pressure starts to increase gradually.
  - **When standing**: deep veins continue to pull blood and pressure increases reaching (90 mmHg)
  - **When walking**: calf muscle pump starts working and pushes blood up the vein through the valves→ so pressure drops to (25 mmHg). Valves then close, to prevent the pressure from increasing again by preventing the blood from refluxing.
  - If you stop and stand still, calf pump stops and the deep veins start to pull blood from the superficial veins again, so pressure builds up again.
- IF VALVES DEFECTED;
  - When rising and standing: blood will reflux from the valve, so the pressure will increase **rapidly**.
  - When walking: blood will reflux through the valve and the pressure remains high.
- Values:
  - Supine and walking positions are not a problem (low pressure) standing is the problem (high pressure)

- Supine = 10 mmHg
- Standing= 90 mmHg
- Walking= 25 mmHg

- Methods of measurement: see below in investigations

Figure 10



2 CHRONIC VENOUS INSUFFICIENCY

- ***The presence of (irreversible) skin damage in the lower leg as a result of sustained venous* HYPERTENSION**

2.1 PATHOPHYSIOLOGY

- Primary valve incompetence (figures 11-13)
  - Defect in the valve itself "floppy valve" (fig. 11-13)
  - Congenital causes
- Secondary valve incompetence:
  - Reflux (90%)
  - Obstruction (10%)

In secondary incompetence the problem is away from the valve, for example:

*Obstruction in the pelvis (tumor) or a collateral vein bypassing the valve

*DVT

*Pregnancy

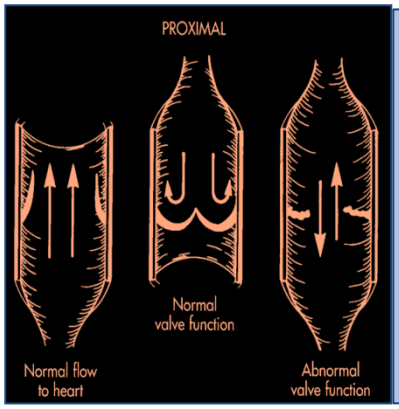


Figure 11

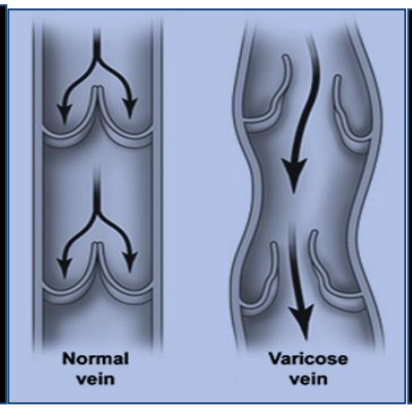


Figure 12

Figure 13

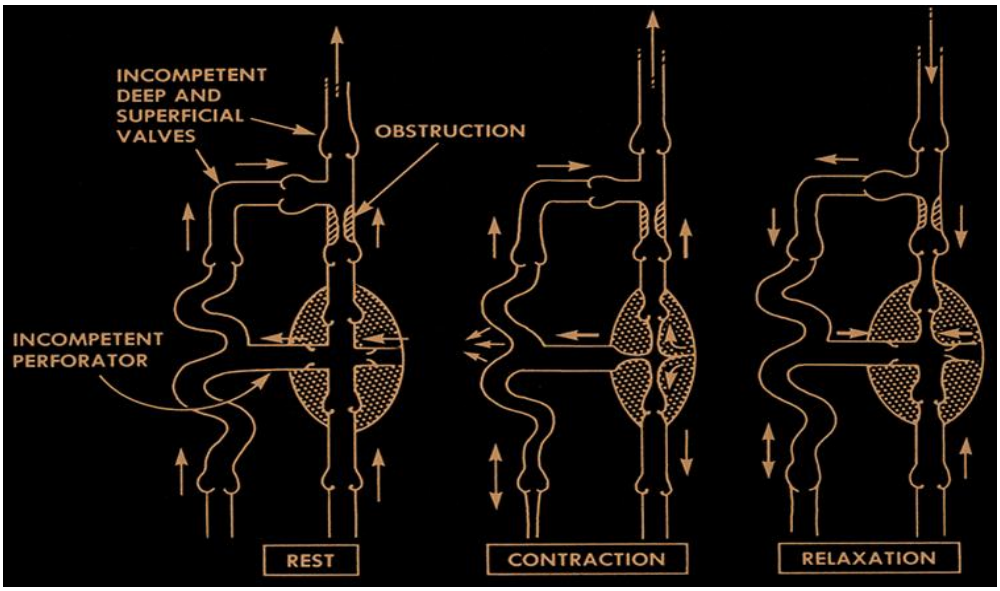


Figure 14: Note: the (down-to-up) and (out-to-in) mechanism is disturbed because of obstruction.

2.2 CLINICAL EVALUATION

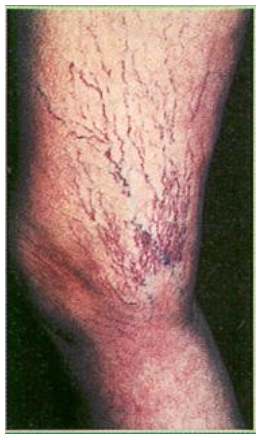
2.2.1 HISTORY

- Take a good history
- Ask about risk factors that can cause secondary causes (pregnancy, DVT, tumors, previous surgeries)
- Ask about skin changes in details

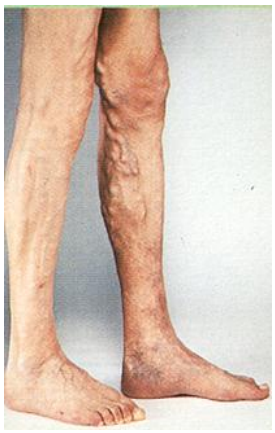
2.2.2 PHYSICAL EXAMINATION

- Make sure you thoroughly examine the Gaiter's Area for clinical classification!!
- Clinical manifestations (classification; C1-C6) of chronic venous insufficiency:

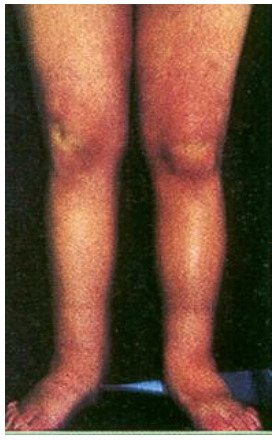
Figures 15-20



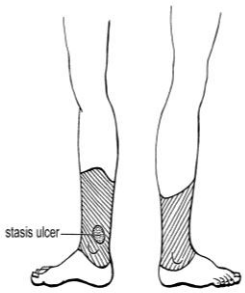
C1: Telangectasia/ Spider viens



C2: Varicose veins

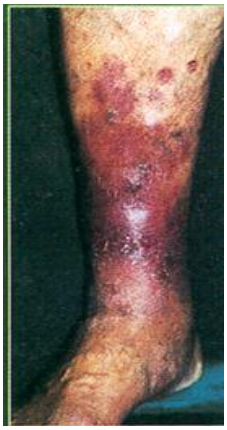


C3: Edema but no skin changes

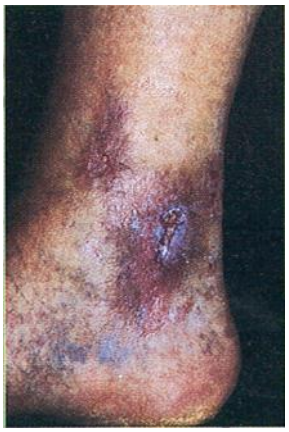


Gaiter's Area:

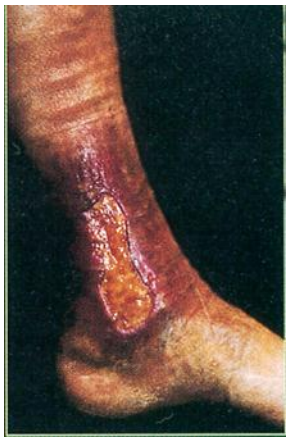
Area around medial and lateral malleolus is common site for venous ulceration because this area is skin on bone (no tissue and fat between them) so the blood and inflammatory stuff will go directly from the veins to skin leading to certain manifestation.



C4: Lipodermatosclerosis/ Pigmentation/ eczema



C5: Healed ulcer



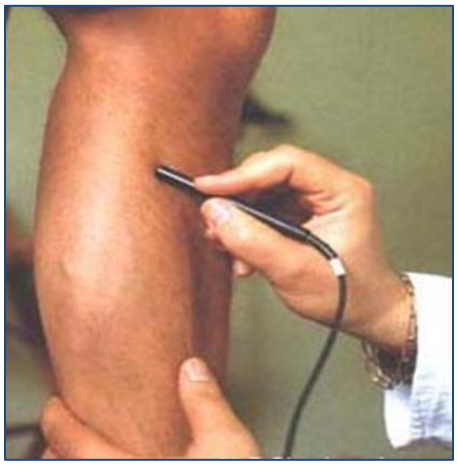
C6: Active ulcer

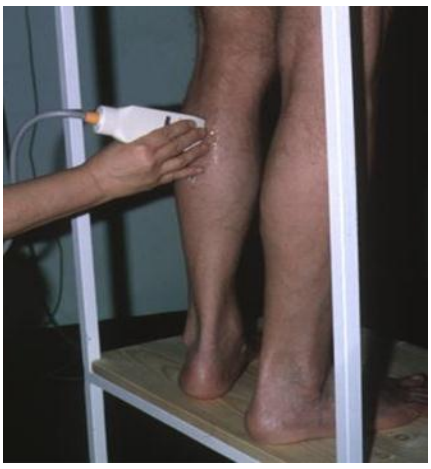
2.2.3 INVESTIGATIONS

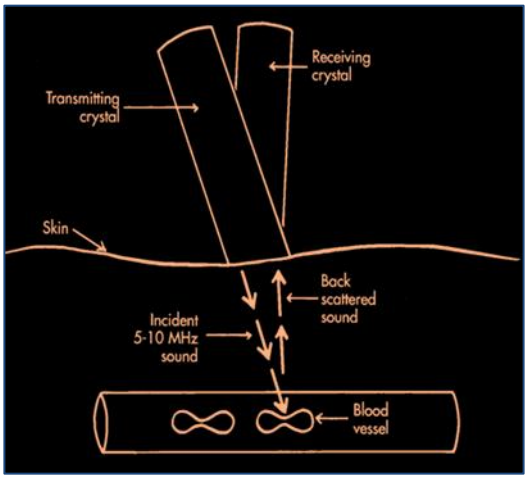
2.2.3.1 NON-INVASIVE

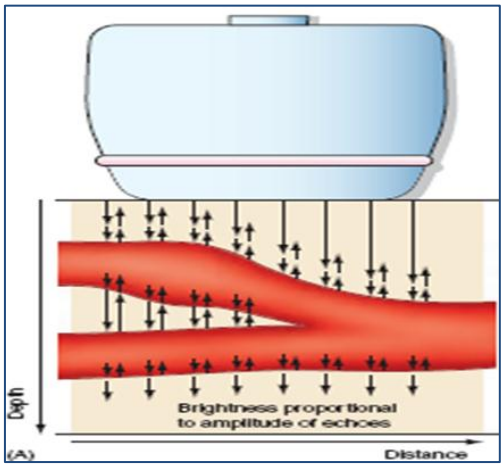
- Doppler (figure 21, 23)
  - Machine used to HEAR blood flow in the veins
  - You can detect which valve isn't working, or an obstruction, by listening for abnormalities in the sound of the flow.
- Duplex: (figure 22, 24-26)
  - This is a form of ultrasound machine that allows visualization of a portion of the venous system. It can determine the direction and speed of blood flow within the veins.
- So duplex has the same principle of Doppler but you can use it to visualize also
- Check the slides for more pictures

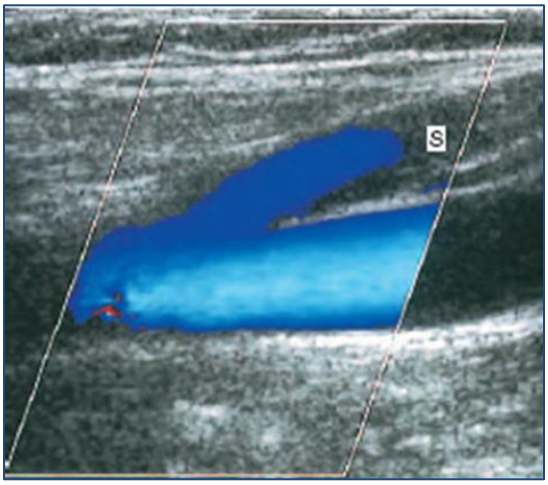
Figure 21-26

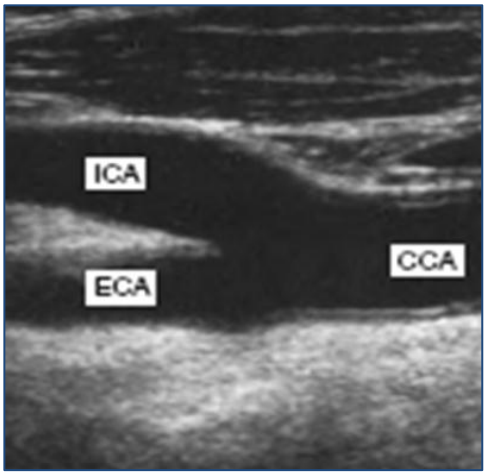












2.2.3.2 INVASIVE TESTING:

- AVP (ambulatory venous pressure) ⓘ
  - It is a test to measure the venous pressure in supine, standing, and walking positions. To compare it with normal changes.
  - Method:
    - It is performed by placing a small needle into one of the veins on the back of the foot and connecting the needle to a blood pressure measurement machine.
    - 20-21gauge Butterfly Needle
    - Superficial Dorsal Vein (Foot) or Ankle Vein
  - Normally:
    - when walking
      - A decrease in pressure from Pressure 80 - 90mm Hg to 20-30 mm Hg
      - Or a: > 50% drop
    - Then after standing still Venous Refilling Time: ≥20 seconds
  - Abnormal results if: ⓘ

- Lack of sufficient drop in pressure with ambulation
  - Pressure doesn't decrease enough on walking and the difference between the standing and walking pressure is <50%
  - This means that the pressure remains high in the veins although it is supposed to drop because of walking
- Short venous refill time
  - It takes less than 20 seconds
  - This means the blood is filling veins quickly and the valves aren't working efficiently to stop the blood from refluxing

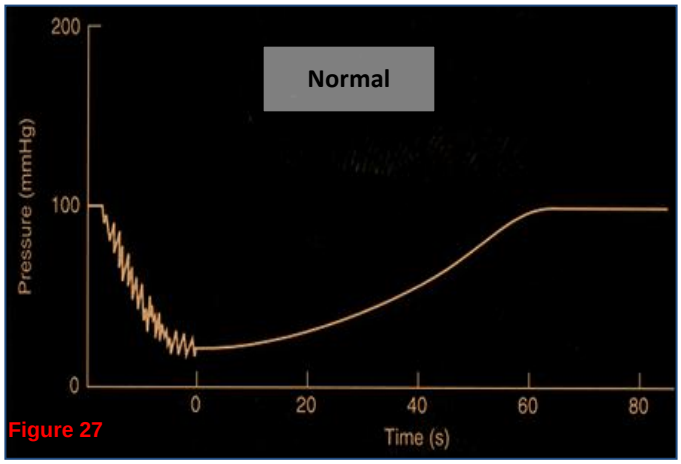


Figure 27

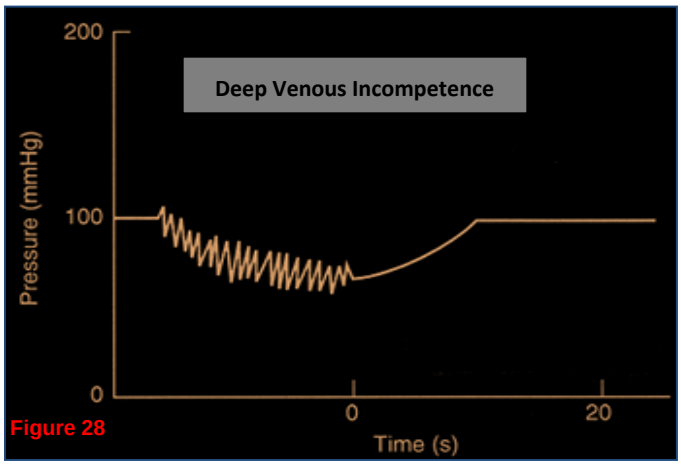
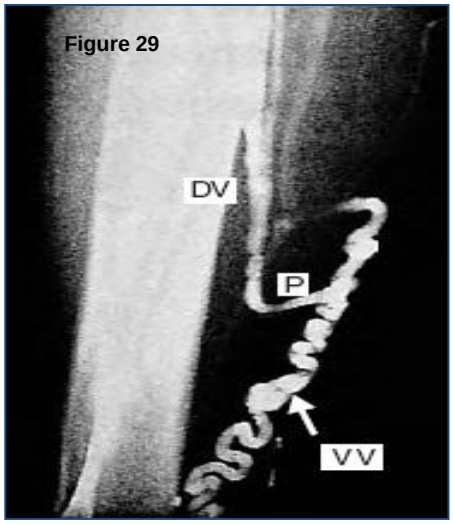
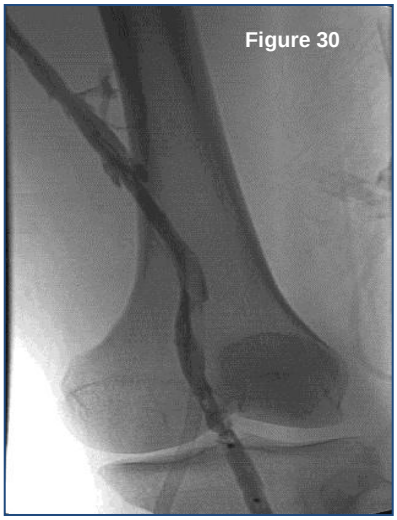


Figure 28

- Venography (phlebography): (figure 29, 30)
  - Contrast injected to visualize veins.
  - Not used much nowadays, due to its complications. But still has specific indications





3 TREATMENT

3.1 PRINCIPLES OF TREATMENT:

1. **Always exclude secondary causes by doing a thorough physical exam and history and investigations**
2. Restoration of blood pumping towards the heart
3. Remove the problematic vein (provided that there is another functioning vein draining the same area)

3.2 SOME METHODS OF TREATMENT:

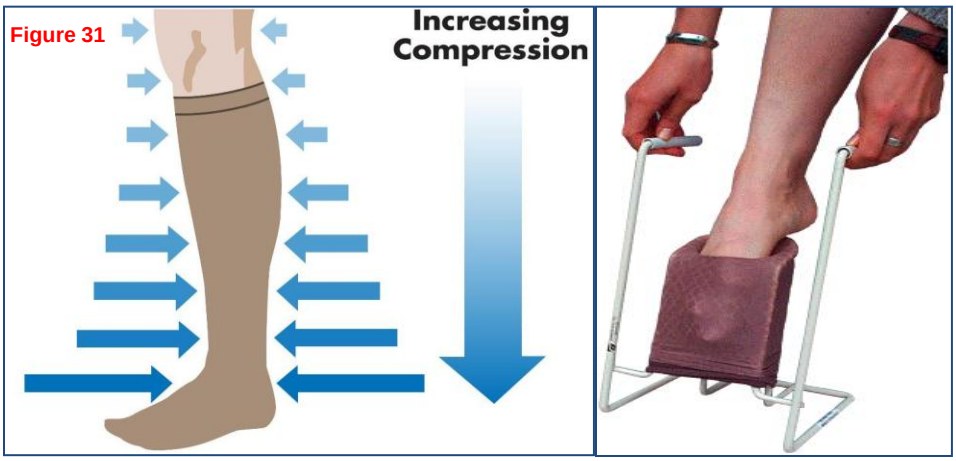
1. Stocking: (figure 31)
   - Physical principle applies pressure, is higher pressure down and lowers pressure up to make blood go up.
   - Problem is **low compliance**, although it usually solves the problem.
2. Ablate vein: (figure 33)
   - Chemically or thermally or laser
     - Denaturation of vein wall collagen → contraction → fibrous obliteration of the vein
   - Provided that there is another functioning vein draining the same area.
3. Sclerotherapy (figure 32)
   - Sclerotherapy is the injection of a sclerosing agent into a vein, causing an inflammatory reaction in the endothelium of the vein wall. The vein walls adhere together under compression and form a scar (fibrotic tissue) that is absorbed by the body. Remember this works only to the small veins not big ones.
4. Conventional surgery: problematic vein tied above and below, then taken out. Not used anymore

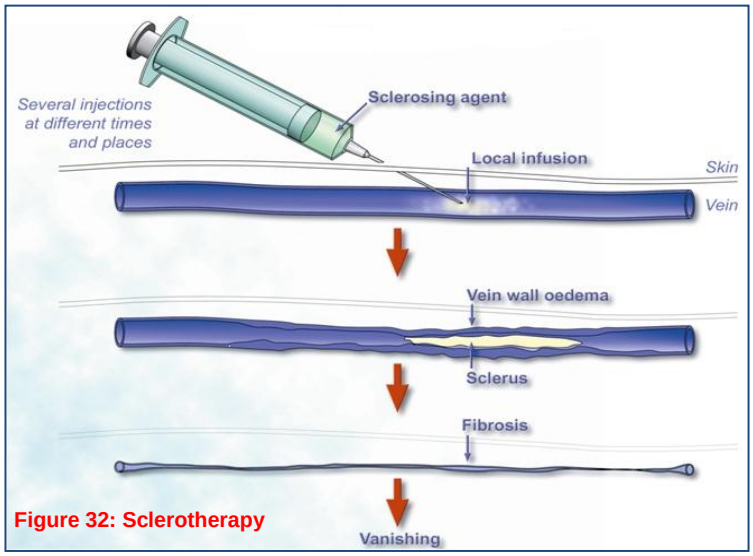
3.3 SPECIFIC MEASURES:

1. Telangiectasias and reticular veins: Stocking or Sclerotherapy
2. Varicose veins

- Stocking
- Sclerotherapy
- Endovenous laser therapy (EVLT)/ Surgery

3. C3 to C6:
   - Stocking/ Sclerotherapy/ EVLT/Surgery





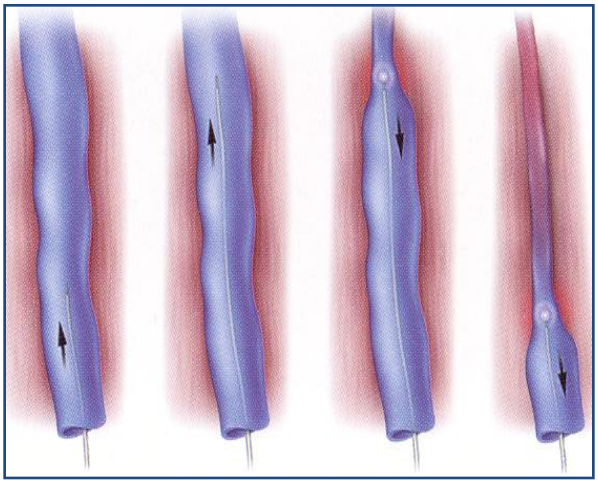


Figure 33: Endovenous Laser Therapy (ELT)

ATHEROSCLEROSIS

1 INTRODUCTION

- It is an inflammatory process that causes clogging, narrowing and hardening of the large and medium sized arteries.

1.1 RISK FACTORS

- Non modifiable
  - Male
  - Advanced age
  - Family history
- Modifiable
  - Major
    - Smoking
    - Hypertension
    - Diabetes
    - Hyperlipidemia
  - Minor
    - Homocystenemia
    - Obesity
    - Hypercoaguble states
    - Physical inactivity

1.2 PATHOGENESIS

- The key word in Atherosclerosis is inflammation
  - Fat deposits accumulate and will cause endothelial injury that will initiate the inflammatory process
  - Formation of fibrous plaque by platelets
  - Calcification of the arterial wall (this is the cause of atherosclerosis)
  - Fat by itself is NOT harmful but it is the rupture of the plaque
  - Rupture of the wall will cause clotting (atherothrombosis)

1.3 CLINICAL SPECTRUM OF ATHEROSCLEROSIS

- The message you should take is that Atherosclerosis is a systematic disease that affects the entire body not only specific areas. The spectrum includes:
  - Cerebrovascular accidents
  - Coronary artery disease
  - Renal artery disease
  - Visceral artery disease (mesenteric)
  - Peripheral artery disease (Aorto-iliac & upper and lower limb is a marker for atherosclerosis)
    - Intermittent claudications
    - Limb ischemia

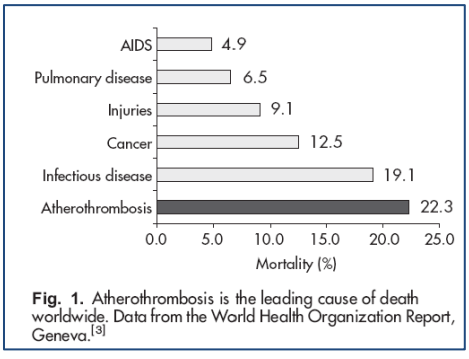


Fig. 1. Atherothrombosis is the leading cause of death worldwide. Data from the World Health Organization Report, Geneva.[3]

1.4 BURDEN OF ATHEROSCLEROSIS

- It is the number one killer worldwide and here in Saudi Arabia and it predicted to increase

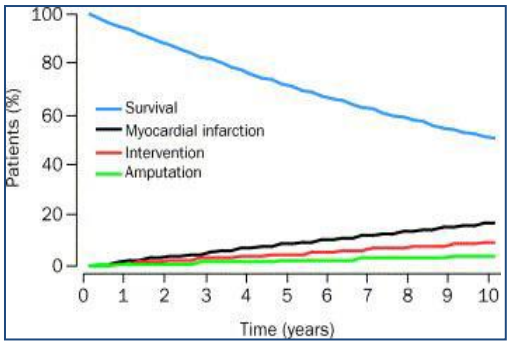
We always concentrate on the symptoms related to the organ itself and forget to think about the bigger picture. When we think about the peripheral artery disease we think only about the peripheral complication like claudications, amputation

In reality we have to think about the mortality about 7% per year

2 PERIPHERAL ARTERY DISEASE (PAD)

2.1 IMPORTANCE OF PAD

- PAD is a marker of systemic atherosclerosis
- Patients with either symptomatic or asymptomatic PAD generally have widespread arterial disease
- Patients with PAD have the following:
  - Coexisting disease:
    - 35-92% have coexisting Coronary artery disease (CAD)
    - 25-50% have coexisting Cerebrovascular disease (CVD)
  - Cause of death in PAD patients:
    - 40-60% die from CAD
    - 10-20% die from CVD



- Patients with PAD have a 6 fold (imp!) increased risk of cardiovascular disease mortality compared to patients without PAD even the patient with or without symptoms
- Natural history ⓘ
  - Annual mortality rate is 6.8%
  - Annual risk of Myocardial infarction is 2%
  - Annual risk of intervention is 1%
  - Amputation 0.4%

2.2 PRESENTATION

- Symptomatic ⓘ
  - Intermittent claudications
    - Pain at a lower limb group of muscles at exertion that is relieved by rest (Think of it like Angina)
  - Critical limb ischemia
    - This is a limb threatening condition
    - Pain at rest
    - Tissue loss (Ulcer)
    - Gangrene
- Asymptomatic ⓘ

The concept you should keep in your mind that atherosclerosis is one disease, one group of symptoms and one treatment. But different arterial trees are affected so patients will present with different symptoms.

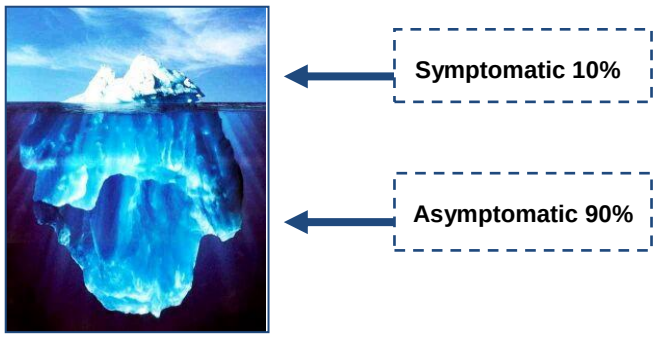
2.3 DIAGNOSIS

2.3.1 SYMPTOMATIC:

- History
- Physical examination
- Investigations ⓘ
  - They are primarily used for:
    - Confirming the diagnosis after a history and examination and exclude other diseases
    - Assess severity
  - ABI measurements
  - Non invasive tests:
    - Arterial duplex: Doppler + US. Good for anatomical involvment
    - CTA: CT + contrast
    - MRA
  - Invasive tests: the conventional angiogram (GOLD standard) ⓘ
    - Very accurate in mapping out the arteries but the Duplex is better in assessing dynamic views

2.3.2 ASYMPTOMATIC: ⓘ

- **SCREENING with ABI measurement**
- Around 90% of patients with PAD are asymptomatic but they also carry the same risk!!!! ⓘ
- We have to screen high risk groups even if they don't carry symptoms, for example:
  - Old age (>50), family history, male
- If you notice in the natural history part we mentioned that the mortality is 7% but the intervention level is 1%. And that is because 90% are asymptomatic!
- That is why we need screening to see past the tip of the iceberg and decrease the mortality and the cardiovascular mortality and morbidity associated with those pts.



2.3.3 ABI (ANKLE-BRAHCIAL INDEX)

- It is the index between the systolic pressure of the Ankle and the brachial systolic pressure
- ABI = (Highest ankle systolic pressure (PT or DP) / Highest brachial systolic pressure
  - PT= Posterior Tibial artery
  - DP= Dorsalis Pedis artery

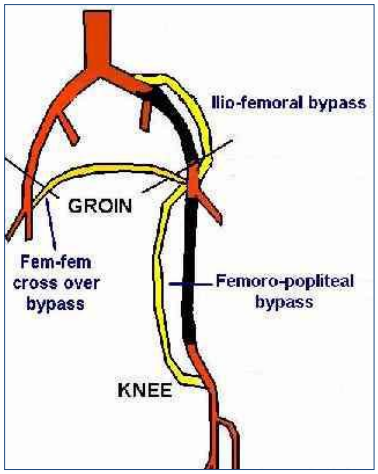
- Results ⓘ:
  - Normally it is >0.9
  - Abnormal if <0.9 (Abnormal results indicate PAD)
    - Mild 0.8 – 0.9
    - Moderate 0.5 – 0.8
    - Severe <0.5
    - Very severe <0.25
- The ABI has limited use in evaluation calcified vessels that are not compressible in Diabetics

2.4 TREATMENT OF PAD

2.4.1 GOALS OF TREATING PAD ⓘ:

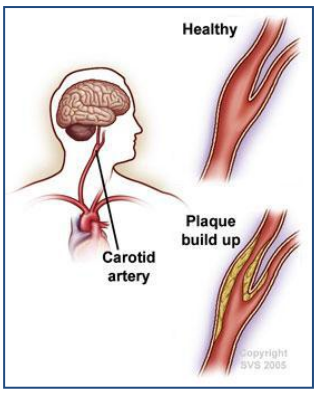
1. Relief of symptoms
2. Improving the quality of life
3. Limb salvage
4. Prolonging survival

2.4.2 STRATEGIES IN TREATING PATIENTS WITH PAD ⓘ:



- Risk factor modification (modifiable)
  - Diet and weight control
  - Exercise
  - Antiplatelets: to prevent thrombus
  - Hypertension control: <140/90 (130/80 for diabetics)
  - Diabetes control: HbA1c <6
  - Lipid control
  - Smoking cessation
- Improve limb circulation
  - Conservative:
    - exercise program that promotes angiogenesis and growth of collaterals around the ischemic area
  - Intervention: Revascularization
    - Angioplasty (+/- Stenting the artery)
    - Surgical bypass
- Last strategy in treating PAD:
  - Major amputation
    - Affects function: Whole leg amputation
    - Primary amputation (we start with amputation)
    - Secondary (we start with angioplasty or bypass but the patient does not respond)
  - Minor amputation: Doesn't affect function
    - BKA=Below knee amputation
    - AKA=Above knee amputation

3 CAROTID ARTERY DISEASE



- Stroke is the third leading cause of death and a principal cause of long term disability
- Prevention of stroke is MORE IMPORTANT than treatment

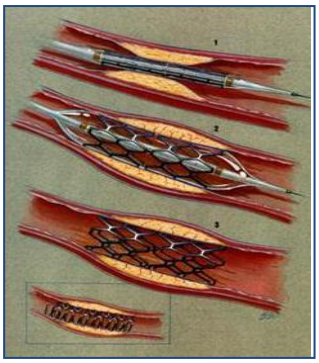
3.1 PRESENTATION

- Symptomatic:
  - Transient Ischemic Attacks (TIA): Loss of motor or sensory function for less than 24 hours
  - Amurosis Fugax: transient visual loss for less than 24 hours
  - Stroke
- Asymptomatic

3.2 DIAGNOSIS

- Symptomatic
  - History
  - Examination
  - Investigations
- Asymptomatic ⓘ
  - It is detected by hearing a carotid bruit. It is very important that you screen for a carotid bruit in all patients with risk factors or over 50.
  - Arterial duplex:
    - Note that stenosis is measured by velocity and not anatomical diameter

3.3 TREATMENT OF CAD



- Goals of treatment:
  - Prevention of strokes
  - Prolong survival
- Strategies in treating patients with CAD
  - Risk factor modification
    - Diet and weight control
    - Antiplatlets double
    - Exercise
    - Hypertension control
    - Diabetes control
    - Lipid control
    - Smoking Cessation
  - Improving brain circulation:
    - Revascularization with **Carotid Endarterectomy** (best method) and standard of care ⓘ
    - Angioplasty with or without Stenting
      - This intervention is currently under investigation
      - Indication:
        - Hostile neck
        - Hostile carotid disease
        - Part of a RCT

Stenting still needs more evidence but it is reserved for certain groups of patients

- Indications to intervene:
  - Symptomatic
    - >70% stenosis: NACET study shows decrease stroke at 2 years from 26% to 9%
    - 50-69% stenosis: Marginal benefit but greater for male
    - Recovered ischemic stroke patients
  - Asymptomatic
    - >60% stenosis: ACAS study shows decrease stroke at 4 years from 11% to 5%

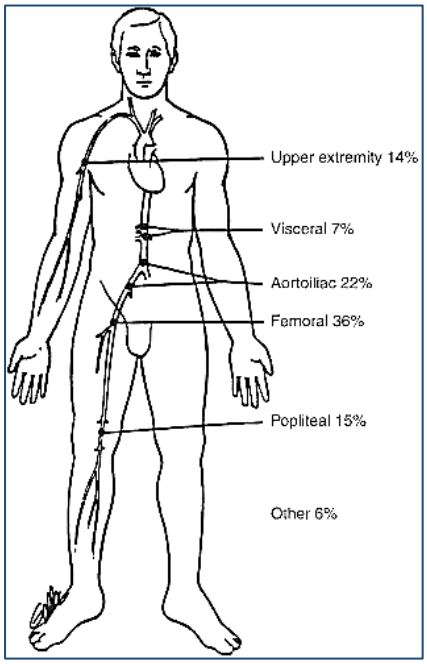
Hostile neck/carotid disease means a patients who already has undergone a endartectomy and surgery is difficult

4 ACUTE LIMB ISCHEMIA

- Sudden decrease or worsening in the limb perfusion causing a potential threat to the limb viability resulting from a sudden obstruction of the arterial system

4.1 CAUSES:

- Embolus (Most common cause) ⓘ
- Thrombosis
- Trauma
- Iatrogenic
- Arterial dissection



4.2 POSSIBLE SOURCES FOR AN EMBOLUS:

- Spontaneous 80%:
  - Cardiac source: most common cause ⓘ
    - Arrhythmias, MI, prosthetic valve, endocarditis
  - Non-Cardiac
    - Proximal plaque, aneurysm, paradoxical emboli
- Iatrogenic 20%
  - Angiographic manipulation
  - Surgical manipulation
- Common sites of embolus lodgement in the arterial tree:
  - Femoral is the most common ⓘ

4.3 PRESENTATION OF ACUTE LIMB ISCHEMIA

- Sudden onset of diffuse and poorly localized leg pain
- 6 Ps
  - Paresthesias
  - Pain
  - Poikilothermia
  - Pallor
  - Pulselessnes
  - Paralysis
- Investigations
  - Acute limb ischemia is a clinical diagnosis ⓘ

- If time allows especially if atherosclerotic thrombus is suggested, preoperative angiography is often wise

4.4 TREATMENT OF ACUTE LIMB ISCHEMIA ⓘ

- Goal of management: Rapid restoration of adequate arterial perfusion without the development of morbid local or systemic complications
- Preserving the limb but not on the expense of life
- EMERGENCY
  - Golden time is 6 hours from the appearance of symptoms ⓘ
  - ABC (most important step)
  - IV Heparin
  - Rapid surgical thromboembolectomy
    - +/- Surgical bypass
    - +/- Thrombolytic therapy
    - +/- Primary amputation

4.5 REPERFUSION INJURY

- It is a worrisome complication of revascularization
- Effects can be, local:
  - Compartment syndrome ⓘ
    - It is a condition where the pressure inside the compartment rises due to edema after the ischemic injury.
    - The raise in pressure will stop the blood flow to the area and cause more ischemia
    - Needs emergency fasciotomy ⓘ
- Systemic: ⓘⓘⓘ
  - Hyperkalemia:
    - Due to muscle ischemia and breakdown
    - Leads to cardiac arrest
    - Treated or prevented with Calcium Gluconate
  - Acidosis: Bicarbonates should be given
  - Myoglobinuria
    - Leads to acute renal injury
    - Patient should be given a lot of fluids

5 MCQS

1. All of the following are signs of critical limb ischemia except:
   A. Intermittent claudications
   B. Pulselessness
   C. Poikilothermia
   D. Paresthesiasis
2. All of the following can happen in reperfusion injury except:
   A. Acidosis
   B. Compartment syndrome
   C. Hyperkalemia
   D. Hyperglycemia

🗝 Answers: 1;A , 2;D

VASCULAR INVESTIGATIONS

1 INTRODUCTION

- Blood vessels are a series of tubes that are used to pump blood throughout the body.
- There are 3 types of blood vessels: arteries, veins and lymphatics.
- Arteries carry oxygen rich blood away from the heart to every part of the body, including the brain, kidneys, intestine, arms, legs and heart itself. When a disease occurs in the arteries, it's called arterial disease.
- Blood flows back to the heart from all parts of the body through veins. When disease occurs in the veins, it's called venous disease.
- Fluids return from the skin and other tissues to the veins through lymphatics.

1.1 VASCULAR DISEASES:

- Arterial diseases: such as aortic dissection which is caused by a tear in the inner layer of the aortic wall and then blood will flow between the layers and separate them or arterial occlusion. A lot of patients come with arterial occlusive diseases.
- Acute: ischemia
- Chronic: intermittent claudication or dilatation (arterial aneurysmal disease)...Etc.
- Venous diseases: deep vein thrombosis commonly referred to as “DVT”, occurs when a blood clot, or thrombus, develops in the large veins of the legs or pelvic area, or chronic venous insufficiency which is an all--inclusive term for vascular malformations, vascular tumors, and other congenital vascular defects. The more commonly used term, Chronic Venous Insufficiency (CVI), implies abnormally formed blood vessels that one is born with... etc.
- Lymphatic.

1.2 TYPES OF INVESTIGATIONS:

- Invasive (in vascular surgery invasive procedures are the gold standard)
- Noninvasive.

2 NON INVASIVE VASCULAR TESTS

- Utilizes instrument; Utilizes the sound energy
- Doppler Ultrasound.
- Sound –longitudinal mechanical wave of any frequency.
- Audible Sound range 20-20,000 cycles/sec ⓘ. 20Hz-20kHz
- Ultrasound-’Ultra’ means ‘Above’ human hearing - >20,000 cycle/sec(20kHz).
- Diagnostic Ultrasound –2MHz-12MHz (2million-12million cycle/sec) ⓘ

More Frequency > less penetration of the tissue e.g. for superficial structures.

-More frequency >better resolution.

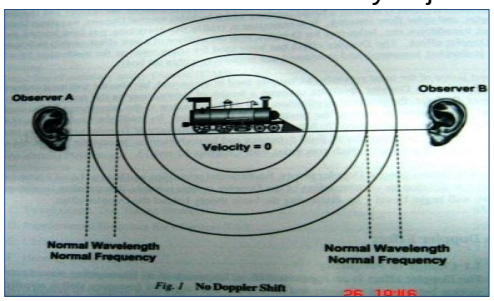
-Less frequency > deeper penetration of the tissue e.g. abdominal investigation,

-Less frequency > lesser resolution.

2.1.1 DOPPLER ULTRASOUND

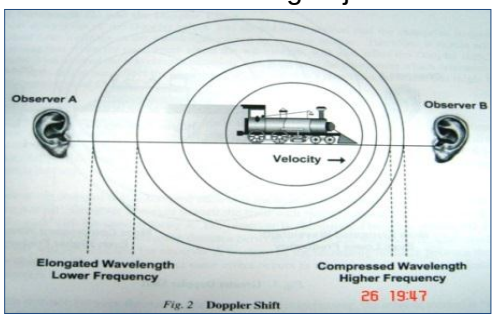
We transmit X frequency and receive Y frequency. These are utilized to know if blood is moving properly or no.

- Based on principle of Doppler effect/shift
- Normally blood vessels contain moving blood if there's a block it'll stop moving.
- Ultrasound interaction with stationary object:



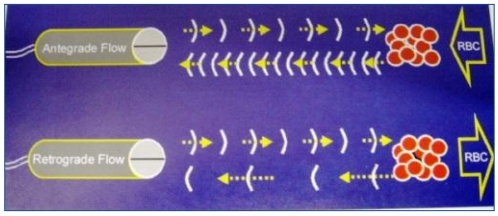
  a. No frequency change.
  b. No Doppler Effect or shift.
  c. Sound won't be heard.

- Ultrasound encounters moving object:



  a. Doppler Effect or Shift occurs.
  b. Change perceived frequency of ultrasound emitted by moving object.
  c. Sound will be heard (3 voices)

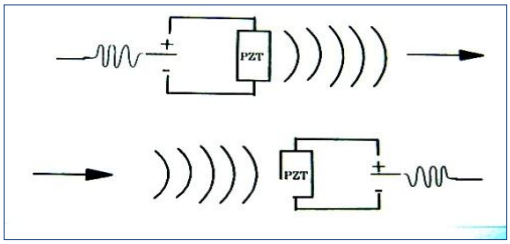
- In clinical practice: moving targets RBC traveling with in the blood vessel.



- Source & Receiver of sound: ultrasound transducer

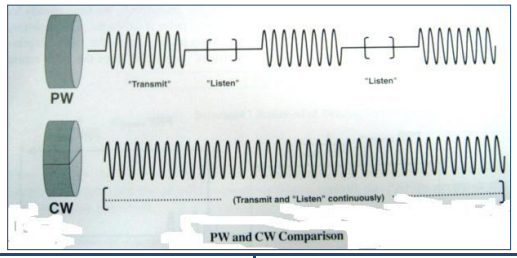
2.1.2 ULTRASOUND TRANSDUCER

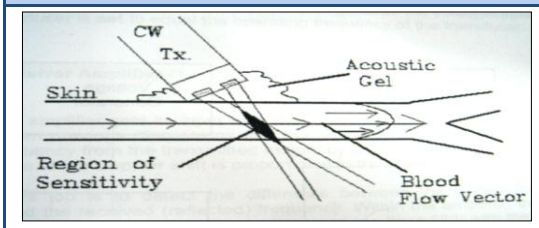
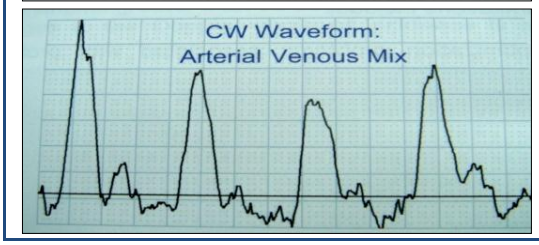
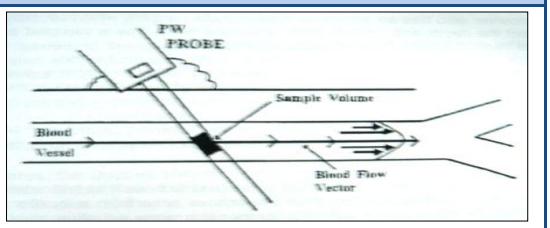
- Transducer: device converts one form of energy to another.
- Ultrasound Transducer:
- Use piezoelectric crystals.
- Converts Electro potential energy (voltage) into Mechanical vibration (ultrasound) & Mechanical vibration into Voltage.



2.1.3 TYPES OF DOPPLER INSTRUMENTS

- Continuous Wave (CW)
- Pulsed Wave (PW)



Continuous Wave (CW)	Pulsed Wave (PW)
Doppler transducer Transmits continuously ultrasound & Receive simultaneously. Have **two Piezoelectric crystals**, one Transmit X & other Receive Y.	**Single piezoelectric crystal** – both transmission & reception. Alternate pulses On & Off. Transmit pulse – system waits – pulse travels to sample volume (specific area) – echo pulse returns
Advantages:	
Magnitude of detectable velocity – limitless.	Specific for depth and range. No mixture of signals like CW Doppler.
Disadvantages ⓘ:	
Not specific for depth Detects any & all vessels in beam path.	Limited maximum detectable velocity unlimited for CW Doppler.
 	

CW: the pocket Doppler, the one the doctor uses.

-CW: It's not specific; it does not give a specific area or a structural picture of the vessels. It only gives the anatomical location in general. (Gives a rough idea)

- PW is more advanced

2.1.4 ANGLE OF INCIDENCE

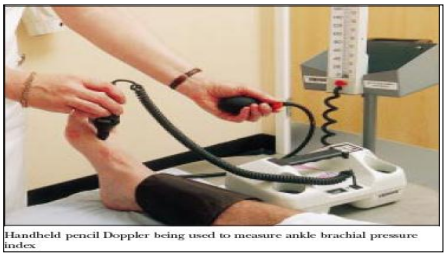
- Doppler or frequency shift is what we hear & see on graphic display.
- Affected by 'angle of flow' or 'angle of incidence'
- The smaller the Doppler angle, the higher the frequency shift.
- **Optimal Doppler signals** ⓘ: transducer angle 45-60 towards direction of flow.

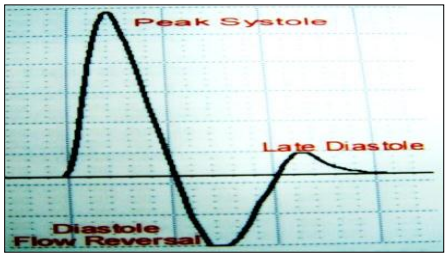
2.1.5 ARTERIAL ASSESSMENT – DOPPLER ULTRASOUND

Triphasic: normal.

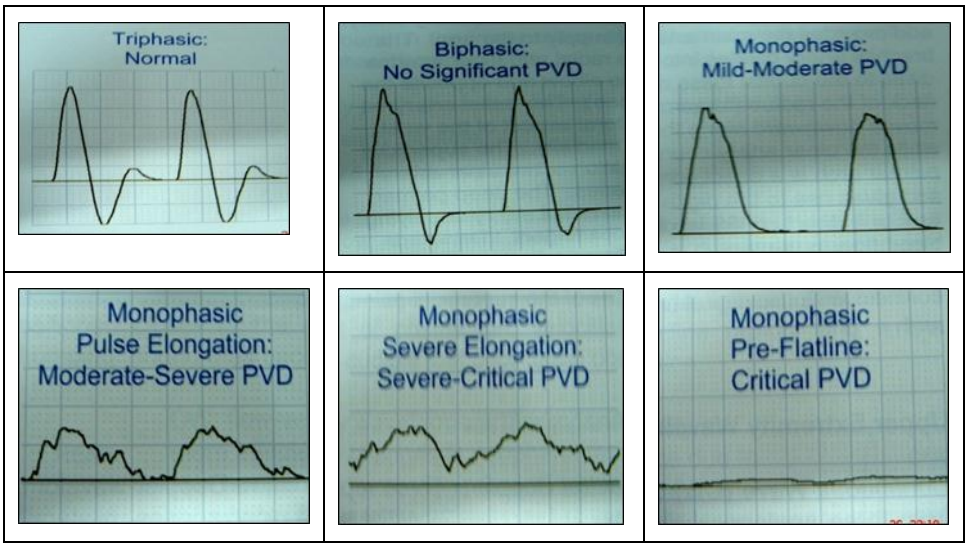
- Monophasic: peripheral arterial disease.

- Audible interpretation
  a. Waveform analysis.
  b. Hand held Doppler.

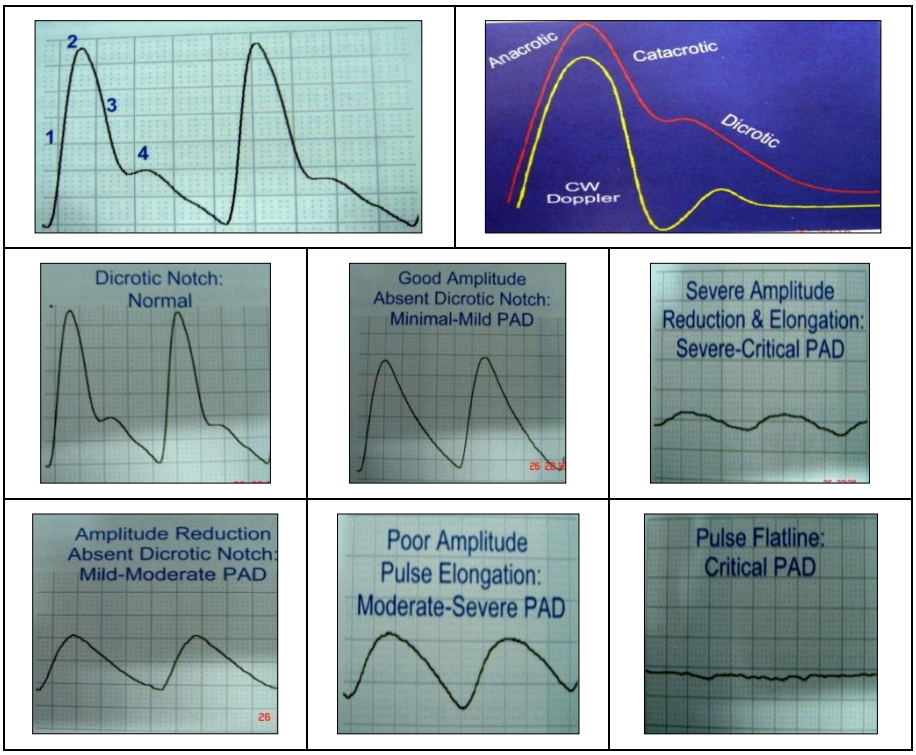

Handheld pencil Doppler being used to measure ankle brachial pressure index



- Normal Peripheral Arterial Doppler signal: TRIPHASIC ⓘ
- **Triphasic arterial signal**ⓘ:
  a. 1st sound – phase: large, high velocity, forward flow, systolic component.
  b. 2nd sound – phase: smaller reverse flow early diastole.
  c. 3rd sound – phase: smaller forward flow late diastole.
- **Audible interpretation & Wave form analysis**



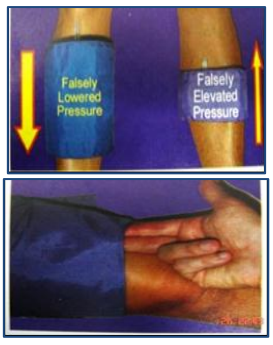
- **PVR (Pulse Volume Recording):** Normal PVR:
  1) Brisk systolic upstroke Anacrotic limb.
  2) Sharp systolic peak.
  3) Gradual down stroke Catacrotic limb.
  4) Dicrotic notch-reflective wave-during diastole normal peripheral resistance.



2.1.5.1 ARTERIAL PRESSURE MEASUREMENTS:

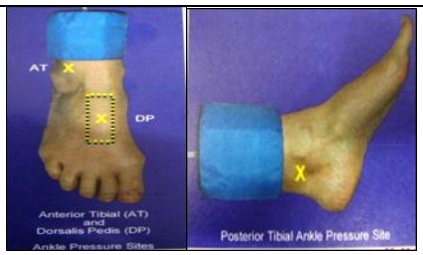
For Peripheral arterial occlusive disease

- Sequence of pressure measurement tests:
  a. Systolic Brachial & Ankle pressure at rest.
  b. Calculation of ABIⓘ.
  c. Toe pressure-non compressible tibial arts.
  d. Segmental pressure and waveforms: low ABI.
  e. Stress testing: severity of claudication & to R/O pseudo-claudication.
- Contraindication to pressure measurements:
  a. Acute DVT; closure of veins makes it worse.
  b. Bandages & casts
  c. Ulceration
  d. Trauma
  e. Surgical site

1) Ankle Brachial Index (ABI):	
Before the test Consider : a) Patient supine arms at sides. b) Basal state (10mnts pretest rest). c) CW Doppler ultrasound. d) Appropriate size pressure cuffs. **When testing :** a) Record bilateral systolic brachial pressure & systolic Ankle pressure (dorslis pedis & post.tib art) b) Interpretation-Ratio highest ankle to brachial pressure.	

ABI & Relation to PAOD:

a) 0.97 -1.25 Normal ⓘ
b) 0.75 – 0.96 Mild PAOD
c) 0,50 – 0.74 Moderate
d) <0.5 Severe
e) <0.3 Critical
f) >1.5 Vessels non compressible ⓘ



ⓘ Vessels are non-compressible in: DM, elderly, renal failure or any condition where arteries are calcified.

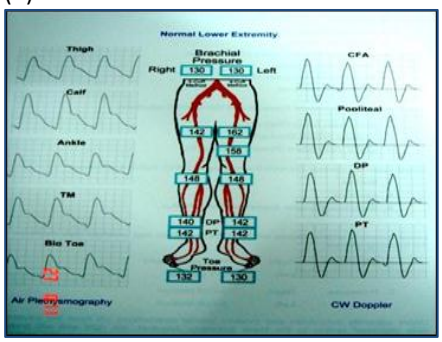
2) Toe Pressure

- This test is done if the ABI showed very high values → like in diabetic patient.
- Normal toe pressure – 2/3rd systolic ankle pressure ⓘ
- Plethysmographic device –it records changes in volume (It is used as a sensor)
- Inflate cuff above 2/3rd of ankle pressure.
- BP cuff (2.5cm) around the base of the toe.
- Gradual deflate until arterial tracing demonstrate return of pulsatile flow – recorded as systolic toe pressure.

3) Segmental Pressures

- Drop in ABI at rest or post exercise indicates hemodynamically significant disease proximal to cuff.
- Segmental pressure measurement – localizes the diseased arterial segment.
- Pressure difference between two adjacent segments <20mm of Hg. **(1)**
- Gradient >30mm of Hg - Hemodynamically significant disease between adjacent levels **(2)**.; Due to the significant drop between two segments e.g. from 120 to 90 (narrowing pressure which is caused by occlusion)

(1)



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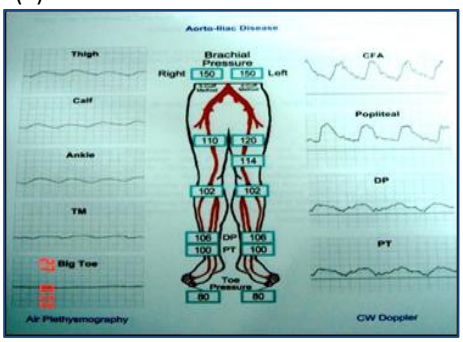
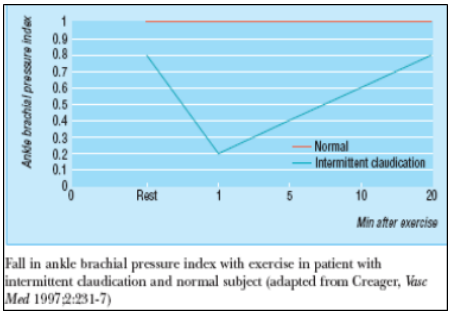


4) Exercise (Stress) Test

- Treadmill stress test.
- Reactive hyperemia stress test.
- Assess functional limitation due to PAOD
- Differentiates PAOD – Pseudoclaudication Ex; neurogenic claudication.
- Resting ankle & brachial pressures.
- Pressure cuffs secured in place –ankle and arm.
- Walk at 2mph at 12% gradient-5mnts or point claudication symptoms.
- Return supine position & measure ankle

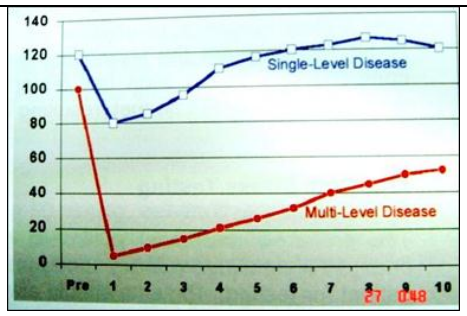


Fall in ankle brachial pressure index with exercise in patient with intermittent claudication and normal subject (adapted from Creager, *Vasc Med* 1997;2:231-7)

pressure 30 seconds and 1 minute post exercise.
- Measure till baseline pressure is recovered.

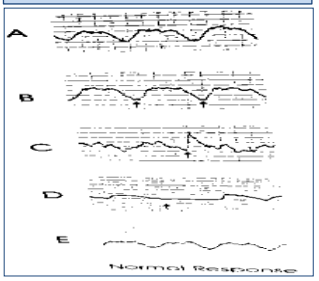
Noteⓘ:
- Duration of exercise.
- Distance walked.
- Symptoms prevented by exercise.

Interpretation:
- Normal: no drop in ankle pressure.
- Minimal disease pressure returns to baseline in 2 minutes.
- Single level disease: pressure returns to baseline in 3-5 minutes.
- Multi-level disease: pressure returns to baseline >10 minutes.



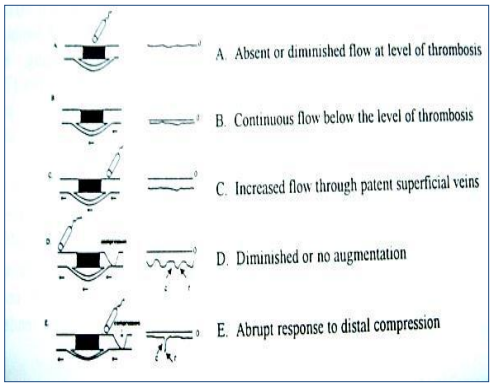
Arterial flow: pulsatile triphasic

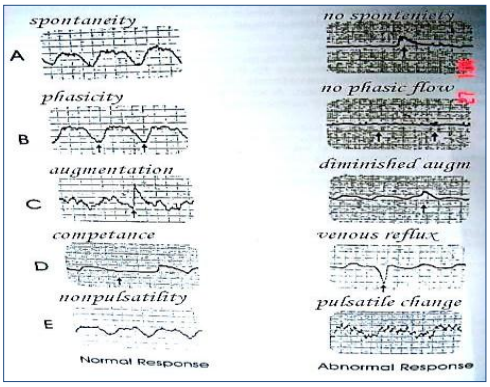
Venous flow: wind-like; normal response ↓



2.1.6 DOPPLER ASSESSMENT OF VEINS

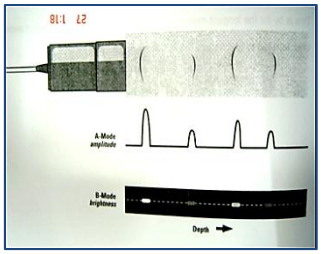
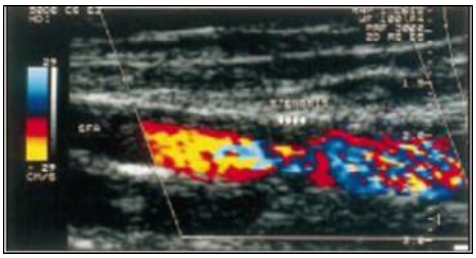
- Five qualities of normal venous flow:
  a. Spontaneity.
  b. Phasicity; with respiration it changes because in inspiration there is no flow because there's no venous return. Expiration increases in venous flow.
  c. Augmentation.
  d. Valvular competence.
  e. Non pulsatility.
- In cases of DVT: Normal five qualities of venous flow are lost; because the vein is closed there will be no sound.





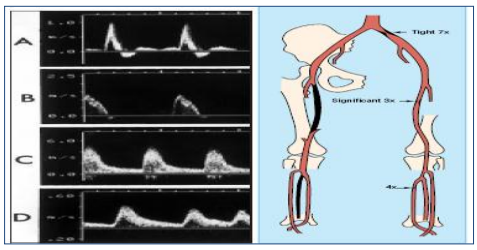
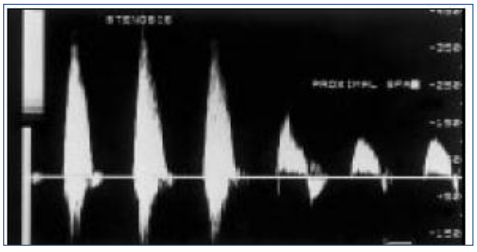
2.2 ULTRASOUND IMAGING DUPLEX

- Imaging Principles:
  a. Amplitude mode (A-mode) method of presenting returning echoes of US on a display screen.
  b. **A-mode**: displayed as vertical deflections or spikes, projecting from baseline. Stronger echoes-higher amplitude signals.

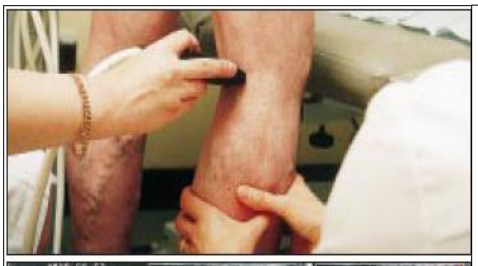
c. **B-mode**: Brightness mode Returning echoes displayed as series of dots.
d. Position of each doctor responds to distance from the sound source rightness corresponds to amplitude of returning echo – Gray scale intensity ⓘ.

- Duplex Scan:
  a. Combination of B-mode imaging with pulsed Doppler US – gives both anatomical & physiological information of vascular system →Duplex Scan.
  b. Addition of color frequency mapping →Color Duplex imaging.

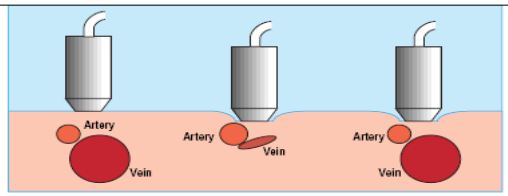



- Uses of color duplex imaging:
  a. Arterial:
     i. Identify obstructive or aneurysmal atherosclerotic disease;
     ii. Peripheral arteries
     iii. Carotid arteries
     iv. Renal & visceral arteries
     v. Surveillance of bypass grafts.
  b. Venous Duplex
     i. Diagnosis of DVT.
     ii. Assessing competence of deep vein valves.
     iii. Superficial venous reflux & identifying Sapheno Femoral & Popliteal Jnc refluxes.
     iv. Preoperative mapping of saphenous vein.
- Criteria for Duplex examination of venous system

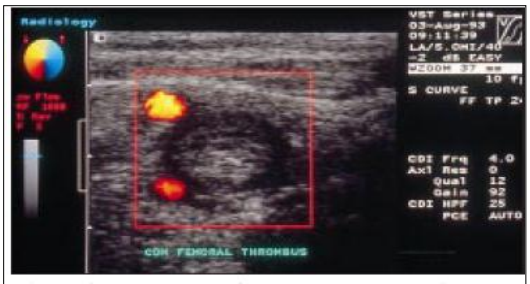
Normal	Abnormal (DVT) ⓘ
• Easily compressible • Should be echo free • Normal valve motion • Normal Doppler signals	• Non compressible • Echogenic thrombus in vein • Incompetent valves • Absent Doppler signals



Colour duplex scanning of saphenopopliteal junction. The calf muscles are manually compressed producing upward flow in the vein (top), which appears as a blue colour for flow towards the heart (panel A). Sudden release of the distal compression causes reflux, seen as a red colour indicating flow away from the heart (panel B)



Ultrasound detection of deep vein thrombosis. The probe is held lightly on the skin and advanced along the course of the vein (left). Pressure is applied every few centimetres by compressing the transducer head against the skin. The vein collapses during compression if no thrombus is present (middle) but not if a deep vein thrombus is present (right)



Colour duplex scan of deep vein thrombosis in common femoral vein adjacent to artery

3 INVASIVE VASCULAR TESTS

3.1 ARTERIOGRAPHY

- Gold Standard ⓘ.
- Good resolution.
- Seldinger technique
- Access –commonly femoral artery & brachial artery ⓘ; easiest accessible artery, least complications with larger arteries, never access small arteries.
- Inject iodinated contrast into the catheter you inserted in the large artery.

3.1.1 TYPES OF CONTRAST

Ionic or high osmolar	Nonionic or low osmolar ; commonly used
• Water soluble • Hypertonic, osmolality 5-10 times of blood. • Causes discomfort at injection site. • More nephrotoxic ⓘ; more complications.	• Has same no of iodine ions ,no cations • Osmolality 1/3rd of high osmolar contrast • Still hypertonic twice that of plasma. • Less nephrotoxic ⓘ • More expensive ⓘ

3.1.2 COMPLICATIONS ⓘ

Local	General	Allergic reaction to contrast
• Hemorrhage • Thrombosis • Pseudo aneurysm • AV fistula • Intimal dissection • Embolization	• Renal – nephrotoxicity • Cardiac- hypertension, arrhythmias, CCF. • Neurological – Carotid angiogram – TIA stroke, convultions. • Pulmonary-bronchospasm, pulmonary edema.	• Minor – nausea, vomiting, head ache, chills, fever, itching. • Intermediate - hypotension. urticaria, bronchospasm. • Major-anaphylaxis, pulmonary edema, laryngeal edema

Pseudo aneurysm; is a pulsatile swelling around the artery due to leaking of blood no dilatation of vessel.

3.2 VENOGRAM

We do it when we are not sure of US results.

3.2.1 ASCENDING VENOGRAPHY

- Relatively invasive study.
- Requires painful venipuncture.
- Injection of iodinated contrast.
- Exposure to radiation.
- Gives information about anatomy and patency of deep veins.
- Locates the incompetent perforator's veins.
- Inject about 40-60 ml of contrast into superficial foot arch veins and tourniquet tied above ankle to visualize deep veins.
- **Indication**ⓘ: High clinical suspicion of DVT with negative or equivocal noninvasive vascular tests (Duplex).
- **Complications**: same as pervious + thrombophlebitis.

3.2.2 DECENDING VENOGRAM

- **Indication**: *to assess the competency of the valves*
  a. To distinguish primary deep venous valvular incompetence from thrombotic disease.
  b. Identify level of deep venous reflux and morphology of venous valves.
- Venographic categories of Deep vein reflux (not imp)
  1) Grade 0 – normal valve function no reflux
  2) Grade 1 – minimal reflux confined to upper thigh
  3) Grade 2 – extensive reflux reach lower thigh
  4) Grade 3 – extensive reflux reach to calf level
  5) Grade 4 – no valvular competence immediate reflux distally to calf.

4 LYMPHEDEMA

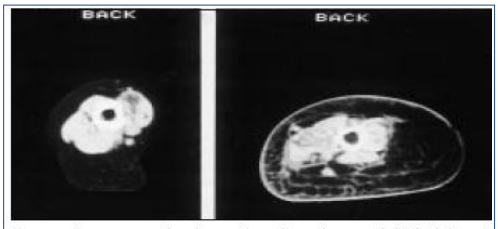
- Lymphedema: accumulation of lymph in the limbs.
- Minimal invasive investigation to identify edema of lymphatic origin:
  a. Lymphoscintigraphy
  b. CT & MRI

4.1 LYMPHOSCINTIGRAPHY

- Isotope Lymphography
  a. Radiolabelled Colloid or Protein injected first web of foot.
  b. Gama Camera monitoring of tracer uptake.
- Measurement of tracer uptake within the lymph nodes after a defined interval – distinguishes lymph edema from edema of non-lymphatic origin.
- Appearance of tracer outside the main lymph routes – dermal back flow indicates Lymph reflux & proximal obstruction.
- Poor transit of isotope from injection site – suggest hypoplasia of lymphatics.

4.2 CT & MRI

- Honeycomb pattern in the subcutaneous compartment, characteristic of lymphedema.



Computed tomogram showing sections through normal thigh (left) and thigh with lymphoedema (right). Note thickened skin and honeycomb pattern

4.3 DIRECT CONTRAST X RAY LYMPHOGRAPHY

- Lymphangiography.
- Lymph vessels identified by injecting vital dyes and lymph vessel cannulated.
- Lipiodol contrast directly injected.
- Normal limb shows opacification of 5-15 main lymph vessels as converge to inguinal lymph nodes.
- Lymphatic obstruction-contrast refluxes into dermal network – dermal backflow.

5 OTHER MODALITIES OF VASCULAR INVESTIGATIONS

Minimally invasive procedures;

- CT, CT Angiogram.
- MRI, MR Angiogram.

6 IMPORTANT NOTES

- Person with abdominal aortic aneurysm , what's the best for diagnostic and surveillance purposes? → noninvasive ultra sound or duplex.
- For follow up of aneurysm, which is 3 cm →US
- Start treating aneurysm when it's 5cm because larger has higher incidence of rupture, less than that don't treat just follow up with US.
- For following up after an open surgery (Endovascular Repair) EVR →CT
- Person with abdominal aortic aneurysm ,what's the best for therapeutic and plan management purposes? →CT
- Local diseases causing Limb swelling →DVT, chronic venous insufficiency, lymphedema.
- For DVT, chronic venous insufficiency →diagnosed by US or duplex US.
- To assess or diagnose lymphatic vessels or lymphedema →lymphoscintigraphy (not lymphangiography) sometimes MRI magnetic resonant angiogram (minimal invasive)
- MRV →venogram (not used because it needs a special software).

7 MCQS

1) In non-invasive assessment of peripheral arterial disease, the following is an appropriate candidate for exercise test:
   a. Patient with rest pain in the foot
   b. Patient with intermittent claudication and normal resting ABI
   c. Patient with venous ulcer
   d. Patient with resting ABI of <0.4
   e. Patient with acute ischemia

2) 15-year old girl presented with progressive painless unilateral leg swelling:
   a. Most likely cause is chronic venous insufficiency
   b. Most likely cause is primary lymphedema
   c. Patient needs arteriogram to confirm diagnosis
   d. Is due to secondary lymphedema
   e. Common treatment is lymphatic bypass surgery

3) 50- year old male patient with swelling, pigmentation and ulceration around the ankle:
   a. Most likely cause is chronic lower limb ischemia
   b. Needs arteriogram for diagnosis and management
   c. Needs non-invasive assessment by Doppler and duplex for obstruction and valvular incompetence of the venous system
   d. Brown skin pigmentation is due to excess of melanocyte activity in the skin
   e. Usually managed by amputation of limb

4) 30 year old female , 26 weeks pregnant has painful swollen and pale left lug and her pedal pulses are well felt:
   a. Arteriogram is indicated because of pale left leg
   b. Optimal initial diagnostic test is venous duplex examination
   c. Appropriate treatment would be warfarin
   d. Venography should be the initial diagnostic test
   e. Heparin is contraindicated in this patient

5) 50 year old diabetic male smoker present with rest pain and gangrene of the 1st toe, the following statement are correct:
   a. ABI in the above patient is the ratio of ankle diastolic pressure to brachial diastolic pressure
   b. ABI in normal person in <0.9
   c. The above patient has critical ischemia and usually ABI <0.4
   d. Calcification of arteries in this patient can give very give ABI results
   e. Always ABI is measured in standing position

6) In vascular investigations:
   a. Doppler is used only for arterial investigations
   b. Duplex scan can be used to evaluate the lymphatic system

c. Bleeding is a common cause of death with venogram
d. None of the above is true

7) Venous system of the lower limb:
a. Consists of superficial, middle and deep systems
b. No connection between its parts
c. Superficial femoral and profunda veins join to form the common femoral vein
d. Great saphenous vein starts posterior to the medial malleolus

8) A 32 year old woman presented to the clinic with thickening skin of her medial aspect of the leg, which was associated with dermatitis and hyperpigmentation. Which type of presentation is this?
a. Telangictasia
b. Lipodermatosclerosis
c. Healed ulcer
d. Active venous ulcer

9) Evaluation does not include which of the following tools?
a. Doppler
b. Duplex
c. Venogram
d. AVP

10) All of the following can treat the previous case except:
a. Stocking
b. Endovenous laser ablation
c. Endovenous laser therapy
d. Surgical ligation

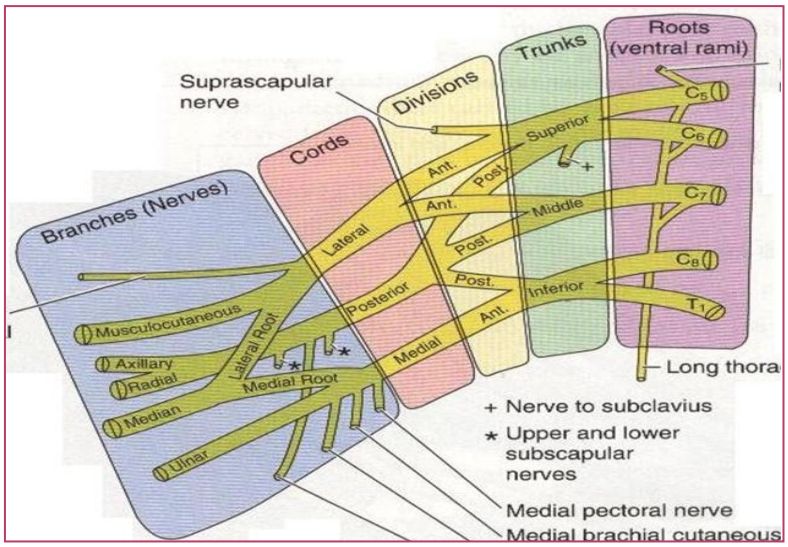
🗝 Answers: 1;B , 2;B , 3;C , 4;B , 5;C , 6;C , 7;C , 8;B , 9;D , 10;A

PERIPHERAL NERVE INJURIES

1 TYPES OF INJURIES

1.1.1 PERIPHERAL NERVE INJURIES

- Axillary nerve
- Musculocutaneous nerve
- Median nerve
- Ulnar nerve
- Radial nerve



2 BRACHIAL PLEXUS INJURIES

2.1 BASIC ANATOMY

- It is formed from the union of the anterior rami of the 5th,6th,7th,8th cervical and 1st thoracic nerves (C5, C6, C7,C8,T1)
- The plexus is divided into **R**oots, **T**runks, **D**ivisions, **C**ords and terminal **B**ranches

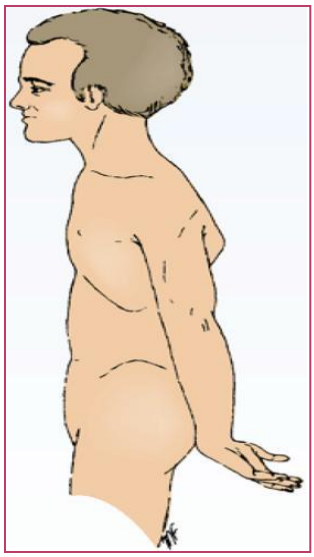
2.2 CLASSIFICATION OF BRACHIAL PLEXUS INJURIES

- **Open injuries** (stab wounds or gunshot wounds):
  - Can be at any level (roots, trunks, divisions, etc.)
  - Classified into:
    - **Supraclavicular** (roots, trunks, divisions)
    - **Infraclavicular** (divisions, cords, terminal branches)
- **Closed injuries**:
  - More common than open injuries
  - **Injury is most commonly at the roots level**
  - Caused by car accidents, outstretching of the shoulder like when playing sports or during difficult deliveries where the baby is pulled in emergency situations

- **Examination of closed injuries: Nerves are not examined, Roots are examined by examining dermatomes (sensation) and myotomes (movement)**

Root	Dermatome	Myotome
C5	Shoulder tip + lateral arm	Shoulder abduction + external rotation
C6	Lateral forearm + thumb and index finger	Elbow flexion
C7	Middle finger	Wrist extension
C8	Ring and little finger + lower aspect of medial forearm	Making a fist
T1	Upper aspect of medial forearm + medial arm	Finger crossing

2.3 TYPES OF CLOSED BRACHIAL PLEXUS INJURIES



2.3.1 UPPER BRACHIAL PLEXUS LESION

- Called **Erb's palsy (Erb-Duchenne Palsy)**
- Injury to **C5, C6 and C7**
- **C5:** loses the ability to abduct the shoulder and external rotation
- **C6:** loses the ability to flex elbow
- **C7:** loses the ability to extend the wrist
  - **Clinically**:
    - The patient will have (opposite to the normal function of the damaged nerves):
    - Shoulder adduction
    - Internal rotation
    - Extension of the elbow
    - Wrist flexion
    - This is called **waiter's tip posture**
- ***Associated injuries:***
  - Injury to the phrenic nerve which arises from the 3rd, 4th, and 5th cervical roots, so half of the diaphragm will be paralyzed
  - In adults X-ray will show elevated hemi diaphragm
  - In children the intercostals are not strong enough to compensate so the baby will have breathing problems (obstetric palsy)

2.3.2 LOWER BRACHIAL PLEXUS LESION

- Called Klumpke's palsy
- Injury to C8 and T1
  - C8: loses the ability to make a fist
  - T1: loses the ability to cross fingers
- ***Clinically*:** The patient will have simian hand and clawing of all fingers
- ***Associated injuries:***
  - Sympathetic nerves to the face come from a branch of the first thoracic nerve T1
  - If T1 is injured then sympathetic to the face are lost on one side and that will result in **Horner syndrome**, which is:
    - Ptosis (dropping of the upper eyelid)

- Miosis (constricted pupil)
- Anhydrosis (inability to sweat)

2.3.3 TOTAL PALSY

- Injury to all roots C5, C6, C7, C8, T1
- Patient is unable to move entire limb: **flail limb**
- ***Quick clinical hints:***
  - Upper lesion (C5, C6, C7) → Erb's palsy and phrenic nerve symptoms
  - Lower lesion (C8, T1) → Klumpke's palsy and sympathetic symptoms
  - Total lesion (C5, C6, C7, C8, T1) → flail limb and both phrenic and sympathetic symptoms

3 PERIPHERAL NERVE INJURIES

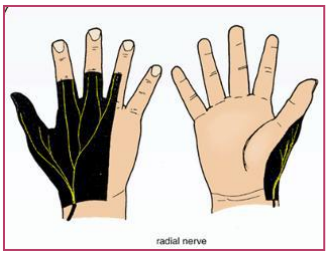
3.1 AXILLARY NERVE

- Isolated injuries to the Axillary nerve most commonly happens with **shoulder dislocation**
- Supplies the **Deltoid** and **Teres minor** muscle
- Clinical features:
  - ***Motor*:**
    - To the deltoid muscle so the patient will not be able to abduct his shoulder
    - The patient can still initiate abduction (action of supraspinatus)
    - It also supplies teres minor that does external rotation which is the same action of infraspinatus, so the patient can still externally rotate his arm
  - ***Sensory***:
    - Loss of sensation over the skin of the lateral arm on lower half of the deltoid
- **Summary: loss of abduction** and **sensation** over the **lateral arm**

3.2 MUSCULOCUTANEOUS NERVE

- Isolated injuries usually happen with stab wounds or gunshots
- Supplies corachobrachilis, biceps , brachalis muscles
- Clinical features:
  - ***Motor:***
    - Corachobrachilis and brachalis are not important clinically
    - Biceps:
      - Weak supination (because the supinator muscle can compensate)
      - Loss of flexion
  - ***Sensory:***
    - Loss of sensation over the lateral forearm and the thumb
- **Summary: loss of elbow flexion** and **sensation** over the lateral forearm + weak supination

3.3 RADIAL NERVE



- Runs in the spiral groove so injuries happen in humours bone fractures
- ***Distribution:***
  - Upper arm (axilla): supplies the triceps -strong extensor of the elbow
  - Lower arm (above the elbow):
    - Brachioradialis
    - Extensor Carpi radialis longus – wrist extension
  - Forearm:
    - Sensory branch: sensation over the three and a half fingers laterally on the dorsal side
    - Motor branch called the posterior interossous nerve: thumb and finger extension
- ***Clinical features:***
  - Humours fracture in spiral groove with radial nerve injury:
    - Normal elbow (triceps is supplied higher, spared)
    - No wrist extension (drop wrist)
    - No thumb and finger extension
    - Numbness or loss of sensation
  - Posterior interossous nerve injury:
    - Stab wound in the forearm
    - Elbow and wrist are normal
    - Thumb and finger extension are lost
      - Finger muscles:
        - metacarpophalengeal (MP) joints
          - Extension is by the radial nerve
          - Flexion is by the ulnar nerve by the interossie and lumbrical
        - Intraphalengeal joints (IP)
          - Extension is by the ulnar nerve by the interossie and lumbrical muscles
          - Flexion by the long flexors of the forearm
- No sensory symptoms!!! Pure motor nerve
- ***Saturday night palsy:***
  - Very high injury of the radial nerve due to compression of the nerve in the axilla
  - Everything is affected (wrist, elbow, fingers, thumb and sensation)
  - Called like this because drunk people sleep with an arm behind the chair that causes the compression
- **Summary**:
  - Remember where the lesion happened
  - Injury to the radial nerve in the axilla: all motor and sensory functions are lost
  - Injury to the nerve in the spiral groove: triceps is spared and everything else is lost
  - Injury in the forearm to the posterior interossous nerve: elbow, wrist and sensation are normal.

3.4 FOREARM

3.4.1 MUSCLES

- ***5 superficial muscles:***
  - Pronator teres → pronation of the forearm
  - Flexor carpi radialis → wrist flexion
  - Palmaris longus → wrist flexion
  - Flexor carpi ulnaris → wrist flexion
  - Flexor digitorum superficialis → flexion of the proximal Intraphalengeal joints (PIP) so flexes the middle phalynx
- ***3 deep muscles:***
  - Flexor digitorum profundus
  - Flexor pollicis longus
  - Pronotor quadrates

3.4.2 NERVE SUPPLY

- All of these muscles are supplied by the median nerve except 1 and a half are supplied by the ulnar nerve:
  - Flexor carpi ulnaris
  - Half of flexor digitorum profundus to the little and ring finger
- The median nerve has 2 branches
  - Superficial which supplies the superficial group
  - Deep (anterior interossous nerve) which supplies the deep 2 and a half muscles (PURE MOTOR)

3.4.3 HAND MUSCLES

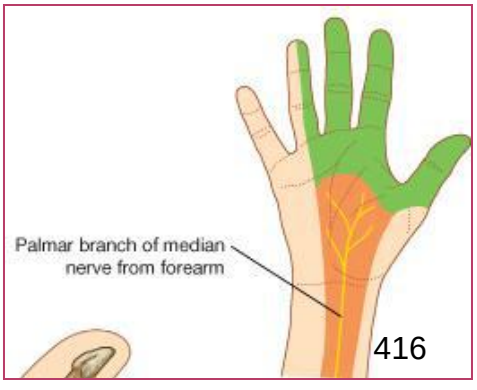
- **Hypothenar**: opposition of the little finger
- **Thenar**: opposition of thumb + adduction of the thumb (adductor pollicis)
- **Interossie**: abduction and adduction of the fingers + MP flexion + IP extension
- **Lumbricals**: MP flexion + IP extension

3.4.4 NERVE SUPPLY

- ***The hand has 20 muscles***
  - 15 supplied by the ulnar nerve (3 hypothenar + 8 interossei (dorsal and palmar) + 2 lumbricals + adductor pollicis + Palmaris brevis)
  - 5 by the median nerve (3 thenar + 2 lumbricals (1st and 2nd)
- All the actions are from the ulnar nerve except 2 are from the median nerve:
  - Opposition of the thumb
  - Index and middle lumbricals

3.5 MEDIAN NERVE

- ***Motor***:
  - Superficial flexors except flexor carpi ulnaris
  - Deep flexors except half of flexor digitorum profundus to little and ring finger
  - Thenar muscles



- Index and middle lumbricals
- ***Sensory***: lateral 3 and a half fingers on the palmer side
- ***Clinically***:
  - Anterior interosseous nerve injury:
    - Affects the deep 2 and half muscles:
      - Half of Flexor digitorum profundus
      - Flexor pollicis longus
      - Pronotor quadrates (pronation is not lost because of pronator teres)
    - Sign: the patient "cannot make a perfect O" with the thumb, index and middle fingers because he can't flex the tips of the index and middle finger (DIP joint: this is the action of the flexor digitorum profundus muscle)
- Median nerve injury at level of wrist:
  - Common in patients who attempt suicide
  - Loss of opposition
  - Loss of sensation 3 and a half lateral
  - Lumbricals are lost but interossie do the job
  - They still can make an "O", bend the wrist and flex the PIP
- Carpal tunnel syndrome:
  - Loss of sensation first
  - If untreated weakness of opposition
- **Summary**:
  - Injury to median nerve at level of the wrist: loss of opposition and loss of sensation
  - Injury to anterior interosseous branch of median nerve: patient cannot make an O + normal sensation

3.6 ULNAR NERVE

- ***Motor:***
  - Flexor carpi ulnaris
  - Medial half of flexor digitorum profundus
  - Lumbricals + interossie + hypothenar + adductor pollicis
- ***Sensory:*** medial 1 and a half fingers front and back of the hand
- ***Clinically:***
  - Ulnar nerve injury:
    - loss of flexor carpi ulnaris and half of flexor digitorum profundus
    - loss of sensation
    - all of the hand muscles
      - cannot oppose the little finger
      - atrophy of hypothenar muscles
      - Cannot adduct or abduct the fingers
      - Ends up with ulnar **claw hand**
  - Ulnar nerve injury at the wrist:
    - Sensation is lost
    - All hand muscles:
      - Hypothenar atrophy
      - No opposition of the little finger
      - Cannot adduct or abduct the fingers

        - Loss of thumb adduction resulting in froment's sign
        - Froment's sign: you ask the patient to hold a pen with his thumb but he cannot so he contracts the flexor pollicis longus because the adductor pollicis is lost
- **Summary** of ulnar nerve injury:
    - Ulnar claw
    - Loss of sensation
    - Hypothenar atrophy
    - Positive froment's sign
    - Cannot adduct or abduct the fingers

3.7 MEDIAN AND ULNAR NERVE INJURY AT THE WRIST

- Loss of intrinsic muscles
- Loss of sensation
- Clawing of all the fingers = ape hand (semian hand)

4 MCQS

1. **Erb's palsy:**
    a. C5 and C6
    b. C7 alone
    c. C8 and T1
    d. Total palsy
    e. Lower brachial plexus injury
2. **The abductor pollicis longus muscle is supplied by:**
    a. Median nerve
    b. Ulnar nerve
    c. Anterior interosseous nerve
    d. Radial nerve
    e. Axillary nerve
3. **The main action of the C6 root of the brachial plexus is:**
    a. Making a fist
    b. Crossing the fingers
    c. Elbow flexion
    d. Wrist extension
    e. Elbow extension
4. **The intrinsic muscles of the hand are supplied by:**
    a. C5
    b. C6
    c. C7
    d. C8
    e. T1
5. **Klumpke's palsy has all the following characteristics except:**
    a. Can result from motor cycle injury
    b. Anhidrosis
    c. Loss of dermatomes
    d. Phrenic nerve palsy
    e. Miosis
6. **A patient with posterior interosseous nerve palsy:**
    a. Unable to extend his wrist.

b. Can extend the IPJs of the fingers.
c. Can extend the MPJs of the fingers.
d. The sensation over the radial half of the hand is lost.
e. None of the above.

7. **Lateral cutaneous nerve of the forearm is a branch of which nerve:**
   a. Axillary
   b. Radial
   c. Musclocutaneous
   d. Ulnar
   e. None of the above
8. **in a patient with anterior interossous nerve palsy, what is false:**
   a. Can pronate the forearm
   b. Can flex the PIP of the index
   c. Have positive O sign
   d. Can flex the IPJ of the thumb
   e. All of the above are true

9. **After nerve injury, nerve recovery is at rate of:**
   a. 1 mm/day
   b. 2 mm/day
   c. 3 mm/day
   d. 4 mm/day
   e. 5 mm/day

4.1 ANSWER YES OR NO

1. **A patient cut his median nerve at the wrist:**
   a. Has he lost opposition of the thumb?
   b. Has he lost any sensation?
   c. Can he flex the tip of the index finger?
2. **A patient is known to have "Saturday night palsy" is there Loss of supination?**
3. **Can a patient with erb's palsy also have phrenic nerve palsy?**
4. **A patient has klumpke's palsy:**
   a. C5, c6, and c7 are completely intact?
   b. Only c8 or T1 are injured?
   c. Can move his shoulder, the elbow and the wrist?
   d. Can't make a fist?
   e. Can use his intrinsic muscles of the hand?
   f. Will have clewing of all fingers "simian hand"?
   g. Can have phrenic nerve palsy?
   h. Can have Horner's syndrome?
5. **patient has cut his median nerve at the level of his arm:**
   a. Can he flex his wrist?
   b. The patient will flex more in the radial deviation?
   c. Is the FDS completely paralyzed?
   d. Is the FDP completely paralyzed?
   e. Is there sensory loss?
   f. Can he still oppose his thumb?

g. Can he flex the tip of the thumb?
h. Can he flex the tip of the index finger?
i. Can he flex the tip of the little finger?
j. Can he flex the PIP of the little finger?
k. Does he have sensation of the volar aspect of the little finger?
l. Does he have sensation of the volar aspect of the thumb?
m. Can he flex the tip of the ring finger?
n. Can he flex the PIP joint of the ring finger?
o. Can he flex the PIP joint of the index finger?

6. **Can a patient with erb's palsy also have horner's syndrome?**
7. **Patient comes with a stab wound to the axilla which cut his radial nerve:**
    a. He's unable to extend his elbow
    b. He can extend the wrist
    c. He's unable to extend and radially abduct the thumb
    d. He's unable to extent the MP joints of the finger
    e. He will have wrist drop
    f. Can he extend the IP joints of the fingers
8. **Patient presents with superficial radial nerve injury (cut in the mid forearm) will only have sensory loss?**
9. **Patient presents with posterior interosseous nerve injury:**
    a. His triceps is paralyzed.
    b. He has loss of sensation over the dorsum of the thumb.
    c. He is unable to extend the elbow.
    d. Can he extend the wrist?
    e. Can he extend the thumb?
    f. His supinator muscle is paralyzed.
    g. Can he supinate the forearm?
    h. Will thumb radial abduction be lost?
    i. Will MP joint extension be lost?
    j. Will IP joint extension be lost?
    k. Can he extend the IP joint of the little finger?
    l. Is there loss of sensation?
10. **Can a patient with erb's palsy make a good fist?**
11. **A patient comes to the clinic with isolated axillary nerve injury.**
    a. Clinical examination is mainly the teres minor.
    b. He will not be able to initiate abduction.
    c. He will not be able to externally rotate.
12. **Patient has paralysis of the extensor digitorum:**
    a. Can he extend the IP joint of the thumb?
    b. Can he extend the IP joint of the index?
13. **Clinically, only two things are important when it comes to musclocutaneous nerve injury: biceps and lateral cutaneous nerve of the forearm.**
14. **A patient with injury to roots C5, C6, and C7:**
    a. Can't abduct or external rotate, so he will go into adduction and internal rotation.
    b. Can't flex the elbow, so he will go into elbow extension.
    c. Can extend the wrist.
    d. Will have complete claw hand.

4.2 TRUE OR FALSE

1. **Patient presents with injury to the anterior interosseous nerve:**
    a. Patient lost sensation at the tip of the thumb
    b. Patient lost sensation in the palm of the thumb.
    c. Patient's sensation is normal.
    d. Patient's pronation is normal.
    e. Patient cannot flex his wrist.
    f. Patient cannot oppose the thumb.
    g. Patient can flex the MP joint of the thumb.
    h. Patient can flex the IP joint of the thumb
    i. Patient can flex the tip of the index.
    j. Patient can adduct the thumb.
    k. Patient can flex the PIP joint of the index.
    l. Patient can flex the tip of the thumb.
    m. Patient can pronate the forearm.
    n. Patient cannot make a perfect O.
    o. Patient has no sensory loss in the hand.
    p. Patient cannot flex the little finger.
    q. Patient can flex the MP joint of the index finger.
    r. Patient cannot flex the PIP joint of the index finger.
    s. Patient can flex the tips of the index and middle fingers
2. **Patient who has cut his posterior interosseous nerve cannot supinate?**
3. **Patient cut his ulnar nerve at the wrist:**
    a. He can feel the back of his little finger
    b. He can flex his wrist
    c. He can flex the wrist in ulnar and radial deviation
    d. He cannot flex the tip of the index finger.
    e. He can flex the tip of the little finger.
    f. He can flex the PIP joint of the little finger.
    g. He can feel the palmar surface of the little finger.
    h. He can feel the dorsal surface of the little finger.
4. **Patient cut his median nerve at the level of his elbow:**
    a. He had lost the ability to oppose the thumb.
    b. He has sensory loss.
    c. He is still able to flex his wrist.
    d. Thumb tip flexion is normal.
    e. Pronation is lost.
5. **Complete loss of the ulnar nerve:**
    a. The caused by cutting the ulnar nerve at the wrist
    b. Is caused by cutting the ulnar nerve in the arm
    c. Loss of wrist flexion.
    d. Can't flex the wrist in ulnar deviation
    e. Can flex the tips of the fingers.
    f. Inability to flex the tips of the ring and little fingers.
    g. Able to flex the IP joints of the fingers
    h. There's no sensory loss.
    i. Can feel the back of the hand.
    j. Can't feel the front of the hand.

k. Able to oppose the thumb and the little finger.
l. Is able to adduct and oppose the little finger.
m. Will have Froment's sign.
n. Is able to adduct and abduct the fingers.
o. Is able to flex the PIP joints of the little finger

6. **A patient cut his ulnar nerve in the mid-forearm:**
   a. He can feel the back of his hand.
   b. He can feel the front of his hand.
   c. He can adduct and abduct the fingers.
   d. He can adduct the thumb,
   e. He has Froment's sign.
   f. He cannot oppose the little finger.
   g. He cannot oppose the thumb.

🗝 **MCQs:** 1=a, 2=d, 3=c, 4=e, 5=d, 6=b, 7=c, 8=d, 9=a

🗝 **Yes/No:**

1= (a) yes, (b) yes, (c) yes
2= no
3= yes
4= (a) yes, (b) no, (c) yes, (d) yes, (e) no, (f) yes, (g) no, (h) yes
5= (a) yes, (b) no, (c) yes, (d) no, (e) yes, (f) no, (g) no, (h) no, (i) yes, (j) yes, (k) yes, (l) no, (m) yes, (n) yes, (o) no
6= no
7= (a) yes, (b) no, (c) yes, (d) no, (e) yes, (f) yes
8= yes
9= (a) no, (b) no, (c) no, (d) yes, (e) no, (f) yes, (g) yes, (h) yes, (i) yes, (j) no, (k) yes, (l) no
10= yes
11= (a) no, (b) no, he will be able to initiate abduction, (c) no, he will be able to externally rotate the arm
12= (a) yes, (b) yes
13= yes
14= (a) yes, (b) yes, (c) no, (d) no

🗝 **True/False:**

1= (a) F, (b) F, (c) T, (d) T, (e) F, (f) F, (g) T, (h) F, (i) F, (j) T, (k) T, (l) F, (m) T, (n) T, (o) T, (p) F, (q) T, (r) F, (s) F
2= F
3= (a) T, (b) T, (c) T, (d) F, (e) T, (f) T, (g) F, (h) T
4= (a) T, (b) T, (c) T, (d) F, (e) T
5= (a) F, (b) T, (c) F, (d) T, (e) F, (f) T, (g) F, (h) F, (i) F, (j) T, (k) F, (l) F, (m) T, (n) F, (o) T
6= (a) F, (b) F, (c) F, (d) F, (e) T, (f) T, (g) F

HAND INJURIES

1 INTRODUCTION

1.1 HISTORY

- Hand dominance
- Occupation
- Previous hand trauma or injury
- Smoking
  - Patients who smoke have vasoconstriction of blood vessels and that makes connecting an amputated finger have a high chance of failing so the doctor must know before he goes into the OR
  - No point in wasting time, this procedure takes 6-8 hr so if pt smoker from beginning say you can't
- Tetanus
  - Make sure the patient is vaccinated, if not give him vaccination
  - Any open wound there is risk of infection (tetanus)
- Acute vs. Chronic
  - Acute e.g. Trauma, burns, laceration, fractures, dislocation, infection
  - Chronic e.g. Lumps , Carpal tunnel syndrome and nerve compressions, arthritis
- Mechanism of injury and complaint
  - Trauma, Laceration, Swelling or lump, Arterial or Venous injury, Dislocation, Infection, Numbness

The ulnar nerve is the most important nerve in the hand because it controls all action except opposition of the thumb by the median nerve

Ulnar supplies all muscles except thumb muscles (Abductor pollicis brevis, flexor pollicis brevis, Opponens pollicis) and 2 lumbricals by the median nerve

–BUT Adductor pollicis

1.2 EXAMINATION

1. Inspection
   1. Compare both hands (always compare to a normal hand)
   2. Dorsum then volar surface
      - Skin (Ulcers or lesions or color)
      - Swelling
      - Wasting
      - Position normal position of hand if u put it on table: flexion cascade "the flexor tendons are stronger then extensor tendons". If someone can't do this >injury to flexor tendons.
2. Palpation
   - Feel Tenderness, sensation, temperature, Capillary refill
3. Check Movement
   - Move Range of Motion
     - Passive, Active
     - Examine FDS, FDP, & extensor tendons
   - Test Specific Nerves (Sensory + Motor)
     - Median (sensation to lateral three and a half volar side)
     - Ulnar (sensation to medial one and a half on the volar and dorsal side)
     - Radial (lateral three and a half dorsal)

There are no intrinsic muscles on the dorsum of the hand all of them are on the volar surface

•Radial nerve doesn't give any motor supply to hand only sensation

2 groups of hand muscles:

–Extrinsic

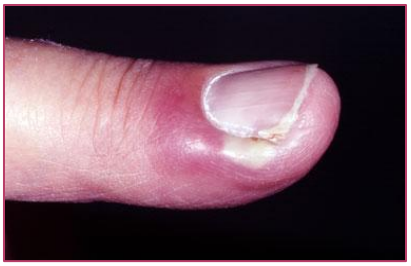
•Originate from the forearm and insert in the hand

–Intrinsic

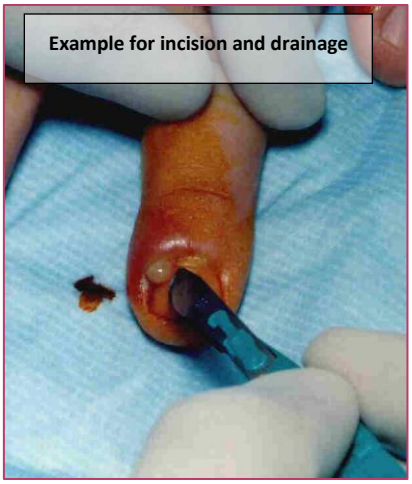
•Originate and insert in the hand

2 HAND INFECTIONS

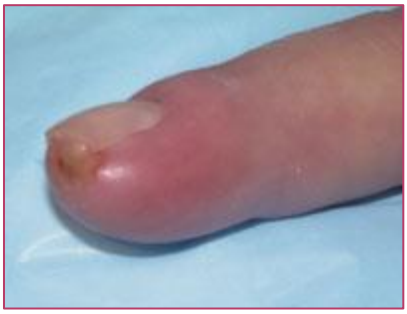
2.1 PARONYCHIAL INFECTION



- Most common hand infection ⓘ
- Infection of the nail bed or nail plate
- Present with redness around the nail
- Could be just cellulitis and redness or abscess
- Most common organism is Staph Aureus
- Treatment:
  - Antibiotics + warm saline soaking
  - If there is no response in 48 hours you must do Incision and drainage
  - If there is an abscess then you must do incision and drainage
- If someone gets paronychial infection frequently (6 times a year) think of chronic infection
  - Most common cause of chronic infection is candida (fungi)
- Treatment:
  - Suspect Candida so send swab
  - If + give oral antifungal or topical
  - If no response then remove the skin and clean then graft

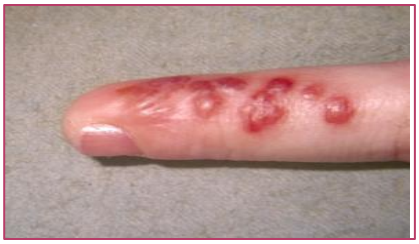


2.2 FELON



- 38% of all surgical infections *(very common ⓘ)*
- Infection of the finger pulp
- This area is very sensitive because it has many nerve endings
- 2 point discrimination is maximal at this area
- So when it develops an abscess between it and the skin it causes nerve compression and SEVERE PAIN
- Treatment:
  - Antibiotics + warm salt soaks
  - If no response incision and drainage
    - Incision must be made from the side to not loose sensation

2.3 HERPETIC WHITLOW

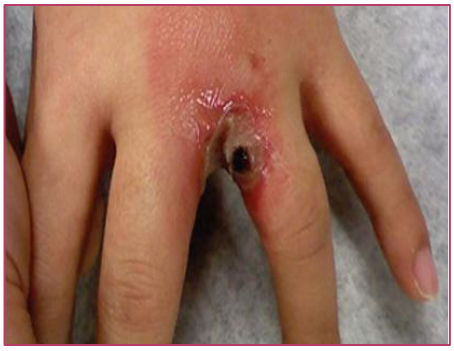


- HSV type 1 vesicular eruption of the fingertip
- Vesicles that contain clear fluid

- Happens to children (biting nails) and dentists
- Very painful
- Very contagious (patients need isolation)
- Treatment by acyclovir (antiviral medication)

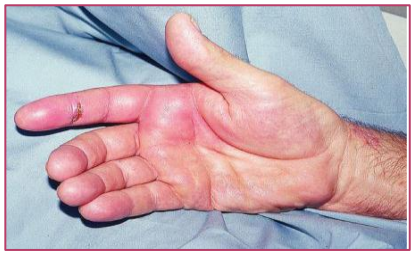
2.4 COLLAR ABSCESS

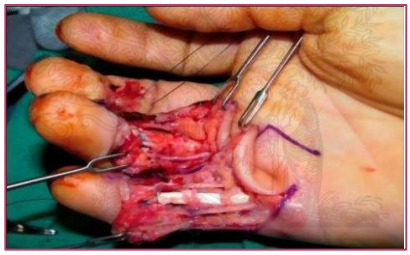
- Abscess of the hand web-Space (Connection point between the volar and dorsal parts)
- Presents with redness, swelling and abducted finger
- Treatment:
  - Antibiotics if early with observation as in or out patient
  - Incision and drainage in the OR (complex area)



2.5 FLEXOR TENOSYNOVITIS

- Each finger has 2 flexor tendons; one moves PIP (attached to middle phalanx), the other DIP (attached to distal phalanx).
- Infection of the flexor tendon sheath due to trauma
- Can extend to the forearm
- 4 signs:
  - Sausage-shaped fingers
  - Flexed position
  - Pain with passive extension
  - Tenderness along the tendon
- Treatment:
  - Must be IMMEDIATE because of high risk of sepsis,necrosis and amputation
  - You have to do incision and drainage
  - Antibiotics
  - Catheter irrigation (irrigate the sheath with saline)
  - If the infection is bad, it can cause thrombosis of artery, ischemia of nerve and insensate the fingers.





Catheter irrigation

•You pass a catheter between 2 ends of the flexor sheath and you keep it there until the area is clean

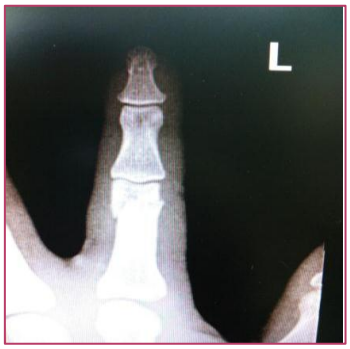
•Until you clean out all the pus, if you are not happy leave this catheter in, take the patient to the ward and nurses will irrigate every 6hr and u will take it out after 48-72 hr,

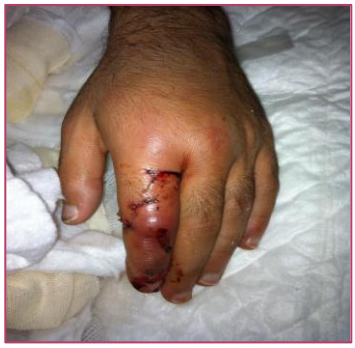
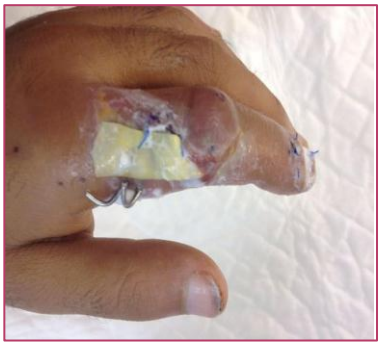
•if you are still not happy with the wound open the whole finger and clean then close loosely never close infected wound completely.

2.6 HAND BITES

- The problem with bites is that the saliva is full of bacteria
- Human: Staph, Strep, Eikenella ⓘ
- Dogs:
  - Pasteurella Multocida (very dangerous), Staph, Strep

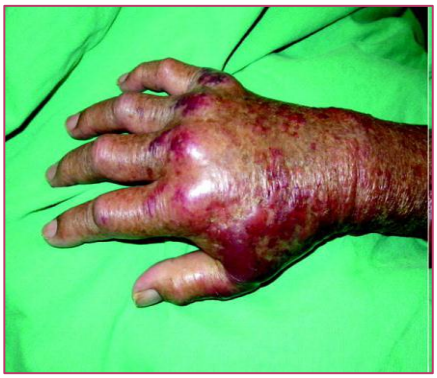
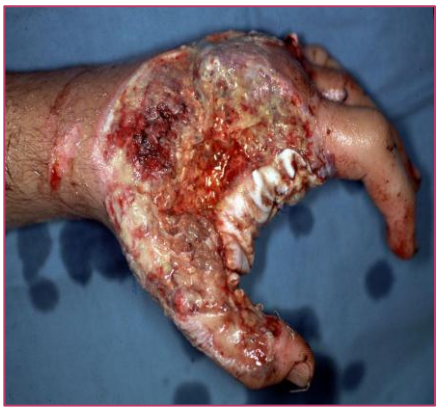
  - With street dogs, the most likely cause is rabies
  - All must get rabies treatment: IgG and rabies vaccine (5 injections in abdomen at day 1,3,7,14,28)
- Cats:
  - More dangerous than dog bites (more concentration of bacteria within the saliva)
  - Pasteurella Multocida
- All of them should be admitted for IV antibiotics
- Most of dog & human bites respond well to Augmentin Tetanus
- If given the antibiotics and there's no response in 48 hours, we do incision + drainage



3 NECROTIZING FASCIITIS

- Flesh eating disease of the soft tissue
- Occurs in diabetics with low socioeconomic status (immunocompromised)
- Pt presents with infection and is unstable (hypotension , tachycardia, ALOC and low urine output)
- Caused by Group A B-hemolytic strep
- Infection of the fascia
- Skip lesions on the skin
- Has high mortality rate
- Treatment:
  - Patient needs to be intubated and admitted to ICU
  - Needs extensive debridement and IV Antibiotics
  - So stabilize the pt, take him to the OR, and open all of the infected area in whichthe fascia will look gray with a bad smell. Once you see a healthy area > skip and open again to make sure that there's no extension.
  - Some patients don't respond to the 1st or2nddebridement > amputation!

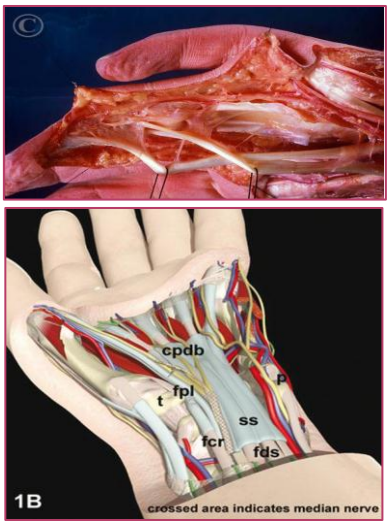



4 FLEXOR TENDONS

> No muscles in finger (only tendons) so it will survive in case of ischemia more than 6 hours.

4.1 ANATOMY

- There are 8 muscles with almost 12 tendons in the flexor side, (4FDS, 4FDP, FPL, FCU, FCR, PL)
  - FCU,FCR, PL: flex the wrist
  - 4FDS: flex PIP joint
  - 4FDP: flex DIP joint
  - FPL: flex thumb
- Origin
  - Medial epicondyle to the forearm then develops tendons and goes through the carpal tunnel to insert into the hand and fingers
- Nerve Supply
  - All of them by the median nerve
  - **Except:** FCU and medial ½ of FDP

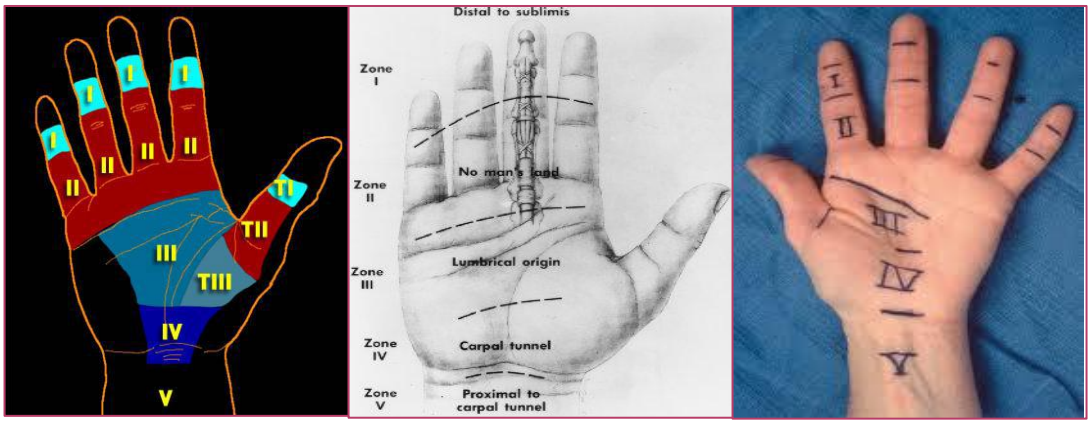


4.2 MECHANISM OF INJURY

- Closed vs. Open
  - Closed:
    - Completely flexed and then sudden severe hyperextension
  - Open
    - Laceration: Knife being the most common tool for it
    - Crush injury
    - Degloving injury

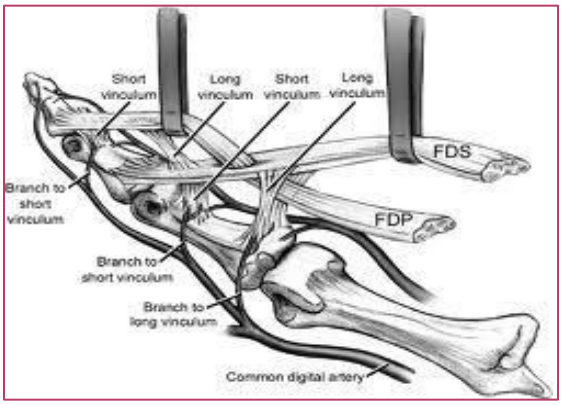
4.3 VERDAN'S 5 ZONES

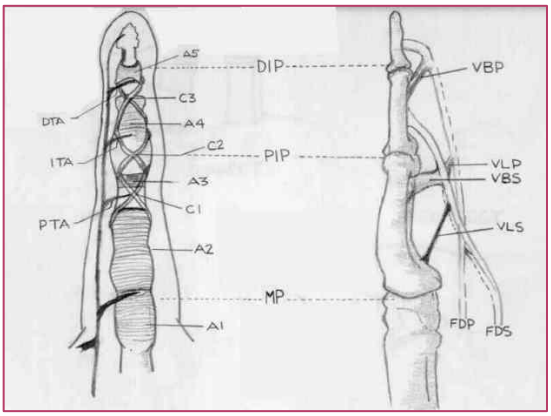
- Classified mainly to get an idea of the expected outcome after repair
- Zones 3,4 and 5 have a good chance; as you go distally (zone 2), chances of full recovery are less because of the small space
- **Zone 1:** Only affects the FDP
- **Zone 2:**
  - FDP&FDS
  - Extends from MCP joint to insertion of FDS
- **Zone 3:**
  - From distal area of carpal tunnel to MCP joint
  - Dangerous because it also affects nerves and arteries
- **Zone 4:** Area under carpal tunnel
- **Zone 5:** The distal forearm



4.4 PULLY SYSTEM AND TENDON BLOOD SUPPLY

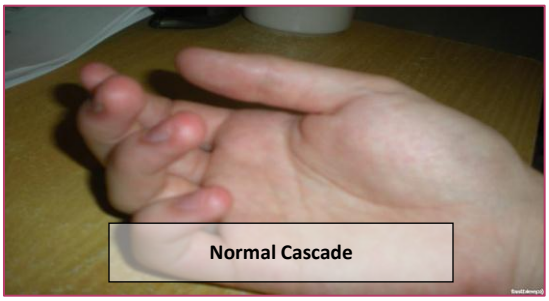
- Small ligaments are present in front of the tendons to hold them in place (A1-A5, C1-C3). Each tendon has its own blood supply.





4.5 CLINICAL EXAMINATION AND FINDING

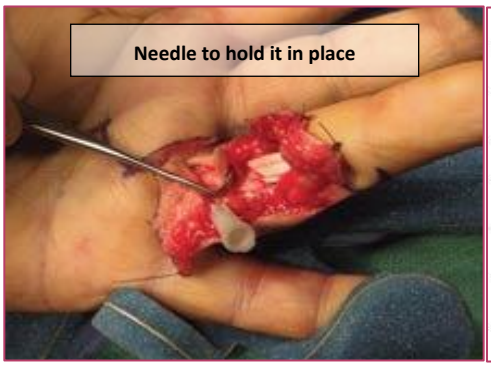
- Loss of flexion cascade
- Open wound most commonly
- Tendon could be visible in the wound
- Inability to flex the digit at PIP or DIP

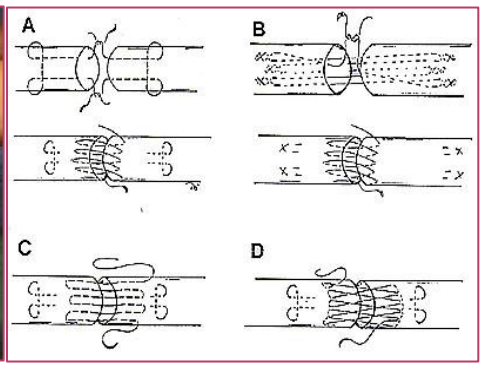


How to examine FDS and FDP?	
FDS	**FDP**
The Flexor Digitorum Superficialis (FDS) inserts into the middle phalanx of each finger. It is tested by blocking the finger MCP joint and asking the patient to flex the PIP joint. To block the MCP joint, hold the proximal phalanx in extension just distal to the MCP joint, so that the MCP joint is unable to bend when the patient tries to flex the finger.	The Flexor Digitorum Profundus (FDP) inserts into the distal phalanx of each finger. It is tested by blocking the finger PIP joint and asking the patient to flex the DIP joint. To block the PIP joint, hold the middle phalanx in extension just distal to the PIP joint, so that the PIP joint is unable to bend when the patient tries to flex the finger.
	

4.6 FLEXOR TENDON REPAIR

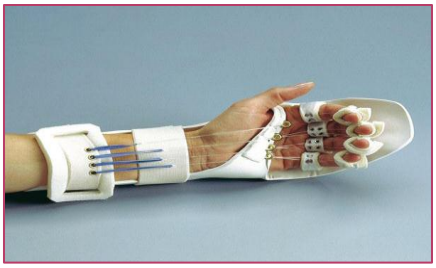
- Explore the wound in zigzag fashion
  - In OR; because this area has nerves and blood vessels
  - Zigzag not straight cut why? It'll cause flexion contraction
- Find the 2 ends of the cut tendon and pull it out then insert needle
- Repair : > 25 different technique for the repair
- Non absorbable suture> because of the poor blood supply





4.7 FLEXOR TENDON SPLINTS

- You can't let patient use his hand the repair will be cut! Also to keep it in the functional position to make sure adhesions will not damage function



- The wires allows pt to move fingers without tension. U don't want cause adhesion if u let it 3-4 weeks without movements.

5 REPLANTATION

5.1 INDICATIONS AND CONTRAINDICATIONS

- Indications
  - Amputated Thumb: It provides 50% of hand Function
  - Children: The risk of loss is higher than adults because vessels are very small & more difficult.
  - Multiple digits: You try to fix 2-3 so he can hold things.
  - Partial or whole hand: Because they have a lot of function problems.
- Contraindications
  - Life threatening injury: You want to save the pt's life it's more important.
  - Sever chronic illness
  - Multilevel injury
  - Severely crushed injury
  - Single digits: Because the pt will not have functional defects.
  - Severe contamination
  - Avulsion injury: Finger gets pulled out; artery needs to be reattached at wrist level
- Duration of surgery 6-8 hours
- 40% chance of failure
- Can't work 3-6 months

5.2 GENERAL PRINCIPLES

- Resuscitate the patient
- Preserve amputated part in cold water not directly on ice (frostbites)
- Warm ischemia time > must operate within 6-8 hours. If cold > within 12-24 hours (longer)
- Successful replant after 28 hours: The longer its preserved, the better.
- X-ray the hand and the amputated part
  - Make sure no fractures because in that case you can't replant it
- Consent for vein, nerve, tendon and skin graft
- Prepare the amputated part
- 1st Shorten the bone
- Arthrodesis
- 2nd Repair flexor and extensor tendon
- Repair (3rd) Digital artery (4th) vein and (5th) nerve
- 6thSkin closure +/- skin graft

5.3 COMPLICATIONS

5.3.1 WHITE FINGER

- No blood Flow (Low arterial flow)
- Technical or non- technical
- If patient is a smoker don't bother to replant
  - Ensure pt is warm
  - Well-hydrated
  - Prevent hypotension
  - Loosen dressing
  - Remove sutures
  - Re-Explore and check arteries if all doesn't work

5.3.2 BLUE FINGER

- Veins are not draining (High venous flow)
  - Elevate limb
  - Loosen dressing
  - Remove sutures
  - Leeches
  - Remove nail
  - Heparin injections
  - Re-Explore
- Leeches, in case of venous congestion, suck the blood relieving the congestion

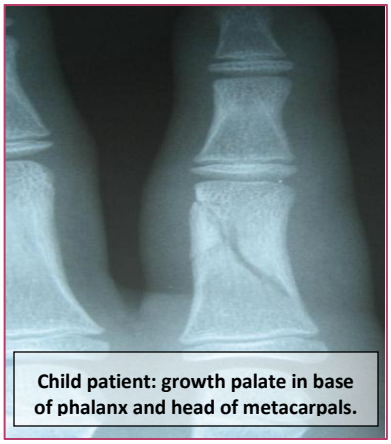
6 HAND FRACTURES

6.1 UNSTABLE FRACTURE

- Cannot be reduced closed or cannot be held reduced without fixation
- 30% risk of infection in open fracture including open Distal Phalanx fracture
  - Reduced to 3% with antibiotics
  - The distal phalanx fracture with subungal hematoma (bleeding in nail) should be considered an open fracture
  - Healing 4/52's for phalangeal fracture whereas 5-6/52's for metacarpal fracture

6.1.1 ACCEPTABLE HAND FRACTURES

- Tuft distal phalanx
- AP displaced metapheseal fracture in children
- MC (metacarpal) neck fracture
  - <15 in index and middle finger
  - <30-40 in ring and little finger
- MC (metacarpal) base fracture
  - Adult < 20 angulation
  - Children < 40 angulation

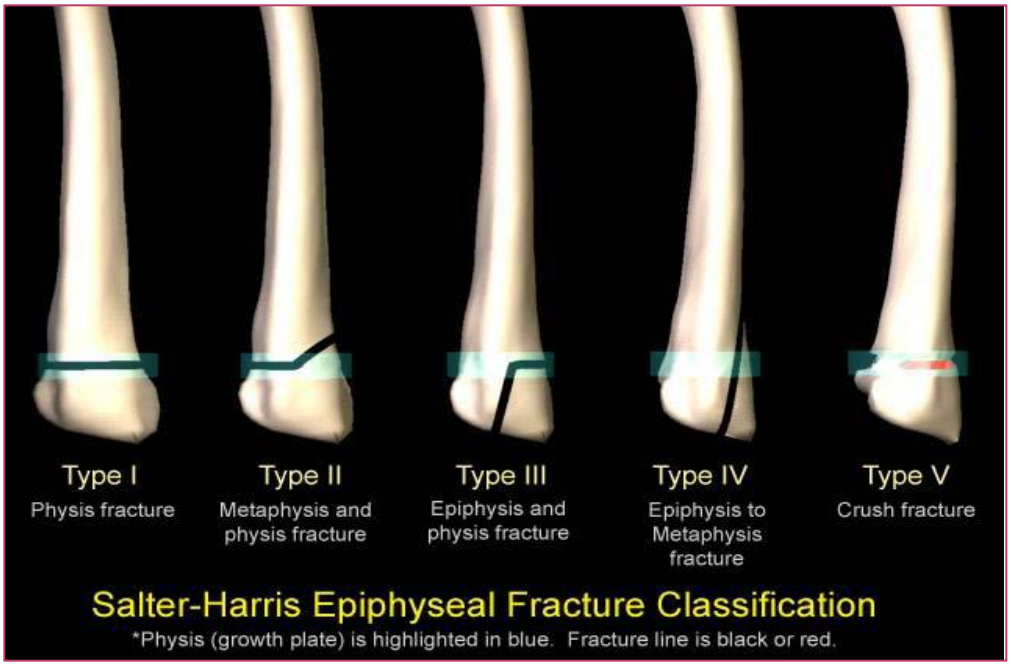


6.1.2 UNACCEPTABLE PHALANGEAL FRACTURES (NEED FIXATION)

- Rotational angulation
- Sever dorsal angulation
- Lateral angulation

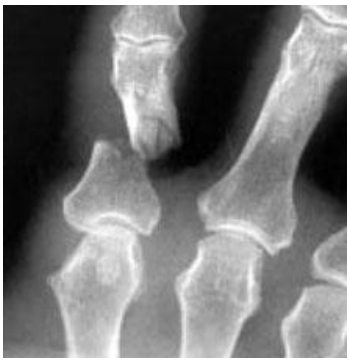
6.2 PEDIATRICS HAND FRACTURES – SOLTER HARRIS CLASSIFICATIONS

- Type I – A transverse fracture through the growth plate 6%.
- Type II – A fracture through the growth plate and the metaphysis, sparing the epiphysis 75% incidence, away from joint.
- Type III – A fracture through growth plate and epiphysis, sparing the metaphysis 8% goes to joint
- Type IV – A fracture through all three elements of the bone, the growth plate, metaphysis, and epiphysis 10%, above and below joint.
- Type V – A compression fracture of the growth plate (resulting in a decrease in the perceived space between the epiphysis and diaphysis on x-ray) 1%
- Fracture in child (growth plate) will affect grow, if the fracture in one side after 6 years pt will come with angulation of finger b/c one side grow and other didn't.

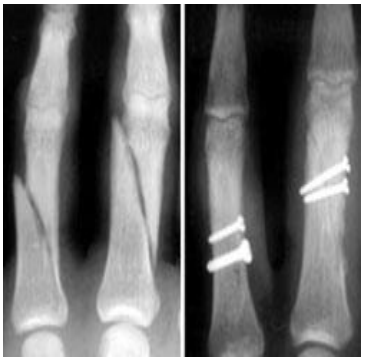


6.3 INDICATION FOR FIXATION NON-ARTICULAR

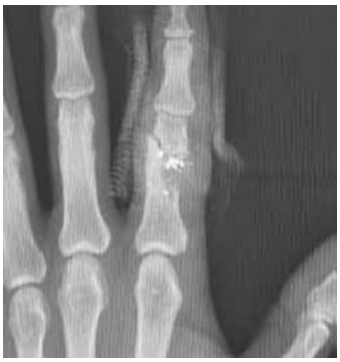
- Angulation
- Rotation
- Shortening



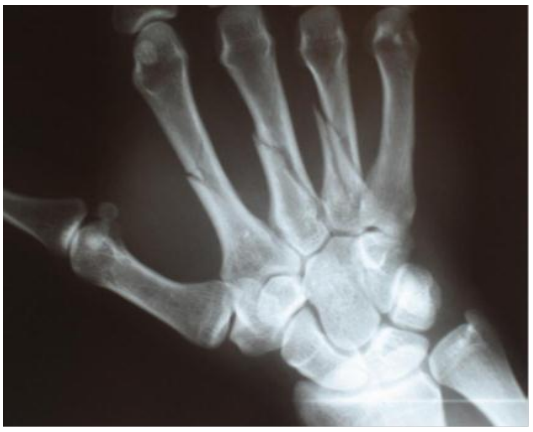
1. Transverse fracture of proximal phalanx.

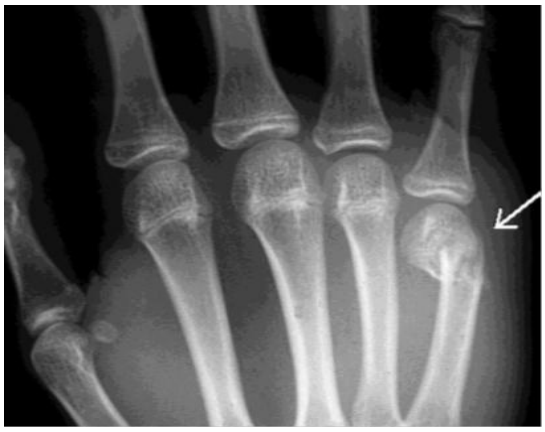


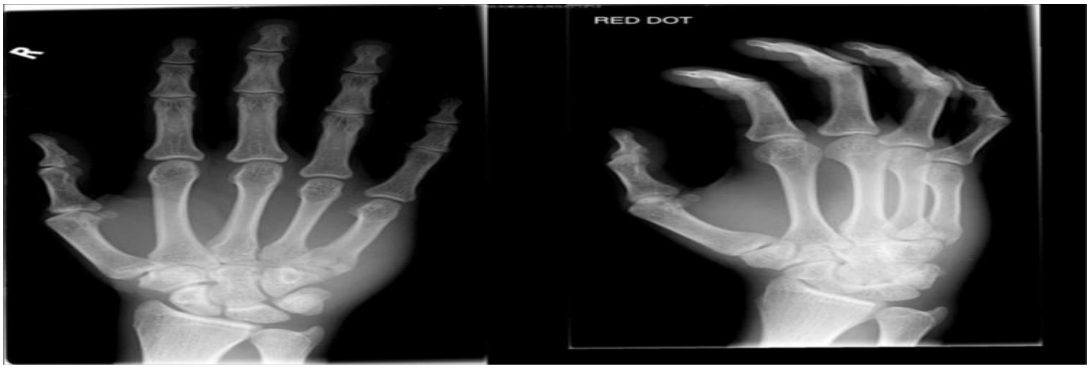
2. Spiral fracture



3. Gun shots and bone loss



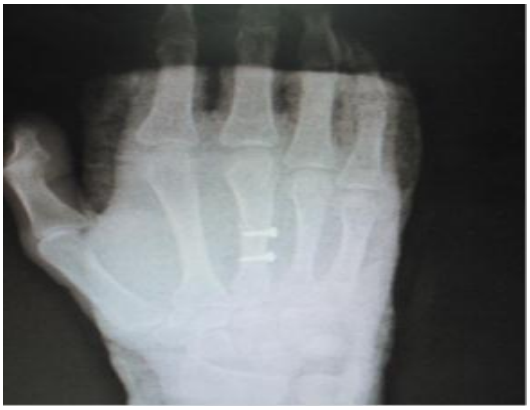


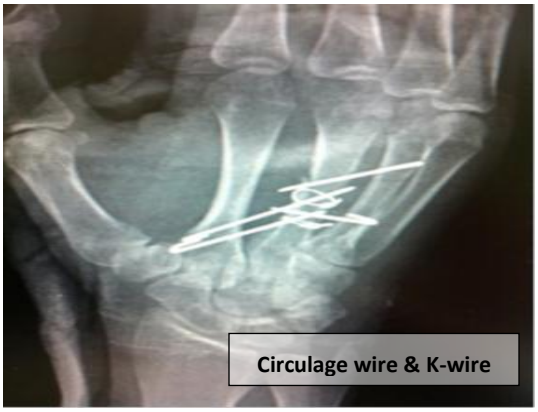


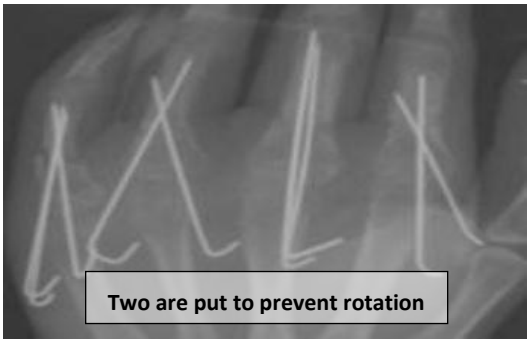
- Fractures of metacarpal bone:
  - Head
  - Shaft
  - Base
- Ask pt where the area of maximum tenderness is, thenlook at this area on X-ray

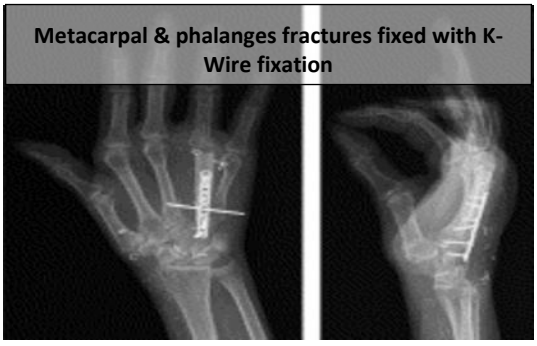
6.4 TECHNIQUE OF FIXATION

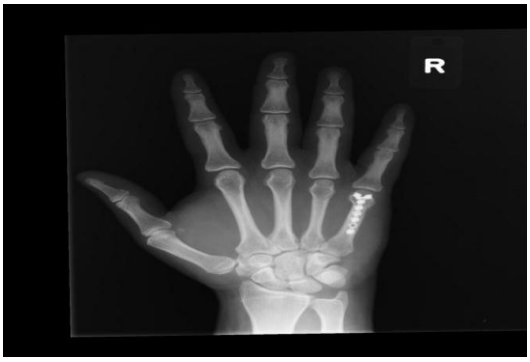
- 1st do x-ray; if it's reduced, you don't need to fix it>Close reduction splint
- If it doesn't stay in place > Close reduction K-Wire fixation
- ORIF (Open Reduction Internal Fixation)
  - Lag Screw
  - Plate
  - Circulage wire











7 CARPAL TUNNEL SYNDROME

7.1 INCIDENCE

- The most common nerve compression in the upper limb: 1 – 10% of the population
- As high as 60% in people with repetitive hand movement: Because of hand swelling
- Anatomy
  - Base (floor) is the bony carpal arch
  - Bridge (roof) is the flexor retinaculum
  - Borders: scaphoid, trapezium, pisiform, triquetral.
  - Has 9 flexor tendons and the median nerve

7.2 AETIOLOGY

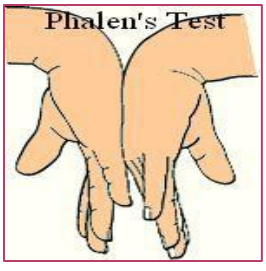
- Due to increase in volume of the content or reduction of the tunnel size
  - Acromegaly
  - Trauma
  - OA
  - Ganglion, Lipoma
  - Inflammation Tenosynovitis, gout
  - DM, Thyrotoxicosis, Pregnancy
  - Congenital:
    - Abnormal muscle, persistent median artery

7.3 SYMPTOMS

- Pain
- Numbness
- Paraesthesia in the median nerve distribution
  - Radial 3.5 digits
- Night pain
  - When the patient sleeps on his hand, everything swells so he wakes up with more numbness in the morning.
- Pain radiates proximally to the shoulder
- Weakness
- Clumsiness

7.4 CLINICAL FEATURES

- Weakness & wasting of the hand thenar muscles. When they hold something, it falls.
- Altered sensation in the median nerve distribution
- Positive Tinels sign
  - Tap over the carpal tunnel area of the wrist 5 or 6 times> tingling or paresthesia in the median nerve distribution
- Positive Phalan test
  - This position should be held for about 1 minute> numbness or tingling along the median nerve



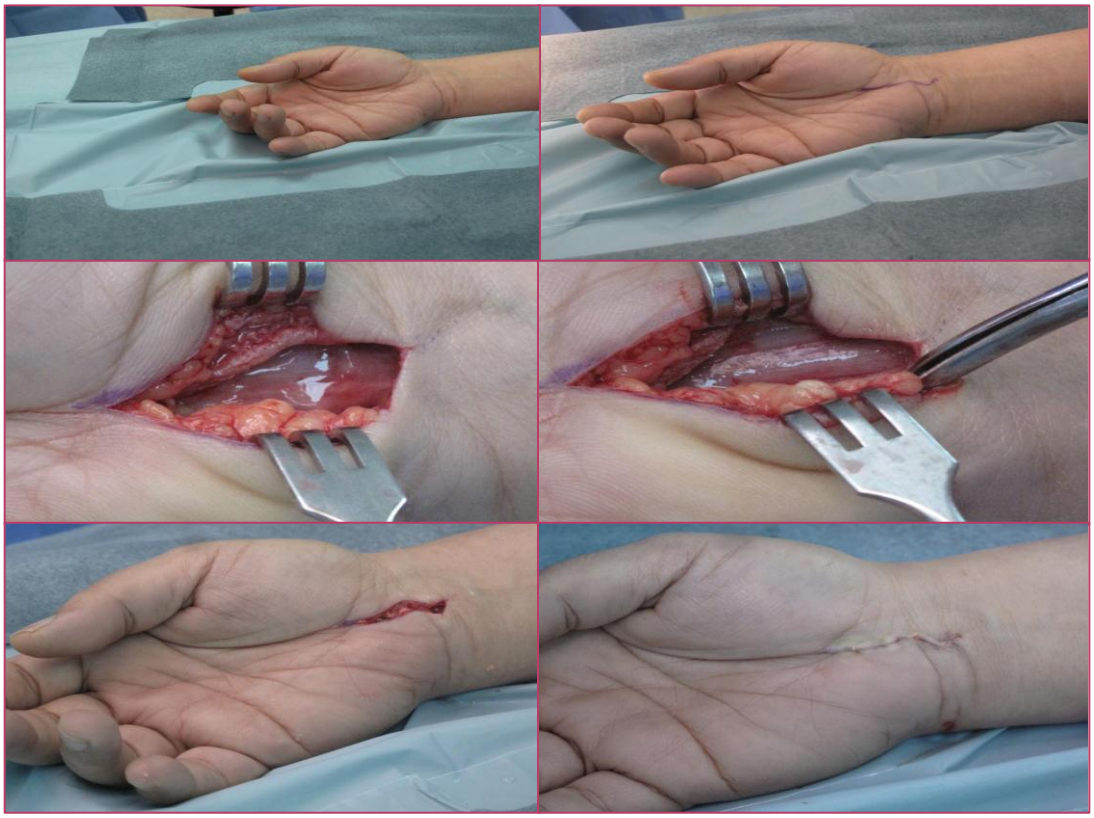
distribution
  - Reverse Phalan test
- The more severe the compression the faster the numbness

7.5 INVESTIGATIONS

- X-Ray
- CT scan
- MRI
- Nerve conduction studies: Most common test used

7.6 TREATMENT

- Non-Operative (Mild)
  - Splints
    - Rests the hands but once stopped > symptoms will return
  - NSAID's
  - Steroid Injections
- Operative (Persistent)
  - All Open technique
    - The best approach
  - Limited incision Technique
  - Endoscopic Techniques
    - Lots of reports of injuries to the median nerve



8 MCQS

1. The true statement regarding tendon injuries in the hand is:

a) Flexor digitorum superficialis inserts on the distal phalanx
b) Flexor digitorum profundus inserts on the middle phalanx
c) The tendons of flexor digitorum superficialis arise from a common muscle belly
d) The best results for repair of a flexor tendon are obtained with injuries in the fibro-osseous tunnel (zone 2)
e) The process of healing a tendon injury involves formation of a tenoma

2. Which of the following statements regarding carpal tunnel syndrome is correct?

a) It is rarely secondary to trauma
b) It may be associated with pregnancy
c) It most often causes dysesthesia during waking hours
d) It is often associated with vascular compromise
e) Surgical treatment involves release of the extensor retinaculum

🗝 Answers: 1;E , 2;B

SKIN AND SOFT TISSUE TUMORS

1 HEMANGIOMA

- It is the commonest skin tumor, and the commonest benign tumor of infancy. ⓘ
- It is classified based on the likelihood of proliferation or regression to:
  - Involuting; will regress on its own.
  - Non-involuting; won't regress on its own.

1.1 INVOLUTING HEMANGIOMA (HEMANGIOMA OF CHILDHOOD)

- It makes up to 95% of all hemangiomas.
- It is a neoplasm of endothelial cell origin, i.e. it is a hamartoma, not a true neoplasm.
- Presents at birth or during the first 2-3 weeks after birth, and grows rapidly for 4-6 months.
- Undergoes complete spontaneous slow involution; usually completely disappears at the age of 5-7 years.

Both benign tumors and hamartomas are composed of normal cells in excessive quantities, but benign tumors have a normal arrangement whereas hamartomas have an abnormal arrangement of cells.

In involuting hemangioma, the deeper they go the bluer they become, whereas the more superficial the more cherry red they get.

1.1.1 CLASSIFICATION:

A. Superficial (strawberry news):

- A type of capillary hemangioma (a nevus vasculosum capillary hemangioma).
- Very superficial in the dermis.
- Appears as a sharp demarcated, red, slightly raised lesion with an irregular surface.

B. Deep (cavernous hemangioma):

- Arises from below subcutaneous tissues.
- Appears as a blue tumor covered by normal skin.

C. Combined:

- Combined dermis and deep dermis.
- Firm, usually purple to blue depending on the depth.
- May extend deeply into subcutaneous tissues.

1.1.2 TREATMENT:

- No need for treatment, just observe, unless it involves a vital organ or interferes with physiological functions, e.g. eyelid.

1.2 NON-INVOLUTING HEMANGIOMA:

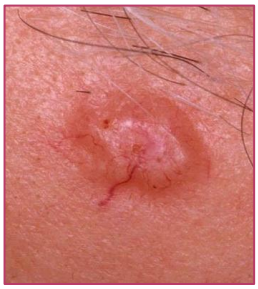
- True benign tumors.
- Usually present at birth.

- There is no rapid growth phase; its growth is proportional to the growth of the child.
- Persists to adulthood.
- Causes severe aesthetic (cosmetic) problems.
- May cause arteriovenous fistulas eventually leading to cardiac failure.
- Treatment is not satisfactory.

1.3 PORT WINE STAIN

- An extensive intradermal hemangioma, just below the epidermis, which is mostly made up of a collection of dilated venules and capillaries. It has a deep purple red color.
- May involve any portion of the body, usually as flat patches in the face.
- Usually follows the correlation of sensory branches of the 5^{th} nerve; so if it involves one branch of the trigeminal, it will spread to half of the face, whereas if it involves both branches it will spread to the whole face.
- Microscopically, it appears as thin walled capillaries distributed throughout the dermis, lined by thin mature flat endothelial cells.
- Treatment:
  - Unsatisfactory.
  - Tattooing.
  - Radiotherapy: causes a scar as it destroys both blood vessels and the skin overlying the lesion.
  - Laser has a special wavelength affecting the blood vessels without affecting the skin, but it is expensive.

2 BASAL CELL CARCINOMA (BCC; RODENT ULCER)



- The most common malignant cancer of all skin tumors.
- Locally invasive malignant tumor, which may lead to massive ulceration.
- Very rare to metastasize. ⓘ
- Affects ages over 40, and men are more affected than women.
- Risk is increased in:
  - Individuals with high cumulative exposure to UV light through sunlight, e.g. live in tropical areas.
  - Those with fair, white skin, especially when the person has blond or red hair and blue, green, or gray eyes, e.g. westerners working in KSA.
- Mostly presents in the face and the neck.
- Grows slowly (not aggressively), steadily and painlessly, and several months or years may pass before the patient finally visits a doctor.
- Death may occur due to secondary complications of invading deeper tissues or major blood vessels.
- Appearance:
  - Small translucent, skin elevated nodule with rolled pearly edges.
  - Tetangiectatic vessels may occur on the surface.
  - Flat and white or waxy appearance with firm palpation.

- Histologically, it appears as elongated strains of basal cells that infiltrate the dermis.

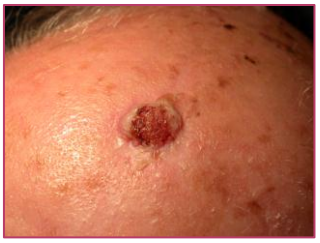
2.1 BASED ON APPEARANCE, THERE ARE 4 FORMS:

1. **Erythernatous (superficial) basal cell carcinoma:**
   - Occurs most frequently on the trunk.
   - Appears as a reddish plaque with an atrophic center, and smooth, slightly raised borders.
2. **Pigmented basal cell carcinoma (frequent in our country):**
   - Sometimes mistaken with melanoma, but it is darker.
   - Extends deep to the subcutaneous tissue.
3. **Nodular basal cell carcinoma.**
4. **Cystic basal cell carcinoma.**

2.2 TREATMENT:

- Curettage and electrodessication (cautery), with excising a safety margin of 2-3 mm.
- Surgical excision (the best treatment): small moderate sized lesions, with removal of the subcutaneous tissue and do reverse face-lift flab if the lesion occurs in the face. ⓘ
- Radiotherapy: good for treatment of structures that are difficult to reconstruct but hospitalization is not required. Should not be used in patients under 40 years, due to mutation, or in patients who failed to respond to radiation therapy. Treatment usually lasts 4-6 weeks.
- The more well differentiated the tumor the more radioresitant it is. And the more undifferentiated the tumor the more radiosensitive it is.

3 SQUAMOUS CELL CARCINOMA (SCC)



- The second most common cancer in ***light*** skinned people, but the first in ***dark*** skinned ones.
- There is a potential for metastatic spread. ⓘ
- The causative agents are the same as basal cell carcinoma, along with:
  - Excessive contact with hydrocarbons such as tar, gasoline, and paints. (i.e. occupational hazard related)
  - Exposure to ionizing radiation.
  - Chronic ulcers.
  - Scars of thermal bums healed repeatedly by fibrosis (especially if it was over a joint), which may lead to Marjolin's ulcer.
- Most common sites are the face and neck, e.g. ears cheeks, and the lower lip, and the back of the hand.
- Presents as:
  - Locally invading without metastasizing.
  - Premalignant tumor, as Bowen's disease or chronic radiation dermatitis.

- Rapidly growing, widely invasive with metastasis, especially SCC arising from normal skin.
- Initially starts as an erythematous plaque or nodules with indistinct margins.
- Surface may be: flat, verrucose (warty) or ulcerative.

- Histologically, malignant epithelization is seen extending down into the dermis like horns of pearls, which is not seen in basal cell carcinoma (BCC). ⓘ

3.1 TREATMENT

- Surgical excision with 4-5 mm margin in all directions.
- Radiotherapy: the more well differentiated the tumor, the more it resembles normal skin, the less potential to metastasize, and the less radiosensitivity, and vice versa.

Sarcomas metastasize through blood, while carcinomas metastasize through lymphatics.

4 MALIGNANT MELANOMA (MM)

- Incidence is over 300,000 of skin tumors every year in USA, 9000 of these are melanomas, i.e. 4.6%.
- 2/3 of all skin tumor deaths are from melanomas. ⓘ
- Incidence of and survival also were increased from 41% to 67%.
- Whites have a higher incidence than blacks, but there is **NO** sexual predominance.
- Risk factors:
  - UV radiation.
  - Family history.
  - Average person has 15-20 nevi, 1/3 of the melanomas arise from a pre-existing pigmented nevi.

4.1 TYPES OF NEVI:

1. **Junctional nevi:** (arise from the junctional layer, which is the dividing layer of the skin)
   - Small, circumscribed, light brown or black colored, flat, slightly raised and rarely contains hair.
   - Mainly lies between epidermis and dermis.
   - May be formed in mucous membranes, genitalia, soles and palms.
   - More likely to be malignant.
2. **Intradermal nevi:**
   - Small spots, color ranges from blue to bluish black, flat and dome shaped.
   - Compound; found in both epidermis and dermis.
   - Less likely to become malignant.
3. **Dysplastic nevi:**
   - Pink base with indistinct irregular edges.

- Usually have embryonic tissues, i.e. ectoderm, mesoderm, or endoderm.
- Most dangerous type in newborns.
- Family history is important.
- Most lesions are small, and suspicious lesions must be excised.
  - Congenital: excision in 1% of newborns also with dysplastic is considered to be premalignant.

4.2 HISTOLOGICAL CLASSIFICATION:

- **Superficial spreading melanoma (the commonest):** arises from a preexisting mole; common in blacks without sexual predominance.
- **Nodular melanoma:** becomes large and ulcerated before it is noticed.
- **Lentigo maligna (melanoma):** most commonly occurs in old patients, especially from a preexisting mole.
- **Acral lentiginous melanoma**.

4.3 CRITERIA THAT SUGGEST MELANOMA FROM MOLE, AND CONSEQUENTLY SUGGESTS ITS EXCISION:

- **Color**: focal shades with red, blue, white or darkening in color.
- **Size**: recent rapid diameter enlargement of more than 10 mm.
- **Shape**: irregular margin, notching and indentation.
- **Surface**: ulceration, bleeding, crusting, irregular elevation.
- **Symptoms**: pruritus, inflammation and pain.
- **Location**: back, lower extremities, location is subjected to **BANS** area; **B**ack, posterolateral part of the **A**rm posteroiateral part of the **N**eck and **S**calp; they are the anatomical areas that have a higher risk rate and a lesser survival rate.

4.4 STAGING (CLARK'S CLASSIFICATION):

- Based on the histologic level of invasion of the tumor.
- Performed after excisional biopsy.

Level	Feature	Mortality and morbidity rates
I	In situ; above basement membrane (confined to the epidermis)	0%
II	Invades the papillary layer of the dermis	4%
III	Lesions reach the junction of the papillary and reticular layers	33%
IV	Lesions invades the reticular dermis	61%
V	Lesion invades subcutaneous tissue	78%

4.5 NODE DISSECTION:

- Advised prophylactically as:
  - Level **I** and **II**: no need of dissection.
  - Level **III**: some will do it and some will not.

  - Level **IV** and **V**: dissection is mandatory.
- Not advisable in:
  - Lymphatic drainage of sites involved. (e.g. if there's a melanoma involving the breast, you can't simply excise all of the lymphatics groups!)
  - Patients over 70 years old.
  - Serious concurrent disease.
  - Unresectable distant metastasis.

4.6 PROGNOSIS:

- Depends on the tumor size and depth of invasion.
- Less than 2 cm in diameter and less than 0.7 mm in depth is curable by wide local excision.
- Nodular melanoma with ulceration has a poor prognosis.
- Lesions in the extremities have a better prognosis than trunk lesions.
- Women have a better 5 years survival rate than men.

4.7 NONSURGICAL TREATMENT (IMMUNOTHERAPY):

- Small metastatic lesions treated with BCG may be tried on healthy patients.
- Melanoma is radioresistant; so radiotherapy is rarely used in treatment and may be used in palliation.
- Chemotherapy with phenylalanine and alanine-mustard and other drugs.
- Survival is better in limbs because a limb can be isolated and treated
- Long-term palliative treatment of large lesions, which underwent surgery, is with radiotherapy and chemotherapy.

5 MCQS

1. According to Clark's classification invasion of papillary layer in malignant melanoma is:
   a. Clark 1
   b. Clark 2
   c. Clark 3
   d. Clark 4
   e. Clark 5

2. Basal cell Carcinoma:
   a. Metastasis is usually to Lymph nodes before systemic Metastasis
   b. Metastasis is usually systemic before lymph nodes Metastasis
   c. Metastasis is usually to both lymph nodes and systemic Metastasis at the same time
   d. Metastasis is usually to skin as " Satellite " Lisions
   e. Does not develop Metastasis

3. Patients with Gorlin Syndrome are known to develop:
   a. Basal Cell Carcinoma
   b. Melanoma

c. Squanous Cell Carcinoma
d. Bowen's disease lesions
e. Dysplastic nervi

4. Squanous Cell Carcinoma of the skin:
   a. Is Radio Sensitive
   b. Is best treated by Chemotherapy
   c. Surgery is done with 5cm skin margin
   d. Usually seen in children
   e. Its Metastasis is usually systemic before lymph node metastasis

5. Melanoma:
   a. Nodular melanoma has a better prognosis than all other types
   b. Acrol Melanoma is known to have the best prognosis
   c. Is Radio sensitive
   d. Usually develops metastasis to lymph nodes before systemic metastasis
   e. Is more common is black populations

6. A melanoma with Clark level II:
   a. Reaches the epidermis
   b. Reaches the Basal layer
   c. Reaches the Reticular Dermis
   d. Reaches Junction of Reticular and papillary dermis
   e. Reaches the papillary Dermis

7. Majolin's ulcer:
   a. Is a type of basal cell carcinoma
   b. Is a type of squamous cell carcinoma
   c. Is a type of Melonama
   d. Is a type of ulcer is a blue nervus
   e. Is a type of an ulcer in a dysplastic nervus

8. Strawberry hemangioma in a newborn in the cheek:
   a. Best treated by surgical excision
   b. Best treated by steroid injection
   c. Best managed by observation for 4-5 years
   d. None of the above
   e. All of the above

🗝 Answers: 1:b, 2:e, 3:a, 4:a, 5:d, 6:e, 7:b, 8:c.